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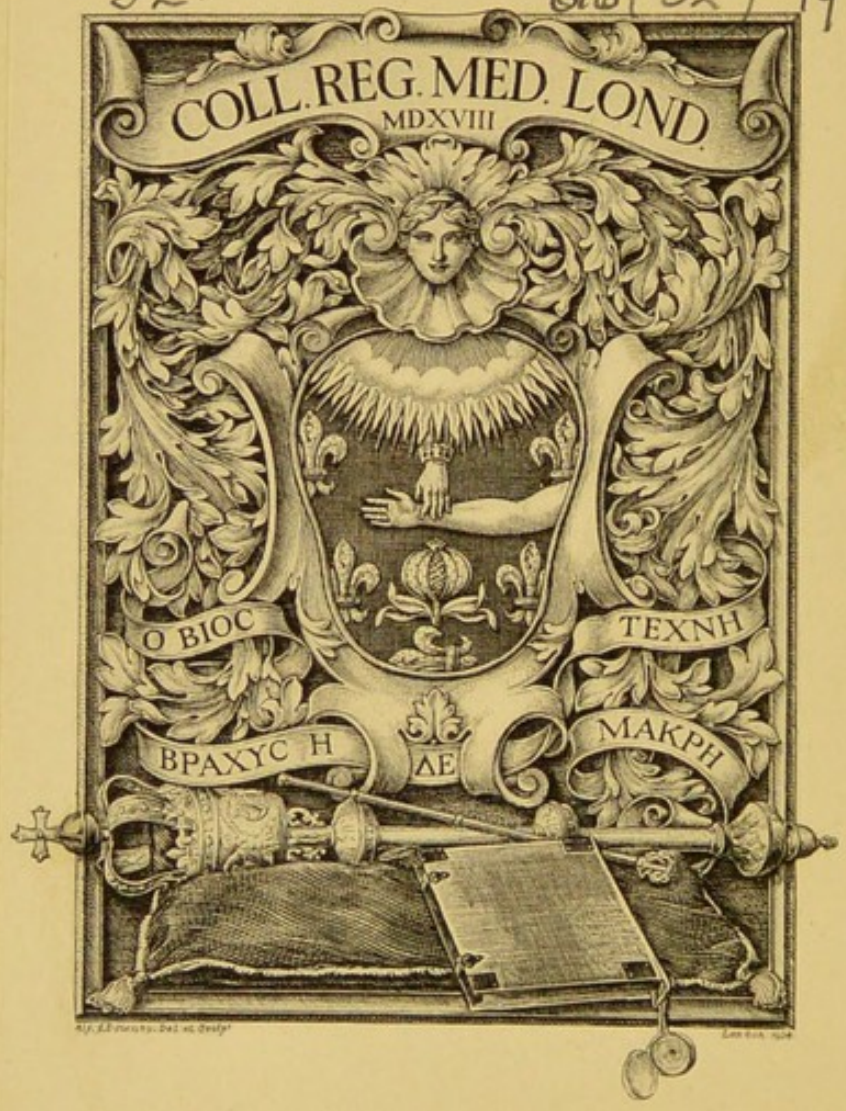

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PRESS NOTICES OF THE FIRST EDITION OF DR. SAVILL'S SYSTEM OF CLINICAL MEDICINE.

"It was a happy inspiration that suggested the writing of this two-volume text-book. Dr. Savill has rendered a great service to the general practitioner by writing this book. "Its plan is simple. . . . Dr. Savill's method of approaching his subject is from the standpoint of symptoms and physical signs. He selects a symptom or symptom group and then proceeds to differentiate the various diseased conditions in which this may be presented to the physician. Not only is the diagnosis given, but in many instances the etiology also, and in every case the prognosis and valuable suggestions as to treatment. At the end of Volume I. is given a useful series of formulæ, many of which are entirely original.

"Although the work as a whole is unimpeachable, we think Dr. Savill is at his best in the chapter on Diseases of the Nervous System. To the student and practitioner these present difficulties with which the specialist has no trouble whatever. The writer, however, anticipates these in a very skilful manner. His scheme for the investigation of these diseases is simplicity itself. If we take such a condition as hemiplegia for example, we find the ten chief causes of this affection distinguished according as the onset is sudden or gradual. Then each of these causes is dealt with seriatim, and in such a clear and simple manner that no medical reader of average intelligence can fail to make a correct diagnosis in any particular case. This is but one instance; the whole work abounds with valuable facts for the guidance of physicians. There is no easy road to acquiring exact clinical diagnostic skill, but Dr. Savill's work goes far to smooth that path. . . . The entire work contains little over 1,100 pages, many of them printed in small type, but we know of no other book which contains within the same or even greater compass so much practical and helpful information."—*Medical Times.*

"It is a monument of careful classification, founded on many years' experience in the enormous clinical field of the Paddington Infirmary. . . . There is no doubt that a careful study of it will give a broader conception of medicine than can be obtained from many larger works. Its great virtue is that it is obviously born of extensive practical knowledge. . . . The illustrations are good, and the two coloured plates of measles and variola are really excellent."—*St. Mary's Hospital Gazette.*

"The manner in which the diagnosis and prognosis of disease is dealt with is not only adequate, it is actually, according to our present knowledge, exhaustive."—*Journal of Balneology and Climatology.*

"In writing 'A System of Clinical Medicine' Dr. Savill has adopted a plan which we believe will be found of great assistance both to students and to practitioners. He has approached the subject from the standpoint of symptomatology. . . . This scheme has been admirably carried out, and for the most part Dr. Savill has been successful in producing a very useful and practical work . . . a very original work. . . . We have formed a high opinion of Dr. Savill's work, and we wish it the success that it deserves."—*The Lancet.*

"This work attempts much more than the books dealing with medical diagnosis merely, for sections upon prognosis and treatment and, where thought advisable, pathology also are introduced. . . . The book deserves praise for its general get-up, and for its abundance of information and references."—*St. Bartholomew's Hospital Journal.*

"The plan of this work is quite original. . . . This method obviously imitates the actual process of reasoning employed at the bedside and in office practice as closely as it is possible to reproduce a practical procedure in print, and has the great advantage of preserving the relative value of diseases which is often lost sight of in systematic text-books. . . . The practical problems in diagnosis are presented as they are actually met in practice."—*American Journal of the Medical Sciences.*

"This book fills a distinct want. It will serve as a clinical index of diseases, and the advantages of passing in rapid review all the possible diseases which may give rise to a patient's leading symptom must be very obvious to those engaged in clinical work. We thoroughly agree with the author that his plan gives a truer view of nature's facts than one which deals with disease as so many entities. . . . The work is thoroughly practical and is eminently personal. Throughout its pages Dr. Savill offers to his readers the ripe fruit of a vast experience, and we have the greatest confidence in recommending this work to both students and practitioners of medicine."—*Glasgow Medical Journal.*

"The plan Dr. Savill has adopted we find altogether excellent. . . . The whole matter in the book is complete, concise, and well written; the author modestly in the preface informs us that he has attempted clearness rather than literary style, but he has certainly succeeded in avoiding 'dryness,' without, as some authors do, introducing what is known as 'padding.'"—*The General Practitioner.*

A SYSTEM OF CLINICAL MEDICINE

WORKS BY THE SAME AUTHOR.

CLINICAL LECTURES ON NEURASTHENIA.
Fourth Edition. London, 1908.

LECTURES ON HYSTERIA AND ALLIED VASO-MOTOR DISORDERS. London, 1909.

A SYSTEM

OF

CLINICAL MEDICINE

DEALING WITH THE

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASE

FOR

STUDENTS AND PRACTITIONERS

BY

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LONDON

EDWARD ARNOLD

1909

PREFACE TO THE SECOND EDITION

THIS work differs from current books on medicine chiefly in this respect, that it approaches the subject from the point of view of symptomatology, first describing the symptoms or *effects* of disease, and then tracing these symptoms to their *causes*—namely, the various diseases which may be in operation. It was written to aid my professional brethren in general practice in their daily work (which consists of tracing symptoms to their causal disease) and to help senior students who had reached the stage when medicine may be profitably studied in the wards. It was an original, and perhaps a too ambitious, project; but the rapid sale of the first edition, which has now been out of print nearly two years, and the number of kind and suggestive letters received from medical men in general practice in many different parts of the world, and from teachers and senior students, encourage me to believe that I have in some measure succeeded.

This edition appears in one volume instead of two, as being more convenient. The amount of material remains approximately the same, new matter replacing old. In deference to the wishes of many of my readers, the smallest of the three types (nonpareil) in the first edition has been replaced by medium-sized type (brevier), so that now only two main types instead of three are used. Space has been saved by the lines of type being closer, and consequently the bulk of the volume forming the second edition is somewhat smaller than the bulk of the two volumes together which formed the first.

The arrangement and scope of the work are unchanged. Clinical medicine moves less rapidly than pathology, so it has not been necessary to make any very extensive alterations. The book has, however, been brought thoroughly up-to-date. The exigencies of a busy life have prevented my giving as much time to its revision as could be wished, but I have been fortunate in securing the valuable aid of Dr. Frederick S. Langmead and Dr. Agnes F. Savill. The last-named is mainly

responsible for the chapter on Diseases of the Skin, the first-named for the alterations in the chapter on Diseases of the Joints; both are responsible, with me, for general revision throughout.

I should like to take this opportunity of acknowledging the valuable criticisms I have received from many correspondents, and to say that further suggestions and criticisms from future readers will be gladly welcomed.

T. D. S.

September, 1909.

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INTRODUCTION

EVOLUTION.

Those who ponder on general principles and methods will have observed that a considerable change has gradually taken place during the last half-century in the methods of studying the science and art of medicine. Formerly, men were content to observe the symptoms or effects of disease at the bedside and in the dead-house, and to speculate on the etiological connection of these two series of phenomena. Wherever the association of such phenomena during life and after death was sufficiently constant they were spoken of collectively as a "disease"; when a group of symptoms without anatomical lesion constantly recurred, it received a name and place among the list of "disorders." Then each disease or disorder was taken as a separate entity, its anatomy, symptoms, diagnosis, and treatment were described, and its various possible etiological factors discussed; and the result was known as "Descriptive" or "Systematic Medicine." The guiding principle of this descriptive process was the tracing from an assumed *cause* to a known *effect*.

In later times great advances were achieved, almost synchronously, in two very different directions. On the one hand great improvements were made in the methods of observing and investigating the symptoms or effects of disease during life, and thus Clinical Medicine came into separate existence. This stage was marked by the appearance in this country of two very successful works—one by Dr. Samuel Fenwick, of London, on "Medical Diagnosis," first published in 1869, dealing with the symptoms and diagnosis of disease; another by Dr. James Finlayson, of Glasgow, entitled "A Clinical Manual," first published in 1878, dealing with the methods of observing and investigating the symptoms of disease. On the other hand, with the extremely rapid growth of chemical, biological, and bacteriological sciences, and the elaboration of experimental methods in the investigation of disease processes, a new school of pathology arose, whose methods were based upon experiment, and whose leading principle was the artificial production

of a definite *cause* and the observing of its *effects*. The extraordinary advances made by these means, and the new light thus shed upon the science of medicine during the last twenty years, form at once the wonder and delight of the civilised world.

As a result of the movement to which I have referred, and the growth in the two directions named, treatises on Systematic Medicine, which attempt to deal at all fully with both the clinical and the pathological aspects of disease, have come to assume very considerable dimensions. In many of them there seems to be a tendency to become more and more pathological in their arrangement, and to treat diseases as separate entities, so that students of clinical medicine and busy practitioners, whose daily work consists of an endeavour to trace from *effect* to *cause*, have been heard to complain that they do not always find in them the clinical aid they seek.

ORIGIN.

Immediately after embarking on medical practice I realised, as probably many others have done, the importance for diagnostic purposes of reviewing the various diseases or pathological conditions which might give rise to a patient's leading symptom or symptoms, and being unable to find precisely the information desired in any of the current text-books, I proceeded to keep a brief record of all the cases I met with arranged under the heading of their leading symptom. This book is based upon those records, which extend over many years, combined with the valuable knowledge imparted to me at the bedside by my teachers—more especially Dr. Charles Murchison, Dr. J. S. Bristowe, Professor J. M. Charcot, and Sir William Broadbent. Hospital cliniques, at first of a general and later of a more special kind, have always been at my command ; but it was at the Paddington Workhouse and Infirmary that the idea of this work was conceived, its foundations laid, and the chief part of its "skeleton" constructed. It would be hard to conceive circumstances better suited to the task, for our great poor-law infirmaries contain, as all the world now knows, a vast and almost unexplored field of every possible variety of disease, which can be studied from day to day from the beginning *to the end* of its course.

PLAN.

As regards the plan and arrangement of this work, the subject will be approached from the standpoint of symptomatology. The principle throughout will consist of tracing from effect (symptoms) to cause (the morbid process in operation). The order of sequence will be that which should be adopted in the examination of a patient. Thus, the first chapter will give a general scheme for the

examination of a case, and will deal with certain general principles underlying methods of observation, diagnosis, prognosis, and treatment. In the second chapter the physiognomy of disease will be discussed. The succeeding chapters will deal seriatim with the symptoms and signs referable to the several organs or anatomical regions of the body, and the diseases which may cause those symptoms.

Each chapter will be divided into three unequal parts. Part A. will treat of the *symptoms* which may indicate disease of the organ or region under discussion, the fallacies incidental to their detection, and a brief differential account of the various causes which may give rise to those symptoms. Part B. will treat of the *physical signs* of disease in that region, and the various methods used to elicit them. Part C., which constitutes the major portion of each chapter, will be prefaced with a *clinical classification* of the various maladies affecting that region, and a summary of the routine procedure to be adopted ; and this will be followed by a series of sections dealing with the several *diseases*, arranged according to their clinical relationships. For example, in Chapter III., on The Heart—Part A. describes and differentiates the various causes of breathlessness, palpitation, precordial pain, and the other symptoms which may be indicative of heart disease ; Part B. describes percussion, auscultation, and the other methods of examining the heart ; and Part C. deals seriatim first with acute, and secondly with chronic cardiac disorders, classified and arranged on a clinical basis.

Apart from the general plan and arrangement, there are two features special to this work. The first part of each chapter, dealing with symptoms and their causes, forms a feature on which great labour has been expended. To make each list of causes complete without redundance, and to check the various data again and again in the light of experience, has involved an expenditure of time quite out of proportion to the space occupied. These lists will, I trust, be as useful to others as they have been to me in obtaining a clue to diagnosis.

SPECIAL FEATURES.

Another feature consists of the italicised paragraphs in Part C. standing at the head of each section, which deal with a separate malady. These emphasise the salient features by which a disease may be recognised and differentiated from others belonging to the same clinical group. They are, in fact, brief clinical definitions, and form, metaphorically speaking, " sign-posts " or guides in the process of diagnosis. If, after carefully studying the lists of symptoms and their causes in Part A., and examining his patient (Part B.), the reader turns to these

italicised paragraphs in Part C., the work will, it is hoped, serve as a "clinical index of diseases"; for by following the plan laid down he will shortly find himself reading a description of the diagnosis, prognosis, and treatment of the malady from which his patient is probably suffering; while adjacent to this are the disorders which clinically, and very often pathologically, resemble it, and for which in practice it is apt to be mistaken.

Such an arrangement as that proposed must inevitably lead to some repetition, but this difficulty has been obviated to a certain extent by cross references. I would also ask the reader to remember that nothing fixes things so well in our minds, or aids us so much in tracing those analogies to which I shall shortly refer, as constantly looking at the same facts from a different point of view.

An attempt has been made to present the various diseases in some kind of perspective by placing them as far as possible in order of importance and using different sized types. The relative importance of different subjects in medicine is largely a matter of opinion, and I cannot expect to escape criticism in this respect.

It is a standing accusation against medical writers that they are careless in respect to literary style, and I fear that I shall not be found an exception. I have striven to be intelligible rather than academic; and in general I fear that I must plead guilty to having endeavoured to follow the Duchess's advice to Alice in Wonderland, to "take care of the sense and the sounds will take care of themselves." When so large an area has to be covered, a certain amount of abbreviation is indispensable, and in order to condense my material it has been my practice to adopt a numerical method of description. Some may take exception to this, though the student will find it to his advantage in the acquisition of knowledge.

ADVANTAGES.

I may perhaps be pardoned for adverting to certain advantages which appear to me to be associated with the method that I have adopted of approaching clinical medicine. And first let me remark that this method of diagnosis is not what has been called a "process of exclusion." It is a positive rather than a negative process, for by carefully considering the various causal diseases which may be in operation, and balancing the evidence for and against each, the physician is guided, not to the least improbable, but to the most probable diagnosis.

The advantages of passing in rapid review all the possible diseases which may give rise to a patient's leading symptom, are very obvious

to those actively engaged in clinical work. It was Dr. Charles Murchison's method in his bedside teaching; and another equally great clinician, Dr. Matthews Duncan, has aptly remarked: "If you do not know of a thing, you are quite sure not to suspect it; and in all cases of difficult diagnosis, if you do not suspect the disease, you are almost certain not to find it."[1] But I am not aware that any work has yet been published which adopts precisely this plan of approaching clinical medicine.

This plan gives, I venture to think, a truer view of nature's facts than one which deals with diseases as so many separate entities. We see a case in all its clinical and practical bearings. We not only learn that the diagnosis of a patient's malady can at best be only a question of the greatest probability, but with almost mathematical precision we can also assess the probability or improbability of each of the other possible causes in operation. We learn further that all diagnoses can only be provisional, and that the degree of probability of each possible cause changes from day to day, like the coloured pattern of the kaleidoscope, as the course of the malady unfolds itself before us.

It is, moreover, in clinical work carried out on these lines—where diseases presenting analogous clinical phenomena are constantly being associated together from different points of view—that the rôle of the imagination, both in the investigation and in the treatment of disease, finds a legitimate place. The recognition of a clinical likeness between diseases has often led to the erection of a "working hypothesis" which by subsequent research has been found to be correct. Many of our greatest discoveries have been initiated in this way. It was, for instance, a process of this kind which led to the discovery that a large number of, perhaps all, pyrexial disorders are of microbic origin. There are still a number, notably measles, small-pox, and scarlatina, in which such a working hypothesis, based on clinical resemblances, forms at present the full extent of our knowledge; but so precise are these foundations that the microbic nature of these diseases is never doubted. Hypotheses framed in this way should always be tested and confirmed in the laboratory and dead-house, whenever the morbid conditions can be produced experimentally, or when they are attended by fatal results. But unfortunately there are still a great many diseases, such, for instance, as the two great groups of clinical conditions we call hysteria and neurasthenia (conditions which form a not inconsiderable portion of the practitioner's daily work), which cannot, excepting in the most isolated

[1] "Clinical Lectures on the Diseases of Women," 4th edition, p. 15.

instances, be observed in the dead-house, and which have not yet been produced in animals. In these cases the method of analogy or comparison to which I have just referred is not only a valuable means of investigation, it forms almost the only means we have.

It is given only to few to devote the necessary time to laboratory research ; but all can study their cases at the bedside in the way indicated, and many a valuable and often unrecorded idea as to treatment will occur to the practitioner who thinks out and traces clinical analogies between diseases.

There is yet another advantage which has always appeared to me to accrue, especially to the young observer, by this process of balancing evidence and comparing diseases. It not only impresses important facts upon his memory, but it constitutes one of the best possible means of training him to habits of accurate and complete observation, and of systematic and productive thought. The scope of his horizon is widened, his faculty of systematising his knowledge becomes by practice wonderfully increased, and his reasoning powers strengthened and corrected. He finds intuitively that without accuracy in respect to the most minute details he may be led astray in the more important ones, that without system in the arrangement of his facts he will never be able to attach the proper significance and importance to each ; and finally, that without judgment in attaching due weight to each item of evidence, his conclusions may be erroneous although his premises and facts are correct.

I have now described the scheme of this work, its purposes and scope —in a word, the ideal which I hoped to compass ; and I believe no one could approach a task of this kind without realising the responsibilities and difficulties involved in its execution. Amidst the bewildering records of medicine there are many excellent treatises both on systematic medicine, the medicine taught in the schools, and on one or other of the several departments of clinical medicine. These deal with their respective subjects in a manner which I cannot hope to rival, and they have been to me an abundant source of instruction, but they have afforded me no exact precedent or guide along the path I wished to travel. The contemplation of the wide range of knowledge and experience required, of the immense advances which have recently been made both in the theory and practice of medicine, of the supreme importance of the subjects here dealt with, involving as they do questions of life and death, has filled my mind with a painful sense of the obligation imposed upon me to sift my facts,

RESPONSIBILITIES.

and to cull my knowledge, truly, from all sources, but, before all, to obtain my material as far as possible by careful observation and patient thought from the book of nature which lay open before me from day to day at the bedside in infirmary, hospital, and private practice.

In these circumstances I have gladly availed myself of the help and advice of many friends, and there are some to whom special acknowledgment is due. In certain parts of the chapter on fevers, notably on scarlet fever, measles, diphtheria, and enteric fever, I have had much valuable advice and suggestion in the revision of the proofs from my old friend Dr. Foord Caiger. Similarly in the subject of aneurysm and in parts of the subject of pulmonary disease I am indebted to Dr. Robert Maguire, in parts of the chapter on diseases of the throat and nose to Dr. St. Clair Thompson and Dr. Scanes Spicer, in parts of the section dealing with serum-therapeutics to Dr. George Dean, in parts of the chapter on diseases of the heart to Dr. Alexander Morison, and in parts of the chapter on the urine to Dr. C. O. Hawthorne. The illustrations, with few exceptions, are taken from actual cases, and have been drawn specially for this book under my own supervision ; my grateful thanks are due to the artist, Mrs. Stanley Berkeley, a Royal Academy medallist, who has lent her talent to enrich these pages with drawings which are not only accurate but, as far as scientific drawings can be, artistic. Finally, it is difficult for me to express in measured terms my indebtedness to my wife, who has assisted me in the elaboration of this work during the greater part of four years. Her skill and knowledge have largely helped to give it such completeness as it may possess ; her patient industry has afforded me not only assistance, but example ; and her companionship and encouragement have made many rough places smooth, and have often transformed what at times seemed to be a laborious and interminable task into a pastime.

T. D. S.

March, 1903.

A SYSTEM OF CLINICAL MEDICINE

CHAPTER I

CLINICAL METHODS

Preliminary Definitions—Case-Taking—Methods of Diagnosis, Prognosis, and Treatment—Rules for Clinical Investigation.

§ **1. Definitions.**—Disease is a departure from health, and is manifested in an individual during life by symptoms. These are of two kinds—"*subjective symptoms*," which are recognisable only by the patient, and present no external indication, such as pain, itching, or a feeling of chilliness; and "*objective symptoms*,"[1] which can be detected by the observer—*e.g.*, abdominal enlargement or dulness on percussion. The word "symptom" is used in two senses. Sometimes it is used in a general sense to indicate all the subjective and objective evidences of a disease; but more usually it is employed in a narrower sense, as synonymous with the subjective manifestations of a disorder. Confusion is obviated by using the term "subjective symptoms" when the latter sense is specially intended. Objective symptoms are usually spoken of as *signs;* and those objective symptoms which are made out by physical examination are known as *physical signs*.

Just as the value and significance of physical signs depend on the skill and experience of the physician who observes them, so the significance of subjective symptoms has to be weighed and considered in relation to the character and constitution of the patient who complains of them. Thus a certain symptom may appear trivial and unimportant to a patient of strong character not addicted to introspection, although serious disease may be present; whereas in a delicate woman with a susceptible nervous system every subjective symptom, however slight, may be to her a cause of great anxiety or exaggeration, and even real

[1] These words "subjective" and "objective" are borrowed from philosophy. Subjective reality is reality which exists in the mind only, whereas objective reality is that which can be demonstrated by means of tangible, visible, or outward signs.

suffering. Submammary pain, for instance, in the first might indicate aneurysm ; in the second, hysteria.

General (or constitutional) symptoms are those which relate to the whole body, such as debility or pyrexia. By some writers the term is used solely to indicate pyrexia and its concomitant symptoms, such as headache and general malaise.

A *latent disease* is one which is unattended by any very obvious symptoms. Thus, we speak of latent pulmonary tuberculosis when a patient suffering from tuberculosis of the lung has none of the more usual and constant symptoms of that disorder, such as cough, dyspnœa, hæmoptysis, and dulness on percussion. Physical signs are not necessarily absent in latent disease, but they are often difficult to detect. Some writers speak of a malady as being latent when the pain, which is usually a prominent feature of the disease, is absent. Thus, pericarditis is ordinarily attended by a good deal of pain, but this is absent in the latent form of pericarditis which frequently complicates rheumatic fever, and in the latent peritonitis which complicates enteric fever.

A *paroxysmal disorder* is one which comes on in the form of attacks separated by intervals of comparative health. Each attack or paroxysm consists of a stage of invasion (usually more or less sudden), leading to an acme, and followed by a gradual decline in the severity of the symptoms. As instances of paroxysmal disorders may be mentioned Paroxysmal Tachycardia, Angina Pectoris, Epilepsy, Nervous Faints and Flush Storms, and Paroxysmal Hæmoglobinuria.

The clinical features which all paroxysmal disorders present, and a close study of those features which admit of a pathological explanation, point to the sympathetic system, and especially the vaso-motor portion of it, as being probably at fault in such cases.

The student will often meet with the termination -ITIS, and it is well to remember that it signifies "inflammation of."

§ 2. **Case-Taking.**—In clinical investigation, or case-taking, our object is, *first*, to elicit all the data of the case ; and, *secondly*, by reasoning based on those data to arrive at its Diagnosis, Prognosis, and Treatment. It will be found in actual practice that everything turns on the diagnosis ; that is our first and principal object ; the prognosis and treatment follow from this.

The investigation of a case consists of two parts : (A) The Interrogation of the Patient, and (B) the Physical Examination. Students should always accustom themselves to learn all that is possible by interrogation before proceeding to the physical examination.

A. By **Interrogation of the Patient** we learn—

(*a*) What is his *chief* or dominant symptom ;
(*b*) The facts concerning the *present illness ;*
(*c*) The patient's *previous history ;* and
(*d*) His *family history.*

Throughout the interrogation of the patient it is well to follow THREE GENERAL RULES :

(1) *Avoid putting what barristers call "leading questions"—i.e.*, questions which suggest their own answer—*e.g.*, "Have you had a pain in the back ?" suggests an obvious answer to the patient. It might be put thus : "Have you had any pain, and if so, where ?" The patient should be encouraged to tell his own story, without interruption. Moreover, the very words he uses should be recorded in inverted commas, and on no account should the words of the patient be translated into scientific terms. Some say that leading questions are permissible when the patient is very ignorant and stupid, but these are the very cases in which leading questions should be specially avoided. The only legitimate way of putting a leading question is in an alternate form—*e.g.*, "Have you suffered from diarrhœa or constipation ?" Time, patience, and tact are necessary to elicit the true facts of the case, without irrelevant detail. Our object is to learn what the patient *feels* and knows, not what he *thinks* of his disease ; and our patience is often sorely tried by a long story of his own or his previous doctors' views on his case. Our record should be comprehensive, including all important data, negative as well as positive, yet concise—*i.e.*, excluding irrelevant facts. Only experience and a knowledge of medicine can teach us what is or is not relevant. The beginner, however, should strive after completeness rather than conciseness.

(2) A *chronological order* should always be adopted, both in eliciting and in recording the facts. Nothing is more wearisome than to wade through a mass of verbiage which mixes up dates. Dates should be recorded always in the same terms. It is very common, for instance, to read in students' reports that "breathlessness began in the year 1892," "palpitation started when the patient was aged forty," "the dropsy came on three years ago."

(3) Always adopt a kindly and *sympathetic manner*. Not only is it our bounden duty to be considerate and patient with those who suffer, but by entering into the spirit of the patient's sufferings we can often get at more important facts, and a truer narration of them, than can one whose harsh or abrupt manner causes the patient to shrink up into his shell like an oyster. Put your questions in as simple and non-technical a form as possible, and be sure that the patient attaches the same meaning to the words as you do. Much will depend on the tact of the physician, and two very good rules may here be added—viz., Never put questions bearing on venereal disease before the husband or wife of the patient ; never inquire concerning a family history of consumption or cancer before a patient whose illness is likely to be of that nature.

(*a*) THE CHIEF OR CARDINAL SYMPTOM.—The first question to ask a patient should always be the same : "What do you complain of ?" Special attention should be paid to the symptom for which the patient

seeks advice or is admitted to hospital, because it is this symptom which guides most of our subsequent inquiries. It should always, as far as possible, be recorded in the patient's own words. *This book is based upon the patient's cardinal symptom;* and in the following chapters I shall, after each cardinal symptom, allude to the principal conditions for which it may be mistaken. The best way to avoid error in this respect is to verify your observations by repeating your examination again and again.

(*b*) History of the Present Illness.—It is better to investigate the present illness first, before the previous and family histories, because it is closely connected with the patient's disease, and because we are tracing backwards from effect to cause. A good question to start with is: "When did you cease work?" or, "When were you last quite well?" *Remember that the present illness dates from this to the time when the patient came under observation.* Our questions should be directed to ascertaining three facts of importance—viz., the precise manner of commencement of the disease, whether sudden or gradual; the date when the patient ceased to work; and the date when he took to his bed. Then the evolution of symptoms can be traced step by step in the order of their development—always in chronological order. It is useful also to know whether he has recently been, or is now, under medical treatment, not only because the symptoms may have been modified in this way, but also because one of the most important ethical principles of the medical profession may be involved.[1] In all of these inquiries the three general rules above given apply (p. 3).

(*c*) The Previous History of the patient bears largely on the etiology, or *causation*, of his illness, and comprises two orders of facts—viz., (1) those relating to any *illnesses* the patient may have had before the present one; (2) those concerning his personal *habits and surroundings.*

(1) Under the former heading, note in chronological order all ailments the patient has suffered from prior to the present one, with the dates of their occurrence and their duration—*e.g.*, contagious diseases of childhood; and especially such ailments as venereal disease, rheumatism, and gout. If the attacks have been at all obscure, it is desirable to add a few of the leading symptoms to prove the nature of the alleged attacks, and in such instances inverted commas should be freely used. For instance, "rheumatism" is a vague term which may mean any disease attended by pains in the limbs, such as alcoholism, syphilis,

[1] By-law CLXXV. of the Royal College of Physicians of London runs as follows: "No Fellow, Member, or Licentiate of the College shall officiously, or under colour of a benevolent purpose, offer medical aid to, or prescribe for, any patient whom he knows to be under the care of another legally qualified Medical Practitioner." This is perhaps the most important guiding principle in the ethics and etiquette of the medical profession. On the other hand, this law gives us no proprietary right in a patient because we have once prescribed for him or his family. He ceases to be our patient directly he ceases our treatment for that particular ailment.

tabes dorsalis, or neurasthenia. The subject of syphilis should always be approached with delicacy in the case of women. Indirect information may often be gained by inquiring for prolonged sore throat, followed by loss of hair, by eruptions, or pains or swellings of the cranial or other bones. In married women a *series* of miscarriages or still-births, or children born with eruptions or snuffles, may have the same significance.

(2) The occupation, home surroundings, and previous habits as regards exercise and food should be inquired into. The daily amount of alcohol taken, and its kind (wine, beer, or spirits), should always be noted ; and also the *time of day at which it was taken*, because far more harm may be done by " nips " between meals (without ever getting actually drunk) than by ten times the quantity taken with meals. Finally, we should ascertain whether the patient has resided abroad, especially in malarial districts. In females, the previous state of the catamenia, and the number of pregnancies, miscarriages, or still-births, should be noted.

(*d*) THE FAMILY HISTORY may, like the previous history, have a causal relationship to the patient's malady. The age and state of health if living, age and cause of death if dead, of near blood relations, should always be noted—*i.e.*, father and mother, brothers and sisters, sons and daughters. Inquiry should also be made as to whether any members of the family (parents, grandparents, brothers, sisters, uncles, aunts, or cousins) have suffered from consumption, cancer, acute rheumatism, gout, nerve diseases, insanity, asthma, heart disease, apoplexy, and especially those diseases to which the patient himself seems liable.

B. The **Physical Examination** (*i.e.*, the State on Admission, or the Present Condition) may with advantage be prefaced by a few general remarks on how and what to observe.

(1) Here, again, having learned by interrogation our patient's chief complaint, we should ask ourselves, IS THERE ANY STRIKING OR PREDOMINANT SIGN OR APPEARANCE (Latin *facies*) ? The importance of INSPECTING our patient cannot be overestimated. In these days of scientific instruments we are too apt to forget the use of our faculties. By simply using our eyes many important data may be learned besides the colour of the skin, the general nutrition, the attitude or decubitus, and the facial expression. For instance, the manner in which a patient answers questions is often the first clue to hysteria, and a peculiar mode of speech is one of the pathognomonic symptoms of general paralysis of the insane, disseminated sclerosis, and other diseases. Moreover, with experience we can by this means form a conclusion as to the kind of patient we have to deal with. Again, never be in a hurry ; it is only by taking time that we can fully appreciate all the points presented to our view. This habit of " observing " the patient is only developed by

long practice; but it will never be developed if the young physician allows himself to be infected by the hurry of modern times.

(2) It is important always to commence our examination with that ORGAN TO WHICH THE SYMPTOMS ARE MAINLY REFERABLE. Some teachers direct their pupils to examine and report on the physiological systems always in the same order (first the heart, then the lungs, then the digestive system, and so forth), whatever may be the malady. But such a course has, to my mind, three objections: (i.) The student goes about his work in a mechanical fashion; (ii.) if the patient suffer from some serious disorder, such as peritonitis, he may be seriously injured by a thorough investigation of the chest and other parts; and (iii.) in many cases it is a waste of time to examine all the organs with equal thoroughness. The same educational advantages and experience can be obtained by the other method, and in that way we come to the most important facts first. As a general rule, the most important data should be mentioned first.

(3) In all cases EVERY ORGAN IN THE BODY SHOULD BE CAREFULLY EXAMINED; for although we may find in one physiological system sufficient mischief to account for the patient's symptoms, the other organs may reveal changes which considerably modify our treatment, our prognosis, and even our diagnosis. Whatever order is adopted, the student should not wander from organ to organ, but examine each physiological system thoroughly before proceeding to the next. It is well to get into the habit of adopting some such order of physical examination as the following: *First*, note the general condition; *secondly*, examine the organ chiefly affected; *thirdly*, the other organs in the following order: Thorax (heart and lungs), Abdomen (alimentary canal, liver, spleen, and genito-urinary system), Head and Limbs (nervous and locomotor apparatus). Further details are given in the scheme below, but for the thorough investigation of the organ chiefly affected reference must be made to the chapter dealing with the diseases of that organ.

The examination should always be carried out *gently*, and *without undue exposure*. In serious cases, especially when the heart or lungs are involved, it may be well to postpone a thorough examination of some organs, so as not to risk harming the patient by making him sit up. At the same time, the young physician should never allow modesty to prevent his making a thorough examination. This rule is especially necessary in patients of the better class, but a little firmness, tact, and a courteous demeanour will generally enable him to perform what is a duty both to himself and his patient.

SCHEME OF CASE-TAKING.

A. INTERROGATION OF PATIENT.

(*a*) The patient's chief or **Cardinal Symptom.**
(*b*) Data concerning the **Present Illness.**
(*c*) The patient's **Previous History.**
(*d*) The **Family History.**

B. PHYSICAL EXAMINATION (i.e., Present Condition—Give Date).[1]

(*a*) **The general condition** may be summarised mainly under three headings: (i.) The Physiognomy or expression (especially in acute disease (Chapter II.); (ii.) The Decubitus, Attitude, or Gait, especially in chronic disorders (Chapter II.); (iii.) The Nutrition, General Conformation, and any Eruption on the Skin (Chapter XVIII.). The temperature should be taken; and any bed-sores noted.

(*b*) **Chest.**

I. Cardio-Vascular System. (Chapters III. to V.)

Symptoms.—Breathlessness, palpitation, cardiac pain.

Physical Signs.—Pulse: rate, rhythm, tension, arterial wall. Heart: apex-beat, percussion area, auscultation, dropsy.

II. Respiratory System. (Chapters VI. and VII.)

Symptoms.—Cough, expectoration, dyspnœa, pain in chest.

Physical Signs.—Rate of respiration, inspection, percussion, auscultation, palpation.

Examine throat and nose.

(*c*) **Abdomen.**

III. Alimentary Canal. (Chapters VIII., IX., X., and XI.)

Symptoms.—Appetite, discomfort after food, nausea, pain, state of the bowels, colour of motions.

Physical Signs.—Examine mouth and tongue. Physical condition of abdomen as regards distension, and presence of fluid or tumour (inspection, palpation, and percussion).

[1] This scheme gives only the *chief points* which should be noted about the different physiological systems, with the object of excluding disease. For an exhaustive examination, such as must be made of the organ to which the patient's symptoms are mainly referable, the student should refer to the chapter dealing with the diseases of that organ.

IV. Liver. (Chapter XII.)

Symptoms.—Pain, jaundice.

Physical Signs.—Size (palpation and percussion), surface (if accessible), tenderness.

V. Spleen. (Chapter XII.)

Any enlargement (palpation and percussion) or local pain.

VI. Urinary System. (Chapter XIII.)

Symptoms.—Any undue frequency of, or difficulty in, micturition. Any dropsy.

Physical Signs—

(i.) *Urine:* quantity, colour, reaction, specific gravity, albumen, blood, sugar, deposit (microscopical examination).

(ii.) *Kidney.*—Any enlargement, mobility, or tenderness.

VII. Generative System. (Chapter XIV.)

Menstruation, frequency, duration, quantity, intermenstrual discharge.

(*d*) **Head and Limbs.**

VIII. Nervous System. (Chapter XIX.)

Symptoms.—Intelligence, sleep, neuralgia, etc.

Physical Signs.—*Muscles:* paralysis, spasm, tremor, character of walk. *Reflexes*, deep and superficial.

Sensation for touch, pain, temperature.

Cranial Nerves.—Vision, pupils, movements of eyes, fundi. Movements of face, tongue, and palate. Hearing. Smell. Taste.

Sympathetic System.—Flush storms, trophic lesions, obscure sensations.

(*e*) **Blood.**

In anæmic and some other cases the blood must be examined (Chapter XVI.).

Progress of Case.—Notes (daily of acute or febrile cases, twice a week of subacute, and once a week of chronic cases) should be made of the progress of the case; and much care is required here to avoid redundancy on the one hand, and on the other to record completely all important changes, or any fresh symptoms, and the effect of the treatment adopted. In acute febrile cases there ought to be a daily note, and the pulse, respiration, and temperature should be noted

several times daily. In chronic cases it will be sufficient to note, once a week, the persistence of the prominent symptoms or any change in the symptoms. In all cases any *sudden* change in the patient's symptoms or general condition should be noticed at once. Each note should have special reference to the previous one ; and before taking a fresh note, the previous one should be read over. The treatment and its effects should always be incorporated ; thus, if the patient has been ordered diaphoretics or purgatives, record should be made of the state of the skin or bowels.

History Sheets, Charts, Diagrams, etc.—A history sheet for recording the history of a patient should be ruled with one vertical line down the page one-third from the left-hand margin, so as to give space for information learned subsequently. It should have printed headings and spaces at the top, thus :

Diagnosis. (Space here for primary and secondary disease, filled in by physician afterwards.)

Name ..... **Age** ..... **Sex** ..... **Occupation** .....

Address ..... **Date of admission** .....

Chief symptom on admission .....

Date of discharge .....

Temperature charts are of the greatest use to record the temperature and other features of diurnal variation.

Outline diagrams of the various regions of the body are now prepared, and are very useful.

A kind of shorthand code for physical signs is advocated by some authors, and, when once learned, may be useful in saving time and space.

§ 3. Examination of Children and Infants.—Here the same general rules apply as to interrogation and physical examination, and we should first endeavour to ascertain the child's leading symptom, either from the patient or its relatives. There are, however, certain additional rules upon the adoption of which much of our success with children will depend.

1. First endeavour to establish friendly relations with your little patient. This may be done sometimes by appearing not to notice the child when you first enter the room ; after a while it may make advances and investigate your watch-chain or ring. Time should always be given for the child to become accustomed to your presence, and anything like abruptness will defeat your aim.

2. The questions put to the child should always be of the simplest character—*e.g.*, " Where does it hurt you ?" From the mother you may learn the age up to which the child remained healthy, the symptoms of the present and previous illnesses. In the case of an infant ask whether it was a full-time child, if born with instrumental aid, whether it was born healthy, or whether it developed a rash or " snuffles," and whether breast or bottle fed. If the child is past early infancy, the same questions may still be put, and in addition inquire when it began to walk, and when dentition commenced. Carefully inquire as to its present and past diet, as to its appetite, and the state of the bowels. Ask also how long it sleeps, bearing in mind that children require much more sleep than adults. Then inquire for any recent illness in other members of the family.

Physical Examination.—Valuable as *attentive observation* may be with adults, it becomes quite indispensable with children, who cannot accurately describe their sensations. Much may be learned while you sit and allow the child to get

accustomed to your presence. Notice its expression, the brightness of its eyes, its attitude, the colour of its skin, the state of nutrition, its size as compared with age, its movements, the condition of its lips (moist or dry), the character of the breathing, the sound of its voice. If it cries, inquiry should be made whether this is constant or only at times. Congenital syphilis may be plainly depicted on its face or skin. If the child be asleep when first you enter, do not wake it, but notice all the above before it is disturbed. The limbs of a healthy child should be constantly on the move; drowsiness, dulness, and listlessness are signs of pyrexia, and especially that of the contagious fevers. The hands are instinctively moved towards a seat of pain—*e.g.*, the head in meningitis. The state of the temper is altered in the prodromal stage of most diseases; but it is markedly peevish in the prodromal stage of meningitis. For other facial alterations, see **Facies** (§ 12). When the child is undressed for examination, the back of the chest should be examined first, while the child looks over the mother's shoulder at someone who attracts its attention with a bright object or a bunch of keys. The binaural stethoscope is the most useful under these circumstances.

§ **4. Methods of Diagnosis, Prognosis, and Treatment.**—Diagnosis, prognosis, and treatment are the objects we had in view in eliciting all the facts concerning the patient by the process of "Case-taking." Of these three, **Diagnosis**—which, as the Greek word (διαγνωσις) implies, means the distinguishing or discernment of the disease—is by far the most important. Everything necessarily hinges on that, because without the recognition of the disease, rational prognosis and treatment are impossible. It will be well, therefore, to consider how the data we have elicited may be utilised in order to arrive at a diagnosis. Several different methods are employed:

The method usually adopted, which is the outcome of the student's studies in systematic medicine, is to erect a *hypothetical diagnosis*, and to see whether the patient's symptoms tally with the description of the disease. When a child, for instance, with disorderly movements comes before us, the diagnosis of chorea at once occurs to our minds. The age of the patient, character of the movements, and all the obvious features of the case appear to correspond with that disorder. It does not seem necessary to consider any other suggestion. This method answers well enough in straightforward, well-marked, typical cases; but in cases presenting anything unusual or atypical considerable difficulty may be experienced.

Another method of making a diagnosis is by a *process of exclusion;* that is, after studying the diseases which might possibly be in operation, we arrive at our diagnosis by excluding those which the disease least resembles. In such diseases as enteric fever, where the symptoms are few in number, this may be the only method possible. The patient, for instance, is suffering from a moderate degree of pyrexia, the illness came on gradually; that is all we may know about the case. There are many possible causes of such a condition, but we arrive at the conclusion that it is probably enteric fever, because all the other possible diseases are rendered improbable for one reason or another.

The third method consists of *balancing the evidence* for and against

all the possible causes (clinical or pathological) which might give rise to the patient's leading or cardinal symptom (such as headache, albuminuria, or vomiting). In this method, after having elicited all the facts of the case, we return to the patient's *cardinal symptom*, enumerate in our own minds the various causes which might give rise to that symptom, and balance the evidence adduced by the other facts of the case for and against each one in turn. It may strike some as being a little tedious, but it is not so when we have got into the habit of employing it. It is certainly the one best adapted for the elucidation of obscure or atypical cases ; and under all circumstances it presents a truer picture to our mind, because diagnosis can never be a matter of absolute certainty. At most a diagnosis is only a strong probability, and this method enables us to ascertain the exact amount of probability in each disease. Even in the simplest and most typical cases it is a good mental exercise for us to keep in mind the other lesions which might produce the same symptom, and then we are always on the look-out for possible errors, and ready at any moment to review the diagnosis—a correct mental attitude when in presence of nature's phenomena. The chapters which follow are based on this method.

EXAMPLE.—Let us suppose, for instance, that the patient, a pale young woman, aged twenty-three, comes to us complaining of **vomiting blood** (*i.e.*, hæmatemesis).

First, we ascertain and verify this, the leading symptom, and find that she has really vomited a considerable quantity of blood.

Secondly, we INTERROGATE her as to the history of her present illness, her previous and family histories, and we find that she has suffered for several years from symptoms pointing to dyspepsia, and that latterly there has been severe pain in the epigastrium. There are always four features we have to investigate about every pain—its position, character, degree, and constancy ; and we find that this epigastric pain is a sharp pain, not constant, but coming on shortly after taking food, and that it is followed *and relieved* by vomiting. The other details of the case we will omit for the sake of brevity.

Thirdly, we proceed to the PHYSICAL EXAMINATION, first of the abdominal organs, but this reveals nothing abnormal. Then we go through the other physiological systems in order, observing (*a*) her General Condition (noting, for example, how pale and thin she is, and how weak she seems) ; (*b*) examining the Chest (cardio-vascular and respiratory systems) ; (*c*) the Head and Limbs (nervous system) ; (*d*) the Blood must also be examined, because anæmia (poverty of blood) may be inferred from the pallor of her skin.

Having elicited all the data (taken the case) by interrogation and physical examination, we return to the *cardinal symptom*—hæmatemesis [1]—and consider its various causes (see the section on Hæmatemesis) *seriatim*, taking the most probable cause in this case first.

[1] Here there was no difficulty about identifying or selecting which was the chief or most important symptom ; but in another case the anæmia (or the vomiting or epigastric pain) might be the more serious or prominent symptom, the hæmatemesis consisting, perhaps, of a few streaks of blood. Then we should deal with the anæmia in the same way as hæmatemesis is here dealt with. Sometimes a good deal depends upon our choice of which is the "leading symptom," for it is not always the most prominent which is the most serious and important ; and by an error in this respect we may be led far afield of the true disease. Sometimes, however, it is useful to change the point of view we take of a case, by regarding it from another standpoint or leading symptom.

(*a*) Simple Ulcer of the Stomach.

For : (i.) The profuseness of the hæmatemesis ; (ii.) the character of the pain (brought on by food, relieved by vomiting) ; (iii.) the history of dyspepsia ; (iv.) the age and sex of the patient.

Against : (i.) No tenderness in the epigastrium.

(*b*) Cancer of the Stomach.

For : (i.) The vomiting of blood ; (ii.) pain in the stomach ; (iii.) pallor and emaciation ; and so on.

Against : (i.) The blood vomited was too profuse, and had not the character special to cancer (coffee grounds) ; (ii.) the pain was only produced by food, and entirely disappeared after vomiting ; (iii.) age of patient much too young.

(*c*) Portal Obstruction.

For : (i.) The profuseness of the hæmatemesis.

Against : (i.) Absence of abnormal signs in the liver ; (ii.) absence of ascites, piles, and other symptoms of portal obstruction.

(*d*) Other and less probable diagnoses can be discussed in like manner, though each of these may be more summarily dismissed thus : *Vicarious menstruation* would not account for the dyspepsia, acute epigastric pain, and other symptoms. *Leucocythæmia, Scurvy,* and *other blood conditions,* if present, would present the other symptoms of those maladies ; and so on.

It follows, therefore, that the balance of evidence is in favour of (*a*) Simple Ulcer of the Stomach, partly because of the weighty arguments in its favour, and partly because the only argument against it is not vital, for tenderness may be absent when the ulcer is situated on the posterior wall of the stomach. Indeed, if a numerical value were given to each of the " reasons " for and against, it would be possible to express the precise degree of probability in each disease in the form of a mathematical ratio. This method may at first sight seem tedious, but after a little practice it becomes automatic and extremely simple ; and it takes much less time than is here implied.

Prognosis (from the Greek word προγνωσις) is a " foreknowledge " of the events which will happen—*i.e.*, of the probable course the disease will run. Nothing but wide experience, combined with careful and minute observation, will enable a physician to prophesy with any approach to accuracy. It will, however, be useful to bear in mind that the prognosis of a case depends upon four circumstances—viz., (1) the *usual course,* duration, and event of the disease in operation (phthisis, for instance, runs a prolonged course, and until lately the event was almost invariably fatal) ; (2) the presence or absence of *untoward symptoms* (*e.g.*, profuse hæmoptysis in phthisis) ; (3) the presence or absence of *complications* (which are sometimes more fatal than the disease itself—*e.g.*, enteric and many other fevers are fatal chiefly by their complications) ; and (4) the *causes* which are in operation, including among the predisposing causes such data as age and sex (bronchitis, for example, in middle life is not a serious affection, but in infancy and old age it is one of the most fatal diseases in the Registrar-General's returns). More reputations are wrecked on the rock " Prognosis " than on any other.

As practical hints to the young physician, I would advise him—(1) Never to commit himself to a prognosis unasked, or before the effects of

treatment have been noted. (2) It is also well to impress upon the friends that a "physician" cannot hope to be also a "prophet"; and that prognosis may depend on many factors in the case which are not yet revealed. The medical work in connection with life insurance is largely a question of prognosis.

Treatment is what the patient comes to us for; and it may be of three kinds: (1) In *Radical* treatment (also called Curative or Rational) our object is to cure the patient of his disease by the removal of the cause. This is the only truly scientific treatment, and it is based mainly upon a knowledge of the pathology of the malady. (2) *Symptomatic* treatment is directed only to the relief of the symptoms. In some incurable maladies symptomatic treatment is the only kind that is possible, and all that we can do is to ease the passage to the grave. But in the practice of busy practitioners, the trouble and time needed for thorough investigation often lead to the adoption of the latter at times when a more radical treatment would be possible. There is an unfortunate tendency to fall into a routine of symptomatic treatment which we should constantly guard against. Both Radical and Symptomatic treatment may be either internal or external on the one hand, and either medicinal or dietetic and hygienic on the other. (3) *Preventive* treatment has within the last quarter of a century developed almost into a separate science, the science of Hygiene or State Medicine.

§ **5. General Rules in Clinical Investigation.**—There are certain habits which the student should strive to cultivate when he comes to the practical aspect of his profession; and he should remember Thackeray's saying: "Sow an act and you reap a habit; sow a habit and you reap a character; sow a character and you reap a destiny." Clinical medicine depends more than anything else on accurate, complete, and well-directed observation, and there are five hints I would give to the student in this connection.

1. *Avoid superficiality* in your observations. Do not try to see many cases in one day, but rather one or two cases *continuously from day to day*, so that you may follow a given malady throughout its entire course. It is of more value to follow up one case in this way than to see a dozen on one occasion only. Practical knowledge must be acquired gradually. The thought will often occur to the student how slowly he progresses with his clinical knowledge. This is partly real, partly apparent. It is partly apparent because a student does not realise at the time the value he derives from listening, for example, to the same cardiac murmur over and over again. It is partly real because it is only by patiently devoting the necessary time to the study of the same case from day to day that he will learn to make his observations adequate, thorough, and precise. That is why many a brilliant intellect falls

behind, and many a plodder comes to the front in our profession. It is vain to attempt to substitute genius for patient industry in this arena. You must learn for yourself the effects of this or that line of treatment; learn to correct and control the observations you make one day by your observations of the morrow; and above all, try to learn what is the sequel or termination of the case, especially in such instances as may lead you to the dead-house. There, more than anywhere else, the most brilliant diagnosticians learn from their own errors more than from a multitude of successful cases.

2. *Do not strive after what is rare and curious.* It follows, as a matter of course, that, other things being equal, a fact is more important in proportion as it is common. Moreover, by studying *only* the exceptions to a rule, our minds will have a distorted view of clinical phenomena. Do not, therefore, be led astray by those pedants who seek after the singular and uncommon. It is well to see rare cases when the opportunity offers, by all means, but be careful that you mentally register them as rare.

3. *Do not study only acute and severe cases.* It is true that in acute diseases there is often more to be done, more heroic and decisive effects to be produced, or apparently produced, and therefore more credit and renown to be obtained. But we shall find in actual practice not one-tenth, perhaps not one-hundredth of our patients will be suffering from these complaints. Our success, therefore, in practice, whether measured by that laudable satisfaction at having done one's duty, or by the pecuniary reward of which every earnest labourer is worthy, will depend very much on our experience of, and our ability to treat, chronic and what we are too apt to call trivial complaints. For one case of Graves' or Addison's disease, the student will, I venture to think, have a hundred cases of dyspepsia, chronic rheumatism, or chronic bronchitis. In the treatment of such complaints the greatest judgment and thoroughness are sometimes needed. No sudden or startling effects can be produced. Chronic diseases require chronic remedies, and it is only by experience that one can learn to produce those gradual effects which lead to a successful issue.

4. *Be accurate in your observations.* State facts precisely as you find them, no matter whether they accord with your hypothesis or not; and state only what you find and know to be the truth. The study of clinical medicine, like the study of any other of Nature's phenomena, should inculcate in the mind of the student a love of truth. It is impossible to have any dealings with Nature without learning that truth is the key to the discovery of her secrets. Accuracy is one form of truth, and it is only by repeatedly going over your observations, and sifting the patient's statements, that you can insure accuracy.

5. *Be systematic in the arrangement of your data,* for it is only by a systematic arrangement that you can attach the proper significance

and importance to each, and get a firm grasp of the whole case. Nothing, for instance, is more liable to confuse and to prevent you from coming to a correct conclusion than wandering from one date to another without regard to the chronological sequence in the history of an illness. And again, in physical examination, nothing is so likely to lead you astray as wandering from organ to organ without first completing the examination of each.

§ **6. Classification of Diseases—Method of Procedure.**—It has been customary, and the practice is convenient, to classify diseases into two great groups—Constitutional and Local. LOCAL diseases are those in which the principal, and perhaps the only, lesion is localised in one organ or situation. CONSTITUTIONAL diseases are those which affect the blood, and therefore the whole of the body ; a considerable number of these are now known to be microbic in origin. As illustrative of the latter, acute rheumatism, typhoid fever, pyæmia, and anæmia may be mentioned ; of the former, iritis, synovitis, and myelitis.

It is convenient for clinical purposes to preserve this division, but the rapid advance of pathology has gradually transferred disorders from the " local " to the " constitutional " group. A large number of diseases formerly believed to be lesions of local origin (such, for instance, as pneumonia, endocarditis, and peritonitis) are now known to be due to some general morbid process, toxic or microbic, having a special local manifestation.

From a pathological standpoint diseases are sometimes divided into two groups —Organic, those in which some anatomical change is found after death ; and Functional, those in which no structural alteration is found. The anatomical or structural change is spoken of as the " lesion." The word " functional " must not be regarded as synonymous with " hysterical."

Now it so happens that local disorders are very often met with as complications or effects of constitutional or general conditions ; and since in clinical work we are engaged in **tracing from effect to cause**, we shall, in the following chapters, take the local diseases which are manifested by a lesion *localised* in some particular organ first, and the *constitutional* conditions afterwards.

When a patient applies to us, if, as the result of our inquiries, we find he is suffering from a symptom localised to some organ (*e.g.*, pain in the liver), turn to the chapters relating to the diseases of the organ (one of the Chapters III. to XIV.).

If, on the other hand, he has no localised symptom, but complains of malaise, feverishness, or a sense of " bodily illness," turn to the chapters on constitutional diseases (Chapters XV. to XIX.).

CHAPTER II

THE FACIES, OR EXTERNAL APPEARANCE OF DISEASE [1]

In our scheme of case-taking it will be remembered that the first step in physical examination was to observe the patient's general condition ; and it will also be remembered how great was the importance of an adequate inspection of the patient while he was telling us the story of his illness.

Some diseases can be identified almost at a glance, before the patient opens his lips, such, for instance, as Chronic Alcoholism, some manifestations of Hereditary Syphilis, Graves' Disease, Cretinism, Myxœdema, Facial Paralysis, and Hydrocephalus, when these conditions have passed beyond the incipient stage. The existence of others can be very strongly suspected, such as Rickets, Postnasal Adenoids (mouth-breathing children), and Chronic Bronchitis with Dilated Right Heart.

But, apart from these, much may be learned from the first glance at a patient—from his *decubitus* (the way in which he lies), from his *attitude* or gait (if he be able to leave his bed), from the expression of his *face*, the colour of his *skin*, and from the *general conformation* of his body—without the employment of any special methods of apparatus for diagnosis. It is to be feared that as scientific methods become more and more perfect, these means, which constitute one of the most useful and important aids to diagnosis and prognosis to the experienced busy practitioner, are apt to be neglected. But, on the other hand, students and young practitioners had better not attempt " lightning diagnoses," or they will certainly fall into the most serious errors. Some men, it is true, like the late Dr. Sibson, seem to be specially gifted in this way ; but it is only by long experience and the possession of special faculties that they can accomplish such feats.

It is a fundamental rule that your patient should face the light at all medical interviews. Similarly your own chair should be in the shade, lest the patient should read too readily what is passing through your mind. It is surprising what important clues can be obtained by an intelligent inspection of your patient, both as to his character and his disease.

[1] The Latin word *facies* signifies an appearance, form, or shape.

The facies of disease may be summarised under three headings: (A) The Physiognomy of Disease. (B) The Decubitus, Attitude, or Gait. (C) Alterations in the General Conformation of the Body.

Hints to be derived from an inspection of the hands are given under Diseases of the Extremities (Chapter XVII.). The various diseases will be only mentioned here. The description and differentiation of the several affections referred to will be entered into more fully in the chapters which follow.

(A) The Physiognomy in Disease.

An observant physician can obtain important clues to diagnosis by the physiognomy—*i.e.*, the aspect and expression of the patient's face—even apart from the insight which can be gained by this means into his character.[1]

§ 7. In **Acute Diseases** more can be learned from the position in which the patient lies (*i.e.*, his Decubitus, § 14) than from the physiognomy or expression of his face. But it is worth remembering that the face assumes an *anxious expression*, which is very characteristic in pericarditis, peritonitis, and severe pneumonia, also during attacks of angina pectoris. The supervention of *acute pericarditis* in the course of rheumatic fever is often unsuspected, as there may be no local symptoms; but it may be recognised by this anxious expression, the dilated nostrils, and the flush upon the cheeks, which were (probably) at our last visit so pale. In acute *croupous pneumonia*, again, the appearance is very distinctive. The flushed face, hot dry skin, widely dilating nostrils, the eruption of herpes beside the mouth, and the profound disturbance of the pulse-respiration ratio (1 : 2 instead of 1 : 4, which is the normal), form a picture which greatly aids the recognition of the disease. The *Facies Hippocratica*—a facies or appearance, of which the description has been handed down from Hippocrates—is the forerunner of death from exhaustion, such, for instance, as the final stage of cholera, and wasting disorders. The temples are hollow, the eyes sunken, the eyelids slightly parted, the eyes glazed, and the lower jaw droops. The *Risus Sardonicus* is a fixed grin, met with typically in tetanus. The corners of the mouth, which twitch at intervals, are drawn upwards as in laughter, and the features assume a fixed sarcastic expression. In several abdominal disorders, such as peritonitis, a similar expression occurs, in which the curved folds stretching from the alæ nasi to the corners of the mouth become marked.

§ 8. A few **Chronic Diseases** may be enumerated in which the physiognomy is characteristic.

(i.) The aspect of a *phthisical* or *tuberculous* patient differs in the

[1] Those who are interested in this method of studying character should consult "Essays on Physiognomy," by Lavater, translated by Thomas Holcroft (Ward, Lock and Co., London), 1783.

premonitory and advanced stages. (*a*) Before any evidences can be detected by physical examination of the chest, the patient has the appearance which is loosely described by the laity as "delicate." The skin is fine and soft, and the fresh, rosy colour of the cheeks is out of keeping with the dark rings around the sunken eyes. But it is by the deficient chest measurements and sloping shoulders that the "strumous diathesis," as this tendency or predisposition to tuberculosis is called, makes itself especially manifest. The shoulders slope, and the transverse diameter is deficient in proportion to the antero-posterior (see Chapter VI.). Sometimes such patients are plump and rosy; nevertheless, they have a deficient chest measurement. (*b*) When the disease is advanced, the phthisical patient often presents an appearance that enables the physician to hazard a diagnosis almost without further investigation. The pale, emaciated face, with sunken eyes, the circular crimson flush of hectic fever on the cheeks, the wasted body, bathed from time to time in sweat, the hoarse voice and easily-provoked dyspnœa, collectively form a picture which is very characteristic.

(ii.) *Chronic bronchitis with dilated right heart* is another condition of extremely common occurrence in the practitioner's daily practice, and the picture these patients present is very characteristic. The florid "healthy" looking cheeks, the pulsating jugulars, in a person over forty (more often of the female sex) is very typical.

(iii.) In *chronic alcoholism* there is a puffiness of the face and a congested watery look about the eyes ("a blear-eyed look"). The eyelids are puffy, so that the person is described by sailors as having "an eye like a poached egg." The cheeks and nose are often red, and dotted with stellate venous capillaries. The belly is corpulent; and on holding out the hands and spreading the fingers, they are seen to be affected with fine small rhythmical tremors. The whole picture is unmistakable, though the eyes alone will tell the tale.

§ **9. Swelling of the Face** and neck, if associated with œdema of the limbs and trunk, may be part of the generalised *dropsy of renal disease;* but on account of the looseness of the cellular tissues around the eyelids it is most obvious in that situation. The puffiness of the eyelids due to renal disease is, however, greater in the morning than in the evening, and in this way may be distinguished from a similar condition due to arsenical poisoning or whooping-cough.

A swollen, œdematous condition of the face, accompanied frequently by a troublesome redness, coming on after meals, is a symptom for which *dyspeptic patients* often seek advice. It also forms part of that troublesome condition urticaria factitiosa. A swelling of the face is also apt to occur with different forms of erythema, and is generally worse after meals.

Chronic œdema around the eyelids must not be mistaken for myx-

œdema. It is a not infrequent sequel to recurrent eczema, or repeated attacks of erysipelas, in that situation. It is also met with in nervous or hysterical conditions, and in vaso-motor derangements.

Œdema confined to the head and neck is found in those rare cases where there is pressure on the *veins within the thorax*, especially the superior vena cava, as in cases of mediastinal tumour.

Myxœdema may often be recognised by a glance at the patient's face and hands (Fig. 1). There is a solid œdema and puffiness of the face—

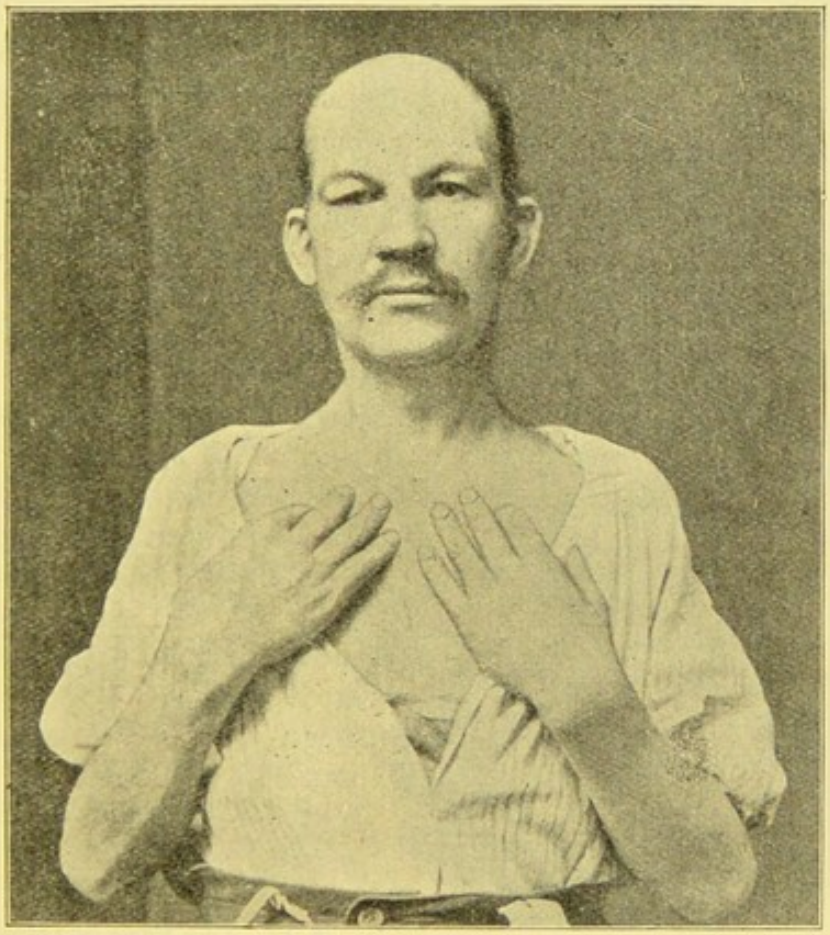

Fig. 1.—MYXŒDEMA.—The patient was a man aged thirty, who was admitted into the Paddington Infirmary in November, 1887, presenting all the usual symptoms of the disease. His movements and mental processes were extremely slow; average temperature 97·2; the quantity of urea passed was less than half the normal. There was a tendency to hæmorrhages (see further particulars in Med. Soc. Proc., vol. xi.).

the body and limbs being also affected—but it does *not pit on pressure*. The vacant, stolid look, flushed cheeks, scanty hair, and slow speech are equally typical of this disorder. The hands are flat, coarse, and swollen (see § 420).

§ **10.** The **Complexion** and colour of the face will repay careful inspection, for thereby the trained observer will acquire some useful hints. Thus, the *pallor* of syphilis or tubercle, and other anæmic conditions,

(Chapter XVI.) is often very striking. So also is the pallor, or rather *sallowness*, of aortic valvular disease; the dead white or *waxen puffy* appearance of parenchymatous nephritis; the *greyish pallor* of chronic interstitial nephritis; the *greenish* colour of chlorosis; the *primrose* colour of that happily much rarer condition, pernicious anæmia.[1] The *orange* colour of jaundice; the *mahogany* colour or *bronzing* of Addison's disease; the *dull earthy* look of malarial cachexia, cancer, and chronic abdominal disease; and, finally, the *purple* (or cyanotic) appearance of the cheeks and lips in mitral and congenital heart disease, are still more distinctive. *Dark rings* around the eyelids appear in states of fatigue; they often indicate want of sleep, or indigestion, and may be so pronounced in malarial conditions as to resemble the ecchymosis of a bruise.

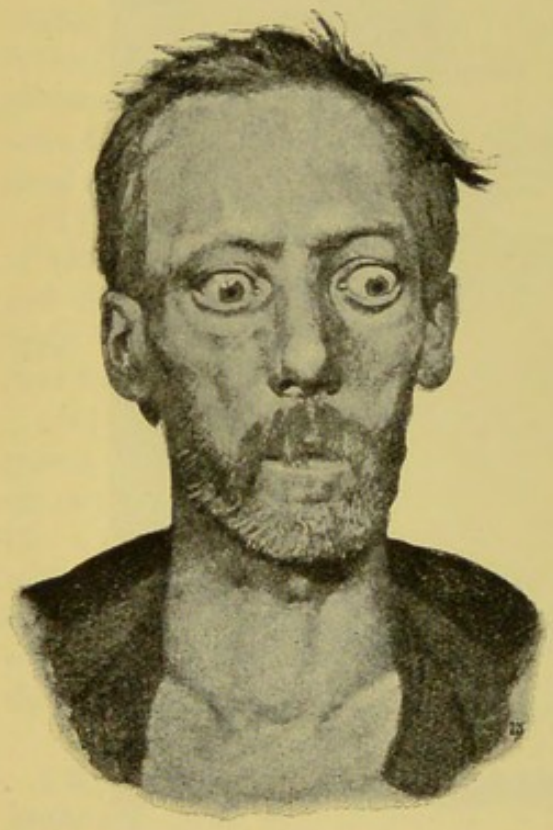

Fig. 2.—Exophthalmic Goitre (Graves' Disease). From Byrom Bramwell's "Atlas of Clinical Medicine."

Seborrhœa oleosa of the scalp gives rise to *greasiness of the face;* and this, with the erythema which frequently accompanies it, produces an appearance somewhat resembling a badly-polished copper kettle. A greasy complexion of this kind is a great affliction to some young and otherwise attractive women, who might easily be rid of it by the cure of the scalp lesion. A *muddy* sallow complexion may be associated with dyspepsia, when the lips are usually dry; but this also may be another result of seborrhœa of the scalp of the kind just mentioned.

§ 11. The **Face in Detail** merits a little closer study, and, first, that most eloquent portion of it, the eyes.

(i.) The *eyes* may be *protuberant* as a whole (Proptosis), as in Graves' disease and intra-ocular tumour (Chapter XIX.). Protrusion of the eyeballs is one of the most constant symptoms of Graves' disease. An equally common sign of this disorder is a goitrous enlargement of the thyroid gland, and therefore the malady is also called Exophthalmic Goitre (see Fig. 2). The eyeballs may *recede* in paralysis of the cervical sympathetic, in wasting diseases, collapse, and the diseases which lead

[1] It is only by long experience that one is enabled to distinguish these refinements of shade.

to collapse.[1] In the *sclerotic*, or white of the eye, the tinge of *jaundice* can often be detected when the yellow colour of the skin is so slight as to escape detection. The "*arcus senilis*" is a white ring of opacity in the cornea, just within its peripheral margin. It is usually believed to indicate senile degeneration of the arteries and other tissues of the body, but I never found it so among the old people in the Paddington Workhouse and Infirmary. In adults who are the subjects of hereditary syphilis, the corneæ may present *striæ*, or the appearance of ground glass (Fig. 3), due to interstitial keratitis. Alterations of the *pupil* are dealt with elsewhere (Chapter XIX.).

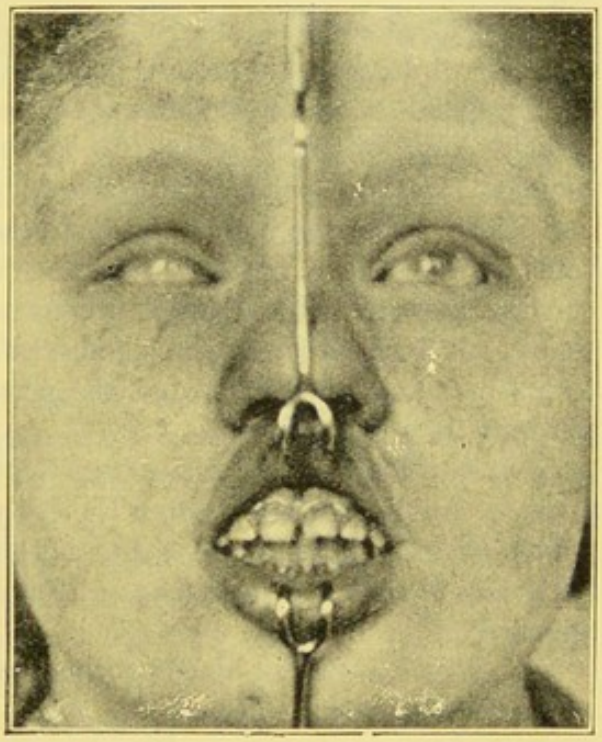

Fig. 3.—HEREDITARY SYPHILIS.—Showing the interstitial keratitis and "pegged teeth" of Hereditary Syphilis.

(ii.) The *lips* may show the pallor of anæmia on the one hand or the congestion of cardiac disease on the other. Fissures and mucous tubercles may indicate that syphilis is in operation. Stellate cicatrices around the lips are a record of previous or hereditary syphilis.

(iii.) The *teeth* also may present the evidences of hereditary syphilis, in which disease, as Hutchinson has pointed out, the permanent incisors (that is to say, when the child has reached the age of seven) are characteristically "pegged"—*i.e.*, narrower at the cutting edge, and notched (see also § 143).

(iv.) Depression of the *bridge of the nose*, if marked, is due to chronic rhinitis in childhood, usually of syphilitic origin. In such cases the nose is characteristically broad and flat, or small and "snub," like a button, the opera-glass nose of Fournier.

§ 12. The **Physiognomy of Childhood** requires considerable experience to appreciate it fully; then it lends us invaluable aid.

(i.) *Congenital syphilis* gives to an infant a very characteristic, pinched, wan, or "senile" face. The complexion is ashy-grey, the skin is "drawn," and it may be flaky or parchment-like. The eyes and cheeks are so hollow that the nose seems unduly prominent, and thus gives to the infant the appearance of a little wizened old man.

[1] See also Treacher Collins, *Brit. Med. Journ.*, 1899, p. 846.

(ii.) When an infant is experiencing *pain* the face will sometimes give a clue as to its situation. Thus, a wrinkling of the forehead or frown is indicative of pain in the head ; a drawing-up of the mouth at the corners, producing marked naso-labial folds, points to severe abdominal pain ; a dilatation of the nostrils and elevation of the eyebrows may suggest intrathoracic discomfort ; and in tabes mesenterica and other chronic wasting diseases the face gradually assumes a fixed or contracted condition, in which the angles of the mouth are depressed.

(iii.) The *fontanelles* afford information as to the general condition of a child. A *depressed* fontanelle is an untoward sign in all acute illnesses of childhood—*e.g.*, the diarrhœa and vomiting of infancy. The fontanelles *bulge* in inflammation of the meninges, and this is a useful diagnostic feature between true meningeal affections on the one hand, and fevers, broncho-pneumonia, and other diseases with cerebral symptoms on the other. The fontanelles are tense and bulging in all diseases causing increased intracranial pressure—*e.g.*, cerebral tumour. Normally, the anterior fontanelle should be closed between the ages of one and a half and two years, and the posterior at birth. In rickets the former is late in closing.

(iv.) Nothing is more characteristic than the *listless* and apathetic facies of children suffering from the early stages of fever.

(v.) *Rickets* presents pallor, but nothing more distinctive in the face until later, when the prominent and rounded forehead and other signs appear.

(vi.) *Mouth-breathing children* (due generally to postnasal adenoids), when they reach the age of about ten to fifteen years, have a very characteristic expression. The broad bridge of the nose and open mouth give to them a vacant, stupid appearance, which sometimes belies their intelligence, though sometimes they are, in fact, mentally backward.

§ **13. Variations in the Form of the Skull** are met with in several complaints, and chiefly in children, because cases of marked deformity of the head seldom reach adult life. It may be remarked, in the first place, that a small degree of *asymmetry* of congenital origin is of fairly common occurrence.

(i.) In *hydrocephalus* ("water on the brain") the head is enlarged generally, dome-shaped and with wide fontanelles, and the face appears relatively small (see Chapters XVII. and XIX.).

(ii.) In *hereditary syphilis* the bones around the anterior fontanelle are thickened, and irregular patches of thickening and thinning are met with. In children this thinning most frequently occurs in the parietal and occipital regions, and is known as "craniotabes." It is generally an indication of rickets in association with infantile syphilis.

(iii.) In *rickets* the vertex is flat, the forehead prominent, and there are bosses on the frontal and parietal regions.

(iv.) The head is unduly small in *microcephalic*, and boat-shaped in *scaphocephalic* idiots. In the former the cranium is disproportionately small ; in the latter the antero-posterior diameter is elongated, and its lateral diameter diminished. The palate is generally high and arched in both.

(v.) Localised thickenings of the bones of the *adult skull* occur in hereditary syphilis, rickets, osteitis deformans, leontiasis ossea, and after injury.

(vi.) In *acromegaly* (Diseases of the Extremities, § 449) the lower jaw is enlarged, and sometimes the nose also. In this condition the face is ovoid, with the long transverse diameter below. In *Paget's disease* (osteitis deformans) it is ovoid, with the long transverse diameter above ; the hands and feet are also big and clumsy, but the skin is normal.

In various **diseases of the nervous system** the face presents a pathognomonic expression. Thus in Bell's or *facial paralysis* the face is distorted, and so also in that rare condition *facial hemiatrophy*. The expression is *vacant* in idiocy, some hysterical subjects, and early disseminated sclerosis. A smooth, *expressionless* appearance (differing from the preceding in that there is a lack of mobility) is very characteristic of paralysis agitans, and among rarer conditions, of double facial paralysis, sclerodermia, and Raynaud's disease (on account of the sclerodermia present). Bulbar paralysis gives a very characteristic, mournful, or sullen appearance to the face. In this disease the orbicularis oris is paralysed, and allows the lower lip to pout ; while the weakness of the zygomatici results in a drooping of the corners of the mouth, such as we usually associate with sorrow or sullenness of temper. In a more advanced stage the saliva dribbles out of the mouth.

(B) Decubitus (in Acute Conditions) and Attitude (in Chronic Diseases).[1]

§ **14. Decubitus** signifies the position in which a patient tends most constantly to lie, and it often gives a valuable clue to the disease, more especially in the diagnosis of **Acute Diseases,** and sometimes as to their probable issue as well. For example :

(i.) *Sitting up in bed*, propped up with pillows, on account of inability to breathe in other positions (orthopnœa), is characteristic of the extreme breathlessness which occurs in advanced cardiac, pulmonary, or renal disease.

(ii.) *Lying on one side* is characteristic of considerable pleural effusion or pneumonia on that side. When a phthisical patient always lies on one side, we may suspect a cavity, bronchiectasis, or empyema of that side.

(iii.) The *dorsal decubitus—i.e.*, lying on the back—is seen in grave illnesses attended by marked prostration. In the " typhoid state " the limbs are stretched out and completely relaxed. But if the prostration be due to peritonitis, the legs are drawn up, so as to relax the abdominal muscles ; and for the same reason the breathing is thoracic and the abdomen is quite still. The typhoid state, so called from its occurrence in typhus and typhoid fevers, is a condition of profound prostration, attended by unconsciousness or muttering delirium, sordes on the teeth, and a dry, cracked tongue.

(iv.) *Opisthotonos* is an arching of the back which occurs in some convulsive and spasmodic disorders. It may be so great that only the head and heels touch the bed. It is met with in tetanus, hystero-epilepsy, cerebro-spinal meningitis, and strychnine poisoning.

[1] The various characteristic gaits are described under Diseases of the Nervous System.

(v.) *Restlessness* occurs in many disorders, acute and chronic, and is generally a grave sign in the former—*e.g.*, in acute pericarditis. Sometimes, as in children, it is an indication of severe pain. *Carphology* (καρφος = the clothes, λέγειν = to pluck), or floccitatio, is the picking at the bedclothes so characteristic of the "typhoid state." The hands seek after imaginary objects. *Subsultus tendinum* is the muscular twitching or tremor which occurs in the same state. Both of these imply extreme cerebral depression. They are met with in the malignant forms of the acute specific fevers, and are of the gravest possible import.

(vi.) *Retraction of the head* is specially characteristic of meningeal inflammations. It is also met with in cases of cervical caries high up, and in infants with digestive disorders or febrile states.

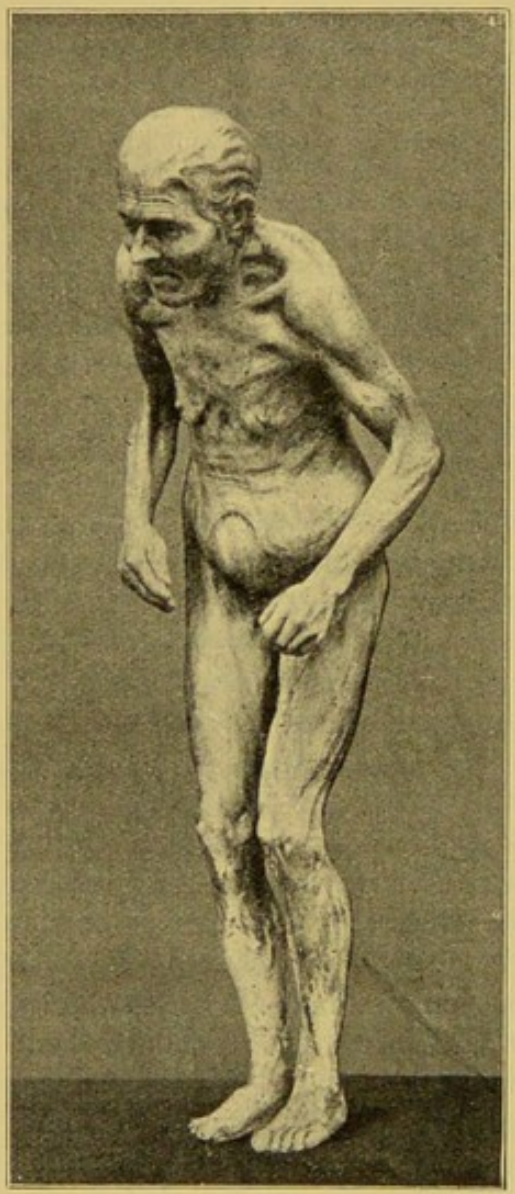

Fig. 4.—The attitude typical of PARALYSIS AGITANS; from a plaster cast by M. Paul Richer.

§ **15.** The **Attitude** which is involuntarily assumed by a patient suffering from certain chronic diseases, if he be able to leave his bed, is very characteristic: Thus:

(i.) In *paralysis agitans* the head, neck, and thorax are bent forwards, and the tendency which the patient has to run forwards is called "festination" (Fig. 4). The disease is recognisable at sight by the smooth, expressionless face, fixity of gaze (always looking forwards), the forward bending of the body, and the short steps which the patient takes as he shuffles along.

(ii.) The attitude assumed by children suffering from *post-diphtheritic paralysis* is somewhat similar to the preceding, and is so characteristic that one can often detect the disease as the patient enters the room. The head hangs forward from weakness of the neck muscles, and the "flabbiness" of all the movements is peculiar.

(iii.) The *rigidity of the spine* in rheumatoid arthritis, osteo-

arthritis, and spinal caries, gives a stiffness and awkwardness to all the movements which is very noticeable.

(iv.) Duchenne's *pseudo-hypertrophic paralysis* (Fig. 5) is a comparatively rare condition, but the arching forwards of the back, prominence of the buttocks, scapulæ, and calves, and inability to rise from a recumbent posture without the aid of the hands, are quite pathognomonic (see Chapter XIX.).

(C) The General Conformation.

§ **16.** Under this heading we note (*a*) whether the patient exhibits any loss of flesh (Emaciation, *infra*); (*b*) whether he presents any increase in volume (General Enlargement, § 17); or (*c*) whether he presents any Deformity or Dwarfism (§ 19).

Here we shall meet with several important diseases affecting the skeleton and general growth of the individual, especially Hereditary Syphilis. The various causes of such alterations will only be mentioned here. They will be described and differentiated under the Diseases of Extremities (Chapter XVII.), and elsewhere.

Variations in Health.—The terms "Emaciation" and "General Enlargement of the Body" are only relative. The healthy man should have an elastic skin, firm muscles, and a slight amount of subcutaneous fat; but *individual variations* are so great that no definite standard can be set up as normal. Health in the wiry, nervous man is consistent with a spareness that would indicate disease in his stouter and more phlegmatic brother. The same holds true with regard to *age*. A child has an amount of fatty covering that would be abnormal in adolescence; an old man

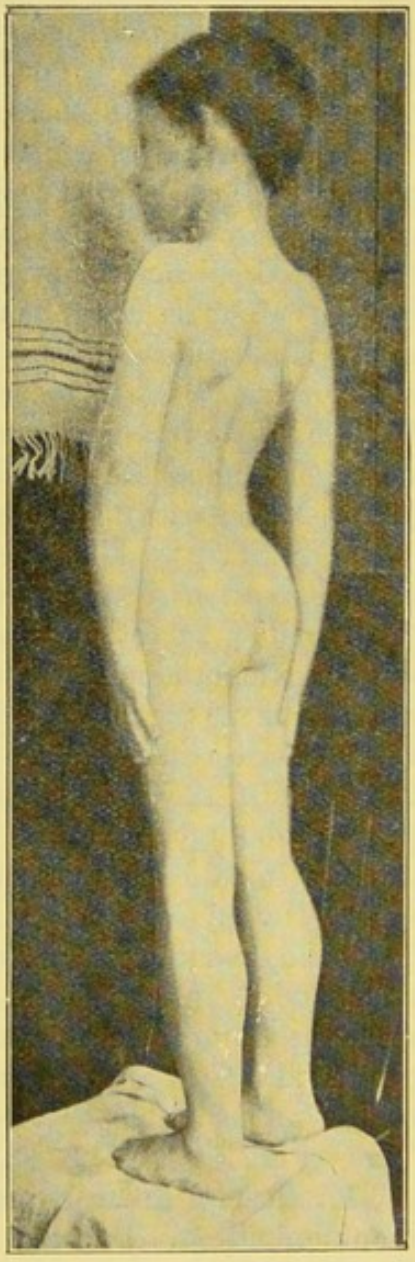

Fig. 5.—Pseudo-Hypertrophic Paralysis of Duchenne.—This patient is a boy, aged nine years. The illustration shows well the two most characteristic features of the disease—namely, the enlargement of the calves and buttocks, and the arching inwards of the back (lordosis). The disease is often combined, as here, with true muscular atrophy in other parts—*e.g.*, the shoulder girdle—and therefore the scapulæ project. The child also exhibited the typical manner of getting up from the prone position. The boy was one of four children, of whom one sister died of spina bifida, aged twenty-one days; one brother died of "water on the brain," aged six months; and one sister, aged fifteen months, living and healthy. The patient seemed normal until he began to walk (aged two years), when it was noticed he "lifted his legs too high." The mother was aged twenty-six when he was born. No known defect in family history.

has atrophy of the soft parts and prominence of the bones which in the middle-aged man could only accompany serious disease. The question of build is very largely one of *heredity*. Stout parents generally have children who tend to become stout, and *vice versa*.

(*a*) **Emaciation** is necessarily attended by more or less weakness, and the subject is dealt with under General Debility (Chapter XVI.).

The chief causes of debility with emaciation are as follows : Malignant disease, digestive disorders and privation, diabetes, various nervous disorders, chronic Bright's disease, syphilis, tubercle, and pancreatic diseases ; and in children, tabes mesenterica, defective feeding, diarrhœa, and hereditary syphilis.

In *advanced life* the first cause which occurs to our minds, if the patient has lost flesh, is cancer ; in *middle age*, diabetes ; and in *young adults*, tuberculosis. In tuberculosis of the lungs or elsewhere, emaciation may occur before any physical signs can be detected ; indeed, loss of flesh which is accompanied by an intermitting pyrexia generally means latent tuberculosis. In *infancy* the two most common causes of acute or *rapid* wasting are Defective Feeding and Gastro-Intestinal Catarrh. The two most common causes of slow, progressive, or *chronic* wasting in infants are tuberculosis of the intestine and mesenteric glands (Tabes Mesenterica), and Hereditary Syphilis (§§ 417 and 404).

In hereditary syphilis the child may be born quite healthy in appearance, but often soon begins to waste, the face presenting a wizened "old man" aspect. Rhinorrhœa, or "snuffles," and condylomata about the mouth and anus follow. Various skin manifestations (§ 455) occur, perhaps the most typical being a scaly eruption of the palms and soles. Periosteal nodes develop, especially about the upper end of the tibia, whilst the skull shows localised thinning of the occipital and parietal bones (craniotabes) or local bossing. At about puberty a new group of manifestations arises, notably keratitis, deafness, and synovitis. Hutchinson's pegged and notched teeth may also be in evidence. The condylomata have been replaced by scars about the mouth and anus, and the bridge of the nose may have fallen in. The deformities resulting from these various lesions may persist, and constitute the chief evidences we have of hereditary syphilis in a patient over the age of fifteen (*vide* Table, § 404).

§ **17. General Enlargement** of the body is much less often met with than diminution. It occurs in *Obesity*, *Generalised Dropsy* (see §§ 9 and 21), *Myxœdema* (see § 9, and General Debility, § 420), and *Acromegaly*.[1] It is probable that the giants of old were specimens of acromegaly. These affections will be described and differentiated elsewhere, but since the treatment of obesity has unfortunately been allowed to get

[1] I have also met with general enlargement of the extremities in certain rare cases presenting vaso-motor symptoms.

into the hands of charlatans and patent-medicine vendors, it will be well to add a few remarks on the causes and treatment of that condition.

§ **18. Obesity** is very largely a question of heredity, and no amount of dieting will make any difference in some people. In others it is an indication of luxurious or sedentary living, or of indulgence in alcohol. Women frequently become obese just about the menopause. Sometimes it is found in chronic cerebral disease, such as idiocy or tumour, and, more rarely, in tumours of the adrenal bodies, associated with sexual precocity.[1] The pathological causes of obesity come under two headings: (i.) excessive intake of those food-stuffs known to produce fat; and (ii.) deficient oxygenation. It is probably due to the latter cause that persons with persistent low tension are apt to become fat. Both causes may be in operation. Successful treatment must therefore depend either upon diminution of intake or increase of oxygenation.

Treatment.—Our first duty when consulted about such cases is to examine every organ in the body, especially the heart, lungs, and liver, because excess of subcutaneous fat is often attended by a similar deposit of subpericardial fat; and, if due to alcohol, by fatty degeneration of both heart and liver. Chronic bronchitis and emphysema are also frequently followed by obesity from deficient oxygenation. If no serious lesion be present, there are at least five methods of reduction: (1) To limit the amount of fluid taken with meals; (2) Banting's system consists in excluding all fats, sugars, and starches from the diet, green vegetables and lean meat alone being allowed; (3) Ebstein's system only excludes all sugars and other carbohydrates; (4) Oërtel's system is the most complete, and consists in (i.) slowly climbing mountains for several hours daily, inspiring with one step and expiring with the next; (ii.) food mainly nitrogenous, with only small quantities of fats and starches; (iii.) meals taken at regular intervals, and in strictly-regulated quantities; (iv.) the fluid limited to 1½ pints in twenty-four hours (see Chapter III., § 51). (5) Some cases of obesity may be successfully reduced by *limiting the food* entirely to *one pound of lean meat* or fish a day, divided into four meals, taken without fluid; between meals the patient should drink as much hot water as possible by constantly sipping it. If the patient will co-operate, this method is very successful,[2] but the diet is a Spartan one. Thyroid or strychnine, either separately or together, are also of value in obesity.

Adiposis Dolorosa[3] is a rare variety of obesity described by Dercum. It is characterized by the local deposition of fat in the form of tender adipose tumours. The condition spreads and becomes more general, the hands, feet, and face escaping. Pains, both constant and paroxysmal, occur in various situations, associated with great muscular weakness and areas of partial loss of sensation.

§ **19. Dwarfism,** or diminished stature, may arise from any cause which affects the growth of the bones of the trunk or limbs, whether local or constitutional. The commonest causes of a stunted condition of the body, in order of frequency, are:

(i.) *Rickets.*—In this disease there is curving of the long bones, together with altered epiphyseal growth. This results in "bandy legs," "knock-knee," and other familiar deformities (see Chapter XVII., § 447).

(ii.) *Hereditary Syphilis,* the means of recognising which are fully given in Chapter XVI., § 404.

(iii.) *Curvature of the Spine,* which may take three forms: *kyphosis* (*i.e.*, the convexity projecting backwards), usually due to tuberculous or other disease of the vertebræ; *lordosis* (*i.e.*, a forward projection), usually compensatory, or the result of muscular weakness; and *scoliosis* (a lateral curve). All these may

[1] Guthrie and Emery, Clin. Soc. Trans., 1907.

[2] A case is published in the *Lancet,* 1893, vol. ii., p. 133.

[3] Dercum, *Internat. Journ. Med. Sci.,* 1892, p. 521; and *Brit. Med. Journ.,* 1889, vol. ii., p. 1553.

diminish the stature, but they differ considerably in importance. A certain amount of scoliosis is normal to nearly everyone, and the kyphosis of muscular weakness is common enough in old age, as a consequence of which our stature becomes slightly less in advancing years. It is the angular kyphosis which is so serious, as indicating organic diseases of the bodies of the vertebræ.

(iv.) *Cretinism* (§ 138) is a peculiar stunting of the growth which is endemic among children in certain districts, particularly the valleys of some parts of Switzerland, and elsewhere. The appearance is so distinctive that typical cases can be recognised at a distance (Fig. 6). The face is broad and flat, and joined

FIG. 6.

FIG. 7.

Fig. 6.—Case of CRETINISM under the care of W. Rushton Parker.—The child, aged six, presented the characteristic aspect of a cretin—viz., sunken nose, swollen mouth, small eyes widely apart, coarse hair, and stumpy limbs. Fig. 7 shows the same child after six months' treatment with thyroid gland (5 grains daily).

almost without a neck to the body. The skin and hair are coarse, the hands broad and stumpy, the stature stunted, for even when twenty years of age a cretin may be only 3 feet high. It is due to a perverted or diminished action of the thyroid gland, and recovery usually results and is maintained while thyroid extract is being given (Fig. 7).

(v.) *Achondroplasia.*—A rare condition somewhat resembling, and formerly confused with, Rickets (see § 447).

(vi.) *Osteomalacia*, when this disease involves the spine (§ 449).

(vii.) *Infantilism* is due to a loss or perversion of internal secretions, the usual changes, both sexual and physical, which normally occur at puberty failing to

take place, and the patient retaining the stature, features, and voice, and often the mental proclivities, of a child. Under the name *progeria* Hastings Gilford[1] has described a condition in which infantilism is associated with premature decay, the appearance, attitude, and state of nutrition of the dwarf becoming senile, and degenerative changes occurring in the vessels and viscera.

(viii.) In addition to the foregoing there are certain rare conditions, of which the celebrated Tom Thumb and his wife, and the race of pigmies of Africa met with by Sir H. M. Stanley[2] and others, are examples, in which the skeleton and the organs are diminished in size, but their proportions maintained. Such cases, however, seem to be functionally normal in every respect.

Some of the diseases above referred to belong so distinctly to the domain of surgery that reference must be made to other works for their differentiation. Others will be described under Diseases of the Extremities.

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[1] *Practitioner*, 1904, vol. lxxiii., p. 188.
[2] F. C. Shrubsall, *Lancet*, vol. i., 1908.

CHAPTER III

DISEASES OF THE HEART AND PERICARDIUM

There are three noteworthy facts in connection with the diseases of the circulatory system. First, the left side of the heart is stronger and much more prone to disease than the right; secondly, the arteries are, in a corresponding manner, much thicker and more often diseased than the veins; and thirdly, heredity plays a very prominent part in chronic disorders of the heart and arteries.

The saying of Björnson that "heredity is a condition, not a destiny,"[1] applies here; for although its application is chiefly ethical, it may also be employed in a physical sense also. Careful living may do much to counteract the hereditary tendency to early death from chronic cardio-vascular disease.

Following out the plan adopted in this work, we shall consider:—

First, the Symptoms which lead us to infer the presence of cardiac disease;

Secondly, the Physical Examination of the patient; and

Thirdly, the Differentiation of the Various Diseases which affect the heart and pericardium, their diagnosis, prognosis, and treatment.

PART A.—SYMPTOMATOLOGY.

The general symptoms (*e.g.*, breathlessness, dropsy, etc.) of cardiac disease, as distinct from the local signs referable to the heart, should be studied very carefully, inasmuch as the gravity of any given case depends not so much on the local signs present as on the general condition of the patient.

The Three Cardinal Symptoms of diseases of the heart and pericardium are **Breathlessness, Dropsy,** and **Cyanosis.** To these may be added **Palpitation, Præcordial Pain, Syncope,** sometimes **Cough,** and, in acute affections, **Pyrexia** and its concomitant symptoms. **Sudden Death** is more frequent in disorders of the heart than in disease of any other viscus, and it is sometimes unattended by any previous manifestation of heart disease.

[1] "The Heritage of the Kurts."

§ **20. Breathlessness,** or Dyspnœa, is a constant symptom in all diseases in which the heart is unequal to the work demanded of it. Breathlessness may be present without cardiac disease ; but it may be affirmed that no serious affection of the CARDIAC WALL can exist without some degree of breathlessness. It may be only on exertion, such as walking up a few stairs, but it can always be elicited in some degree.

Severe disease of the VALVES of the heart may, however, exist for many years—provided the obstruction so caused is adequately compensated for by increased growth in its muscular wall—without the patient having any noteworthy symptoms, or even being aware of its existence, until the cardiac wall begins to degenerate and its cavity to dilate. Then, as time goes on, breathlessness is sure to appear, and it is for this symptom that you are generally consulted in heart cases. In the later stages it becomes so pronounced that the patient is unable to breathe when lying down, and night after night is passed sitting upright in a chair, or, at best, propped up with pillows in bed (orthopnœa).[1] Towards the end, in a certain number of cases, a larger proportion in my experience than is generally supposed, some degree of *Cheyne-Stokes respiration* may be observed.

CAUSES OF BREATHLESSNESS (DYSPNŒA).—Difficult breathing may arise in five different groups of disorders.[2]

1. Cardiac Disease.—The dyspnœa of heart disease has no intrinsic features which distinguish it from that due to other causes, except that it is apt in some cases to be paroxysmal. There is, however, usually a history, or evidence, of some of the other symptoms of cardiac disorder. In cardiac valvular disease the amount of breathlessness present and the distance a patient can walk without producing it are, of all symptoms, the most valuable indications as to the amount of inadequacy of the cardiac muscle (cardiac failure) present in any particular case.

2. Embarrassment of the Heart by Neighbouring Structures, such as a dilated stomach, ascites, mediastinal tumours, or obesity. Murchison used to teach that dyspnœa and nearly all the symptoms of cardiac disease (excepting cyanosis) may be produced by dyspepsia without any structural disease of the heart.

3. Laryngeal or Tracheal Obstruction.

4. Pulmonary Disease, of which emphysema is the most common.

5. Toxic or Hæmic Conditions, the most frequent of which is certainly anæmia. Deficient aeration and other poisonous conditions of

[1] This word simply implies very urgent dyspnœa—ὀρθος, Greek, erect.

[2] This does not include the dyspnœa associated with vaso-motor spasm or with vaso-motor paresis and states of low arterial tension, such as may be met with in great prostration or bodily fatigue. This kind of dyspnœa, I have observed, has for its chief feature a sighing character of the respiration, and long-drawn sighs occur every few seconds.

the blood, such as uræmia (chronic Bright's disease), diabetes, and all pyrexial states, may be attended by dyspnœa, caused in this way.

Causes of Breathlessness which are apt to be overlooked.—The differentiation of the various forms of cardiac disease will be given in the following pages ; but, supposing a patient over thirty-five or forty, who complains of breathlessness, presents no definite signs of cardiac or pulmonary disease, nor any evidences of dyspepsia or anæmia, then there are certain conditions which should be *suspected :*

1. **Cardiac Enfeeblement** or **Fatty Degeneration,** in which case the sounds and impulse would be very feeble, and the other signs mentioned in § 52 should be present.

2. **Arterial Sclerosis,** in which case the radials would be hard and cord-like, but smooth, unless atheroma were also present, and the arterial tension high (excepting in the very last stage of the disease). Here, also, there would be giddiness, especially on assuming the erect posture, and the other symptoms mentioned below (see § 67).

3. **Aortic Stenosis,** the murmur of which is sometimes very difficult to detect, especially when attended by cardiac enfeeblement.

4. Deep-seated **Aneurysm of the Aorta** and other **Intrathoracic Tumours** may give rise to the breathlessness and general symptoms of heart disease without the physical signs. In such cases the dyspnœa may be paroxysmal.

5. **Coronary Obstruction** (*i.e.*, diminution of the calibre of the coronary arteries by atheroma, calcification, or other disease). In this obscure condition the patient probably complains also of "dizziness in the head" on suddenly assuming the erect position, from the incapacity of the heart to pump the blood to the head. But this condition can never be more than suspected during life.

In a patient under thirty-five or forty the three following causes of Unexplained Breathlessness may be suspected :

6. **Cardiac Syphilis** may be unattended by any signs or symptoms, excepting breathlessness (§ 41). Happily the condition is very rare.

7. **Adherent Pericardium,** also, is often unattended by any physical signs (§ 38c).

8. **Latent Pulmonary Disease,** and especially latent pulmonary tuberculosis, should always be suspected in cases of breathlessness without obvious cause.

9. When severe dyspnœa sets in suddenly in the course of cardiac or **Acute Renal Disease,** or during an attack of *scarlatina,* the chest should always be carefully examined, because double hydrothorax may set in rapidly without any general dropsy or other warning symptom, as in a case mentioned by Osler, and a similar one which I have had the opportunity of observing.

§ 20*a*. **Paroxysmal Dyspnœa** is that form of dyspnœa which occurs in attacks from time to time. It is apt, as above mentioned, to occur in some cases of cardiac disease, and in any given case our attention should first be directed to the heart. But there are several other conditions which one would suspect in a patient in whom the chief or only symptom consists of paroxysms of breathlessness.

1. Aneurysm and other Intrathoracic Tumours may give rise to paroxysmal dyspnœa before other signs can be made out.

2. In Asthma the attacks of breathlessness are typically paroxysmal ; and if the disease is of long standing, it is usually associated with alterations in the shape of the chest.

3. Paroxysms of dyspnœa occurring at night is often one of the first symptoms of Chronic Bright's Disease, and is spoken of by the patient as asthma.

4. Neurotic Dyspnœa.—Some neurotic patients are liable to attacks or paroxysms of panting respiration, resembling the panting of fear. It usually ceases when the patient converses with you, and is, of course, unattended by any signs in the lungs.

5. Lingual Varix—*i.e.*, a varicose condition of the veins at the root of the tongue—may give rise to severe paroxysms of dyspnœa.[1]

[1] A case of paroxysmal dyspnœa, cured by the removal of a lingual varix, is published by M. P. Mayo Collier in the West Lond. Med. Chir. Soc. Trans., 1897, p. 206.

6. Foreign Bodies in the trachea in children, and polypi or papillomata of the larynx in adults, give rise to paroxysms of dyspnœa.

7. Sudden dyspnœa, coming on during vomiting, is the main indication of that rare accident, Rupture of the Œsophagus. This dyspnœa is due to pneumothorax, and by promptly opening the thorax on that side the patient's life might be saved.[1]

8. Enlargement of the Thymus Gland in the condition known as Lymphatism, or status lymphaticus, in which there is general hyperplasia of lymphatic structures associated with a persistent thymus, may cause paroxysmal dyspnœa, to which the name "thymic asthma" has been given. It is sometimes rapidly fatal; especially under anæsthetics.

§ 20*b*. **Cheyne-Stokes Respiration** (so called after its first observers) consists, in its typical form, of a series of eight or ten rapid inspirations gradually increasing in depth and rapidity, and then dying gradually away, each series being separated by a pause of five to thirty seconds (the stage of apnœa), in which there is hardly any respiratory movement (Fig. 8).

In a modified form, without the apnœa pause, Cheyne-Stokes breathing is by no means infrequent. It seems to indicate a want of harmony between the cardiac and the vaso-motor regulator mechanism. It is usually a serious symptom, and appeared in the great majority of those of my cardiac patients in the infirmary who were closely observed *towards the end of life*. It has less significance at the extremes of life, for it may be observed during sleep in normal infants, and is

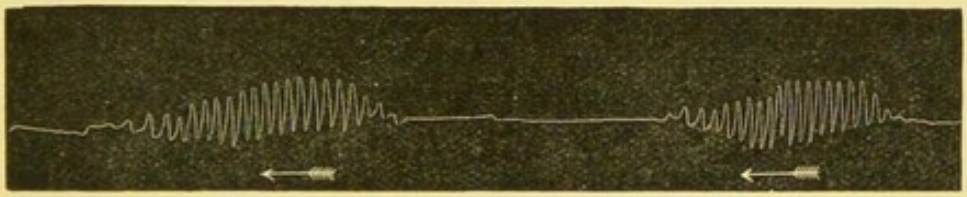

Fig. 8.—Cheyne-Stokes Respiration.—Respiratory tracing of Cheyne-Stokes breathing, for which the author is indebted to Dr. C. O. Hawthorne, who took the tracing from a case of cerebral embolism under his care in the Western Infirmary, Glasgow.

compatible with a hale old age. Another exception to the unfavourable import of Cheyne-Stokes breathing occurred under my notice in the case of a focal lesion of the pons, producing crossed hemiplegia, in which it persisted with only occasional intermissions during the last six months of the patient's life. Its principal *causes* are as follows:

1. Cardiac Disease, which is certainly its commonest cause.
2. Uræmia.
3. Apoplexy.
4. Tuberculous Meningitis, and other states of cerebral congestion or compression.
5. Arterial Sclerosis (used in its widest sense).
6. Sunstroke.

Thus it will be seen that the three pathological conditions in which it is apt to occur are: States of cardiac failure; states of cerebral congestion; and toxic blood conditions.

§ 21. **Dropsy** is a chronic effusion of fluid (lymph) into the subcutaneous tissue (when it is known as anasarca) or into the serous cavity (as in hydrothorax, hydropericardium, ascites). The former, **Anasarca,** is the variety of dropsy we are now concerned with, for it is a very

[1] A few cases of this accident have been recorded. If it were recognised, the thorax might be opened without fear on the side of the pneumothorax, and the œsophagus stitched. See Discussion at Roy. Med. Chir. Soc. in spring of 1900.

constant feature of some forms of cardiac disease. General anasarca has to be differentiated from myxœdema, in which the swelling is harder, and does not pit on pressure. It is best to apply the pressure over a bone, such as the lower end of tibia on its inner aspect.

Causes.—The causes of localised dropsies are given in Diseases of the Extremities (§ 425). There are *three varieties of general anasarca*, which differ from each other both pathologically in their origin, and clinically in the course which they pursue.

1. **Cardiac Dropsy** (1) *starts*, and throughout the case predominates, in the *most dependent parts*, that is to say, in the legs if the patient has been walking about, or in the back if he has been lying in bed. On inquiry, the patient may complain that the ankles swell towards evening around the top of the boot, and on pressing over the tibia the dent will remain after the removal of the finger. (2) Other signs and symptoms of cardiac enfeeblement or dilatation are present ; and perhaps those of valvular disease as well. (3) In the history of the case dyspnœa will have *preceded* the dropsy.

Dropsy does not occur with equal frequency in all forms of cardiac valvular disease. It is common in disease of the mitral valves, but rare in aortic disease, at any rate until quite the end. The dropsy which complicates pulmonary disease has the same features as cardiac dropsy, because it is the resulting cardiac dilatation which produces the dropsy.

2. **Hepatic Dropsy** (1) always begins and predominates *in the abdomen* (ascites), although the legs may swell subsequently by reason of the pressure of the fluid on the veins within the abdominal cavity. (2) There may be also enlargement and other signs of the liver affection which has given rise to the condition ; and if these be absent some other cause of obstruction to the portal vein should be sought (Chapter XII.). (3) The dyspnœa will have *followed* the abdominal enlargement.

3. **Renal Dropsy** is (1) *general in its distribution* from the beginning, occurring in the legs and eyelids at the same time ; though it is probable that the œdema round the eyes on rising in the morning first attracts the attention of the patient or his friends. (2) Examination of the urine reveals the features of renal disease (Chapter XIII.). (3) The patient presents a characteristic pale or waxy appearance.

In some cases of general anasarca associated with albuminuria, the question arises whether the dropsy is of renal or cardiac origin. This may sometimes be answered by finding the liver enlarged, for this is a natural sequence of cardiac valvular disease, but not of renal disease.

Prognosis.—The dropsy of cardiac disease admits of a mechanical explanation,[1] for it is due to the backward pressure within the

[1] That this is not the only cause is evidenced by certain facts. Thus, occasionally, cases of mitral disease are unaccompanied by dropsy at any time. And, again, the degree of cyanosis, which is sometimes also a measure of venous stagnation, may have no ratio to the amount of dropsy present.

veins. Its occurrence is therefore an indication and a measure of the amount of obstruction to the circulation on the right side of the heart.

The *treatment* of all forms of dropsy should be directed to the removal of the cause. But even if this be not removable, the dropsy may frequently be alleviated. The limbs should always be rested, raised to the same level as the body, and kept warm. The additional support of a well-adjusted flannel or stocking bandage is a great comfort to the patient, and helps to prevent further effusion. Diuretics and diaphoretics should be employed. These failing, we may (*a*) employ Southey's trocars, the patient being wrapped in blankets and the fluid allowed to slowly drain away ; or (*b*) make punctures in the skin of the limb with a small two-edged scalpel. Six or eight punctures are sufficient, the positions of the veins being avoided. These procedures are not without some danger of erysipelas and cellulitis, and strict antiseptic precautions should be followed. Dropsical limbs have a tendency to the development of eczema, erythema, cellulitis, and epidemic exfoliative dermatitis, and our efforts must be directed to prevent their supervention.

Obscure Causes of General Anasarca.—If, in a patient who complains of dropsy, no marked evidences of cardiac, renal, or hepatic disease are discoverable, the following causes may be *suspected :*

1. **Anæmia** is not infrequently attended by some swelling of the ankles at the end of the day. This may appear quite early in chlorosis, but is rare in pernicious anæmia.

2. Other **Defective Blood Conditions,** such as leucocythæmia, may be accompanied by dropsy. Swelling of the feet and ankles may be present in the last stages of many exhausting diseases, such as phthisis, in septic states, and in cases of insufficient nutrition and old age.

3. In **Fatty Heart** anasarca is not a prominent symptom, but a slight degree is frequently present.

4. Among the less frequent causes of dropsy in this country are **Beri-Beri** and **Epidemic Dropsy**. In Beri-Beri there are symptoms also of peripheral neuritis. Epidemic dropsy is the name applied to an outbreak which occurred in Calcutta in 1878-80, in which the leading symptom was anasarca, beginning in the lower extremities, accompanied by moderate pyrexia, gastro-intestinal disturbance, and sometimes itching, urticaria, or other rashes.[1]

§ 22. **Palpitation** is the sensation of "fluttering in the chest" experienced by a person when he is conscious of the beating of his heart. It arises under two sets of conditions. It is said to be Symptomatic when a cause can be assigned to it, such as heart disease, anæmia or intrathoracic tumour. It is said to be Idiopathic when no organic cause can be discovered to account for the symptom (compare also Paroxysmal Tachycardia, § 40).

Symptomatic Palpitation may arise from (*a*) causes referable to the heart itself (causes 1 to 3) ; or (*b*) morbid conditions outside the heart (causes 4 to 9).

[1] Trans. Epidem. Soc. Lond., vol. xii., p. 55 (New Series).

1. Most **Structural Diseases of the Heart**, especially such as are attended by rapidity and irregularity of its rhythm, are attended by palpitation. When patients consult us for this symptom, we should therefore first make a thorough examination of the heart and pericardium.

2. Palpitation is also the leading indication of the **Irritable Heart,** in which there may be no discoverable structural disease. Irritable heart is found in young men who have overtaxed their strength in athletics or military work, and occasionally in young women. Palpitation is its most obvious symptom, with a very quick pulse, or one which *easily becomes rapid*, and sometimes with high arterial tension. Breathlessness, sleeplessness, incapacity for prolonged exertion, and nervousness, often accompany the palpitation. Hypertrophy is a common result ; and unless rest be ordered the heart will become dilated. Complete rest is the chief indication for treatment, and is generally successful. Irritable heart is, in my belief, in some cases a symptom of neurasthenia (*q.v.*).

3. Various **Nervous Causes,** such as fright, fear, or other emotion, especially after an exhausting illness, give rise to palpitation (compare Paroxysmal Tachycardia, § 40).

4. In **Anæmia** the palpitation is a frequent and often distressing feature.

5. In **Dyspepsia** palpitation is very often present. In such cases it frequently occurs at night, especially after taking a heavy meal. It may, in these circumstances, be accompanied by morbid dreads—*e.g.*, of impending death—by breathlessness, cardiac pain, and by other cardiac symptoms.

6. Certain **Local Conditions,** such as thoracic or abdominal tumour, or dilated stomach, which hamper the heart's action, may produce palpitation, although the heart be healthy.

7. The excessive use of **Certain Drugs** or **Articles of Diet,** notably tobacco, tea, coffee, and alcohol.

8. In **Graves' Disease** (exophthalmic goitre) violent palpitation and greatly increased rate of the heart are prominent features. In quite a number of my cases this and the other nervous symptoms of the disorder had existed for many months, or years, before the two diagnostic features—thyroid enlargement and exophthalmos—became obvious. Graves' disease should always be suspected in cases of persistent palpitation for which no cause can be made out.

9. In **Hysterical** subjects palpitation is a symptom often complained of, and occasionally it takes the form of a definite and somewhat alarming attack (see a case, § 40).

§ **23. Pain in the Chest** is not always present, even in grave cardiac disease. A feeling of discomfort or constriction, or a sense of suffocation, is a symptom frequently present when the action of the heart is deranged by functional or structural diseases—oftener perhaps by functional.

To avoid certain *fallacies*, ascertain if there be tenderness on pressure. If so, the lesion is probably a *neuralgia* of the intercostal nerves, or the *inframammary pain* so common in hysteria. The latter may sometimes be distinguished in this way from pain of cardiac orgin, but

occasionally can only be recognised by the presence of other hysterical stigmata. Pain in the præcordial region is not infrequently associated with lithæmia, and may be attended by tender spots over the ribs at the apex.

There are FOUR GROUPS OF CAUSES which may lead to præcordial pain :

(*a*) When præcordial pain occurs as the result of **Structural Disease of the Heart** (due to overstrain), it usually consists of a diffuse dull aching, most severe at the apex. As in other organs having a serous covering, pain is more often present when that covering is inflamed (pericarditis) than when the substance of the organ is affected. But pain may be altogether absent ; and it is surprising what serious valvular derangement of the heart may exist without the occurrence of any pain at all.

(*b*) **Cardiac Pain of Functional Origin** may be due to (i.) *pressure upon the heart* by a distended stomach or abdomen ; the differential features of this pain are—it is greatest at the base of the heart, aggravated by the recumbent posture, and associated with dyspnœa. (ii.) *Reflex Pain* may be referred from stomach (chiefly) or uterine disorders (occasionally). (iii.) *Nervous Pain* due to profound grief, sudden fright, or other violent emotion, is of a sharp character, referred to the præcordium.

(*c*) Various **Organic Affections outside the Heart and Pericardium** may give rise to præcordial pain ; thus we may have *intercostal neuralgia*, especially the neuralgia which precedes and follows herpes zoster ; *intercostal rheumatism*, and many *pleuritic affections*. Pain in the chest is also present in *spinal caries* and *carcinoma of the vertebræ*.

(*d*) **Angina Pectoris** is a condition manifested by paroxysmal attacks of extremely severe constricting pain in the chest, with a sense of suffocation and other symptoms. It will be referred to under the acute disorders of the heart (§ 41).

But, in cases of unexplained pain in the chest, and in the absence of cardiac signs, *mediastinal tumour* or *aneurysm of the aorta*, either of the arch or of the descending aorta,[1] should always be suspected.

In the *treatment* of præcordial pain an endeavour should be made to ascertain and relieve the cause, but much relief may be obtained temporarily by the application of an opium or belladonna plaster, belladonna liniment, or glycerine of belladonna.

§ **24. Syncope** is suspended animation due to anæmia of the brain. It is often preceded by giddiness, nausea, and a feeling of faintness.

[1] In a case of aneurysm of the descending thoracic aorta which I have recently seen, almost the only symptom or sign, besides breathlessness, during eighteen months—up to the time of sudden death from rupture of the aneurysm into a bronchus—was continuous pain in the præcordial region. It extended round from the back on the left side, and was thought to be intercostal neuralgia.

The face is ashy pale and the pulse and respiration feeble. Its advent is usually sudden, but recovery, after the attack has lasted some minutes, is gradual.

Diagnosis.—Syncope has to be distinguished from *epilepsy minor*, which it resembles in many respects. First, epilepsy minor (*petit mal*) is usually preceded by an aura, though this is evident to the patient only. Secondly, its advent is more sudden than syncope, and the return to consciousness equally sudden and complete, for the patient in *petit mal* can go on with his usual avocations immediately afterwards. Thirdly, syncope rarely occurs without some definite determining cause, although it may be of a trivial nature—such, for example, as a heated room, or the sight of blood. Finally, in epilepsy minor there is generally a history of major attacks at some time.

Causes.—Syncope nearly always arises from either structural or functional derangement of the heart—more frequently the latter. A careful examination of the heart should always be made, because, as an indication of STRUCTURAL DISEASE, syncope is a symptom of considerable gravity ; whereas the nervous faints of FUNCTIONAL DERANGEMENT are of comparatively little moment.

(*a*) The **Nervous Faints** due to functional derangement of the heart are happily the more frequent. They occur chiefly in young, anæmic, and nervous females ; who, when exposed to grief, bereavement, or any sudden emotion, or too hot rooms full of vitiated air, develop the familiar "fainting attack." Slight transient syncopal attacks are, indeed, one of the "stigmata" of the hysterical diathesis, the other three being hemianæsthesia, ovarian tenderness, and globus.

(*b*) As a symptom of **Structural Heart Disease**, syncope is a much more serious matter. It is a not infrequent symptom in any form of cardiac disease attended by enfeeblement of the heart's action, and is serious as indicating weakness of the *cardiac wall*. It is more often met with in aortic than in mitral valve disease. It may be the first and only symptom of fatty (§ 52), fibroid, or other degeneration of the heart. Syncopal attacks, preceded by giddiness, may arise in old people who are the subjects of arterial thickening and degeneration, this being the cause of what is known as "senile syncope." So important is it to distinguish between the two kinds of fainting attacks that their differential features are given in a table. In both there is pallor of the surface, and there may be feeble pulse, though the pulse in nervous faints is sometimes unaltered. Both recover best in the recumbent position.

Other less common or obvious causes of syncope :

(*a*) *Acting directly through the Heart.*—(i.) Latent organic disease of the heart, such as fatty degeneration, which should always be suspected in obscure cases, or Stokes-Adams disease (§ 58) ; (ii.) compression of the heart, as by corsets or by mediastinal tumour ; (iii.) profuse internal hæmorrhage ; (iv.) drugs and asthenic poisons acting on the heart.

(*b*) *Acting through the Nervous System.*—(i.) Emotions and fatigue ; (ii.) violent injury or operation ; (iii.) irritant poisons, or injury to the intestines.

(*c*) *Acting through the Blood and Bloodvessels.*—(i.) Anæmia, debility, hunger ; (ii.) increased peripheral resistance in the arteries, with insufficient cardiac hypertrophy ; (iii.) excessive heat, as in hot rooms or Turkish baths ; (iv.) suddenly assuming the erect posture, as in jumping from bed, may produce syncope in the aged ; (v.) sometimes, in addition to the preceding, the splanchnic veins are suddenly dilated by emptying the bladder, and this leads to anæmia of the brain and syncope.

TABLE I.

Cardiac Syncope, associated with Structural Derangement of the Heart.	**Nervous Faints**, in which only the Nervous Apparatus of the Cardio-vascular System is deranged.
Usually adults ; both sexes equally affected.	Females, young or at menopause.
May come on without any apparent cause, or after excessive exertion.	Some determining cause always present (*e.g.*, emotion), acting on the nervous system.
Not accompanied by emotional manifestations.	Often preceded or followed by crying or laughter, and other emotional symptoms.
May be fatal.	Never fatal.
Evidences of cardio-vascular degeneration and its causes. If no signs, suspect fatty heart.	Sometimes other evidences of the hysterical diathesis—*e.g.*, hemianæsthesia, ovarian tenderness, globus.

Prognosis.—Syncope in the young is, as we have seen, usually a neurosis, whereas in the aged it generally means cardio-vascular degeneration. In the former, therefore, it is usually as trivial as in the latter it is serious—the gravity depending upon the nature of the lesion.

Treatment (*see Causes above*).—Place the patient immediately in a horizontal position, with the head low. This may be most readily done on the floor, but if there is little space, instruct the patient to bend forward and lower the head between the knees. Apply ammonia to the nostrils, throw cold water on the face, and, in severe cases, apply a mustard-plaster over the heart. If recovery does not promptly take place, and the pulse be very feeble, a hypodermic injection of 15 or 20 ♏ of ether or brandy, or 3 or 4 ♏ of liq. strychninæ, may be resorted to. For further treatment, see Collapse (Chapter XIX.).

§ **25. Cough** is a symptom which belongs more especially to diseases of the lungs (Chapter VI., § 70), but it is met with in diseases of the cardio-vascular system under two circumstances. First, the lungs are very often involved secondarily to the heart, especially when the right side is

diseased, and then the patient has the cough usual to pulmonary disorders. Secondly, when the aorta, by its enlargement, presses on the trachea, or on the recurrent laryngeal nerve, a peculiar dry, brassy, or, as it is aptly called, "gander" cough is present, which is so characteristic as to be in itself a diagnostic feature of aneurysm of considerable value.

§ **26. Cyanosis** (κυάνεος, blue) is the lividity of the surface which appears when the skin becomes congested or the blood too purple or venous in character. It is not one of the most common symptoms in heart disease, but it is one of the most serious and unmistakable evidences of enfeebled or retarded circulation. It is generally most pronounced on the lips, fingers, and toes, and the skin may vary in colour from a faint purple almost to a black. When only a slight degree of cyanosis is present, it may be detected by closely examining the roots of the nails. Cyanosis is an indication of deficient aeration of the blood, which may be due to (*a*) venous stasis, a slowing of the blood-current (*e.g.*, that occurring in the later stages of some cardiac diseases); (*b*) defective oxygenation in the lungs (*e.g.*, that occurring near the end of some pulmonary diseases).

Sometimes both (*a*) and (*b*) are in operation together; but the lividity of cold, rigors, and collapse (*e.g.*, cholera) are probably instances of venous stasis alone. Increased viscidity of the blood is the proximate cause of the latter.

1. If the patient whose leading symptom is cyanosis be a child who has presented this lividity for a long time, perhaps throughout life, the disease is almost certainly **Congenital Heart Disease,** though this is not a very common disease. The lividity may be very marked, without, strange to say, the child appearing to suffer any great inconvenience.

2. The presence of cyanosis in adult life forms strong evidence, if angio-neurotic cyanosis be excluded, of organic disease of the **heart** (especially of the right side), or of the **lungs,** or **both.** It is generally a serious, and it may be a lethal, indication.

The *Causes of Cyanosis*—which depends for the most part on defective oxygenation of the blood—may be arranged into (*a*) those in which the onset of cyanosis is usually GRADUAL, and (*b*) those in which the onset is usually RAPID. *In general terms, the sudden occurrence of cyanosis in those conditions in which its advent is usually gradual is a prognostic indication of considerable gravity.*

A. Causes of cyanosis in which the onset is usually GRADUAL:

1. *Chronic Bronchitis* with *Emphysema* is perhaps the commonest cause of cyanosis. It comes on quite gradually, and slowly increases; but any sudden increase in the degree of cyanosis is of grave import.

2. In *mitral* valvular disease more or less cyanosis is generally present, but never in uncomplicated *aortic* disease. So constant is this that it aids us considerably in deciding which valve is affected.

3. In primary disease of the *cardiac wall*, cyanosis, if it does occur, is a serious symptom.

4. Gradual compression of the lung by a large amount of *pleuritic effusion* prevents due expansion of the lungs, and therefore efficient aeration of the blood.

5. Gradual *compression of the bronchi* by enlarged glands, aneurysm, or other intrathoracic tumour, has the same effect.

6. Phthisis, or any other chronic infiltration of the lungs, as by cancer, diminishes their aerating surface.

7. The presence of large *abdominal tumours* forcing up the diaphragm prevents full expansion of the lungs, and consequently causes cyanosis.

8. *New growths* in the bronchi, such as cancer, or in the larynx, such as polypi, prevent the due entrance of air into the lungs.

9. *Erythræmia.*—This condition, first described in 1892 by Vaquez,[1] presents a triad of symptoms: (i.) Polycythæmia, or excess of red corpuscles in the blood, up to as many as 13,000,000 per cubic millimetre; (ii.) a large spleen; (iii.) cyanosis specially marked in dependent parts. The marrow has been found excessive in quantity.

10. *Sulphæmoglobinæmia*, a condition accompanied by headache, weakness, and constipation. The diagnosis can only be made by a spectroscopic examination of the blood.[2]

11. *Aniline Dye Poisoning.*—Workers in aniline dyes may develop cyanosis, nausea, vertigo, muscular pains, and tremors.

B. The most common causes of cyanosis which comes on RAPIDLY are:

1. An extensive pneumonia, especially when supervening upon emphysema; or acute bronchitis, especially in children. The cyanosis which accompanies an extensive pneumonia is due not so much to the fact that a large part of the respiratory area is rendered useless as that the healthy area does not have time to expand and take on the additional work. In more chronic diseases, such as phthisis, a much larger extent of lung may be diseased without any appearance of cyanosis.

2. Acute miliary tuberculosis of the pulmonary type, in which the cyanosis is often extreme.

3. Diphtheritic membrane invading the air-passages.

4. Spasmodic closure of the glottis (spasmodic croup).

5. Foreign body in the larynx, a condition which should always be borne in mind when cyanosis supervenes suddenly in children.

6. Œdema of the glottis.

7. Pressure on the trachea by an enlarged thyroid, etc.

8. Pneumothorax.

9. Paralysis of the respiratory muscles.

10. Paralysis of the abductor muscles of the vocal cords acts similarly, as in a case which I have recently seen in a patient suffering from tabes dorsalis. The laryngeal crisis took this form, and was attended by considerable danger to life.

11. *Methæmoglobinæmia*, in which cyanosis follows overdoses of certain drugs—antipyrin, sulphonal, veronal, chloral, antikamnia, potassium chlorate, amyl nitrite, potassium permanganate, and others. The spectroscope reveals the condition of the blood.

The *Treatment* must have reference to the cause; but in several cardiac conditions cyanosis, if unaccompanied by dropsy, is a distinct indication for venesection. But when marked anasarca is present, it indicates generally that the venous stasis is too great to admit of relief by this measure.

§ **27. A Sallow Hue** of the skin is characteristic of *aortic* valvular disease, which in this respect presents a marked contrast to the plethoric

[1] Osler, *Lancet*, 1908, vol. i. [2] See the *Lancet*, 1906, vol. ii., p. 72.

florid appearance of patients with *mitral* valvular disease. This sallowness is distinguished from jaundice by the absence of the yellow colour from the eyeballs and the absence of bile in the urine. True jaundice, however, does arise in cardiac disease as a symptom of the hepatic congestion, which is met with more often in mitral than in aortic disease.

§ **28. Pyrexia** and its concomitant symptoms (see Chapter XV.) are present in most of the *acute disorders* of heart and pericardium. The temperature in malignant endocarditis is of an intermittent or remittent type, with an irregular range, such as that met with in other forms of septicæmia.

§ **29. Sudden Death,** or death, say, within a few hours of the apparent commencement of the illness, is a frequent mode of termination of disease of the heart, and it may be the first symptom of disease of this organ. The chief conditions under which sudden death occurs are as follows. The first six of these have reference to the cardio-vascular system.

1. Among the various forms of *cardiac valvular disease*, sudden death is more frequent in aortic than in mitral disease. But sudden death, and, in general terms, the prognosis, depend more upon the condition of the wall than of the valves.
2. It is liable to occur in all forms of primary disease of the *cardiac wall—e.g.*, fatty and syphilitic heart (§§ 52 and 41).
3. It is a very common termination to *aortic aneurysm* (§ 56).
4. A patient may die with the first attack of *Angina Pectoris*.
5. Sudden profuse *hæmorrhage*, internal or external.
6. Pulmonary *embolism—e.g.*, from air in the veins (as in the performance of transfusion), or clotting in the heart.
7. The conditions which cause *Coma* may also result in death, which is relatively sudden (Chapter XIX.).
8. Nerve diseases which in their progress involve the *medulla* terminate suddenly; and thus, among the rarer causes, atlanto-axoid disease and syringomyelia may be mentioned.
9. Sudden emotion, injuries to the head, and other conditions acting on the *nervous system* by shock (Chapter XIX.).
10. Suddenly-acting *poisons*, such as prussic acid, a large dose of morphia or carbolic acid, aconite, veratria, etc.
11. Sudden rupture of a large cyst, an internal organ, or other cause of *Collapse* (Chapter XIX.).
12. Foreign bodies in the trachea, or other causes suddenly stopping the respiration (*asphyxia*).

B. PHYSICAL EXAMINATION.

§ **30. Landmarks of the Chest.**—There is a *ridge* on the sternum formed between the manubrium and the gladiolus which can always be felt, opposite the second costal cartilage (c.c.) ; and the other ribs can be counted from the second one. The *nipple* is usually situated just external to the fourth costal cartilage, near its junction with the rib ; it should correspond to a vertical line dropped from the middle of the clavicle. At the back, the *lower angle* of the scapula is near the seventh rib ; and the *scapular line* is a vertical line drawn through the inferior angle of the scapula. The position and relations of the heart can be studied in Fig. 11, which is a sketch taken from the cadaver. The various regions of the thorax named for convenience of reference are given in Fig. 33 in the chapter on Pulmonary Diseases. The ordinary methods employed for the physical examination of the heart, and the pericardial sac within which it is suspended, are INSPECTION, PALPATION, PERCUSSION, and AUSCULTATION. Modern surgical methods enable us occasionally to employ the method of ASPIRATION under due precautions, and RADIOGRAPHY is now in vogue.

§ **31. Inspection.**—Note, *first*, the attitude of the patient, and the amount of movement he can make without becoming *breathless*. Note also if there be any visible pulsation of the arteries and veins at the root of his neck. In some cases it is desirable to take a side view, and slight deviation from the normal can often be better observed by looking over the patient's shoulder.

Secondly, most useful information may be obtained by simply observing the aspect and appearance of the patient, for the pallor or sallowness of the skin in cases of *aortic* valvular disease presents so marked a contrast to the florid cheeks of *mitral* disease as to form a most valuable aid to diagnosis. Notice also if the dropsy of the ankles or back, which sooner or later attends mitral disease, is present.

Thirdly, carefully observe that part of the chest wall which lies in front of the heart. It is important to notice if there be any undue pulsation or bulging in any part of the chest. The forcible displaced apex-beat of cardiac hypertrophy ; the wavy, diffuse pulsation of cardiac dilatation and pericardial effusion ; the epigastric pulsation and throbbing jugular veins in cases of dilated right ventricle ; and the heaving carotid arteries in cases of aortic regurgitation, may each afford us valuable hints as to the direction of our further inquiries.

§ **32. Palpation and the Localisation of the Apex** (see Figs. 9 and 12).—The apex-beat is the farthest point to the left at which the cardiac impulse is distinctly felt, and after inspection, by which it can frequently be seen, should be first palpated by the flat of the hand, and then

localised with the finger tips. In an adult male it is normally situated about 1½ inches below and ½ inch to the inner side of the nipple-line, at a distance of about 3 inches from the mid-sternal line. *These and other cardiac measurements vary with the age*[1] *and proportions of the patient*—facts which are apt to be forgotten. The *most external* portion of the apex-beat should be marked by a dot with an aniline pencil. The localisation of the apex is a most important matter, and there are three principal features to observe about it—its POSITION, its FORCE, and its EXTENT. You may also note in passing whether a *thrill* can be felt with the flat of the hand. It is important to bear in mind that the apex-beat is considerably modified if the apex happen, as it not infrequently does, to pulsate *precisely behind a rib.* It is only when the apex beats in an intercostal space that these three features can be satisfactorily made out, and this source of fallacy should be remembered and allowed for. It can sometimes be felt more distinctly when the patient is asked to lean forward.

In HYPERTROPHY of the left ventricle the heart impulse is displaced downwards and outwards, and is forcible and heaving. In hypertrophy of the right ventricle there is pulsation in the epigastrium and in the lower interspaces, but the apex is in its normal site. With DILATATION the apex-beat is diffuse and wavy.

The apex is DISPLACED *downwards* in cases of emphysema or pleurisy with effusion ; if the latter be on the left side, the apex may even be displaced beyond the right border of the sternum (see Fig. 42). The apex is displaced *upwards* in pericardial effusion, retracted lung, abdominal tympanites, or with any abdominal tumour pushing up the diaphragm. The apex-beat is OBSCURED by very muscular or adipose chest walls, or emphysema. It is FEEBLE with fatty heart ; WAVY in pericardial effusion. With pericardial adhesions there is a *systolic retraction* of one or more interspaces.

THRILLS.—Of endocardial thrills the presystolic thrill *felt at the apex,* due to mitral stenosis, is the most common. More rarely there is also a thrill in the *pulmonary area* (Fig. 12), due to mitral stenosis. The systolic thrill at the *aortic area* due to aortic stenosis, when present, is very marked. A diastolic thrill can be felt over the *manubrium* in advanced cases of aneurysm or aortic regurgitation. Mitral regurgitation is occasionally accompanied by a *systolic thrill at the apex,* which is the commonest thrill felt in children. A widespread thrill may be present with pericarditis.

§ **33. Percussion** of the *superficial* area of the præcordial dulness—*i.e.*, area not covered by lung. In mapping out this area, the percussion

[1] The position of the heart is considerably modified in childhood. The left border comes out to the nipple line, and the right border extends to the right edge of the sternum ; the apex beats almost directly below the nipple, behind the fifth rib, or may be in the fourth interspace.

stroke should be *very much lighter* and more superficial than that applied when examining the lungs, or the liver, or the spleen, and other deep-seated solid organs (see Fig. 9). The superficial area which is here referred to is a triangular one, with the apex upwards. The measurements of the dull area in a person of average size are $3\frac{1}{2}$ inches transversely, from the mid-sternal line ; and $2\frac{1}{2}$ to 3 inches vertically along the left border of the sternum. Its boundaries are given in Fig. 9. The percussion note over the sternum is very different to that elicited over the chest beside it—it is of a much higher pitch—consequently, we

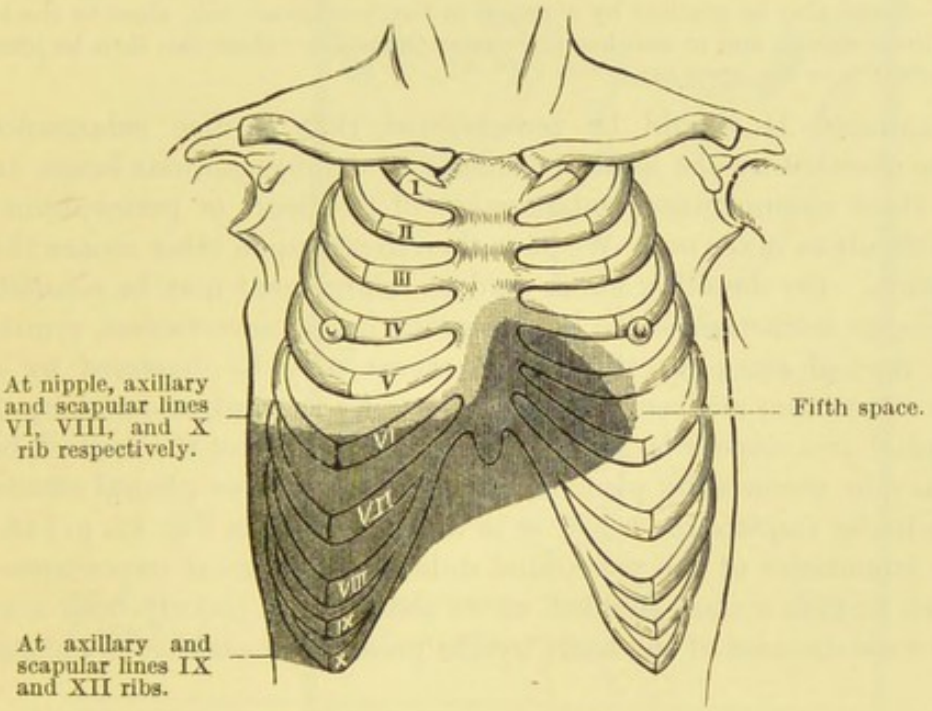

Fig. 9.—**Superficial and Deep Dulness of Heart and Liver.**—The superficial area of cardiac dulness is a triangular one, with the apex upwards. The measurements of this area in a person of average size are $3\frac{1}{2}$ inches transversely, and $2\frac{1}{2}$ to 3 inches vertically along the left border of the sternum. The right border begins at the level of the *fourth costal cartilage*, and corresponds to a vertical line drawn slightly to the left of the middle line of the sternum. The left limit starts from the same point, and runs outwards along the lower border of the fourth costal cartilage to nearly its junction with the rib, then bending downwards to apex-beat. The lower limit is continuous with the liver dulness.

cannot compare the percussion note in these two situations. We ought, therefore, to percuss upwards and downwards in a vertical line along the sternum to ascertain if any part of it is duller than normal.

The area of *deep-seated* cardiac dulness is $\frac{3}{4}$ inch larger on each side, and 1 inch larger upwards, than the superficial area. Its limits are considered by most physicians to be less certain, and therefore less useful for diagnosis, than the superficial area. In some schools, however, it has superseded the superficial cardiac dulness, and is held to denote more accurately the variations in size of the heart.

Method.—The student should lose no opportunity of PERCUSSING THE NORMAL heart and of attending to the following points : (i.) *Having first localised the apex-beat*, begin outside the cardiac area in a perfectly resonant area. The middle finger of the left hand should be placed flat and *firmly* upon the chest wall parallel to the margin of dulness to be made out, and moved $\frac{1}{4}$ inch at a time, *always*

parallel to that margin, towards the centre of the heart. (ii.) Use only one finger—the second of the right hand—as a hammer, making a short sharp tap with the finger *tip*. The percussing finger should rebound immediately—"staccato," as pianists say. The movement should be made from the *wrist*, or from the knuckle (metacarpo-phalangeal joint), as in playing the piano, and the tap should be a light one. (iii.) By listening attentively to the sound elicited, it will be noticed that it is dull and flat over the heart, like that produced by striking any solid object; but louder and more resonant outside the area, like the sound produced by striking an empty barrel. It is only possible to define in this way the right, the upper, and the left limits of the dull area, because at the lower limit the cardiac dulness is continuous with that of the liver. Mark with a blue aniline pencil the right or sternal border in two places. The curved upper and left border of the dulness should also be marked by a pencil in two positions—viz., close to the left side of the sternum, and in another place near the nipple; these can then be joined and continued to the apex-beat.

FALLACIES.—It should be remembered that cardiac enlargement may be *obscured* by the hyper-resonance of emphysematous lungs, and under these circumstances enlargement of the heart or pericardium is very difficult to make out. We have then to rely upon other means than percussion. On the other hand, cardiac enlargement may be *simulated* by a fibrous retraction of the left lung, the heart, nevertheless, remaining of normal size; or, thirdly, the heart may be *displaced* by an aneurysm or other mediastinal tumour pushing forward, and making the præcordial area appear larger. One or other border of the area of dulness may be *obscured* by pleuritic effusion. Ascites or pleural effusion may actually *displace* the heart, as in the case given in Fig. 42, p. 148.

The boundaries of the præcordial dulness are of great importance—so much so that we are enabled, as we shall see, to classify both acute and chronic diseases of the heart by the presence or absence of enlargement.

The CHIEF CAUSES OF ENLARGEMENT of the præcordial area of dulness are two in number—EFFUSION into the pericardium, and ENLARGEMENT of the heart. The latter may be due to *hypertrophy* or to *dilatation*, or, more commonly, to a combination of the two. This enlargement may involve any one, or more than one, of the cavities of the heart.

§ **34.** For **Auscultation** much practice is required, and once more I must warn the student never to miss an opportunity of listening to the sounds of the heart, particularly the *normal* sounds. The whole cardiac cycle—*i.e.*, contraction (systole) and dilatation (diastole)—occupies about one second (Fig. 10). The first sound is due to the contraction of the ventricular muscle or to the closure of the mitral and tricuspid valves (or to both), and occupies about $\frac{4}{10}$ second; then comes a very brief interval, say $\frac{1}{10}$ second; followed by the second sound ($\frac{2}{10}$ second), which is due to the closure of the aortic and pulmonary valves; and, finally, the diastolic interval ($\frac{3}{10}$ second). (Fig. 10 should be studied carefully by the student.)

Methods.—If no stethoscope is handy, cover the patient's skin with a soft handkerchief, preferably silk, and apply the ear ; but a stethoscope localises the sounds better. Personally, I prefer the old-fashioned wooden stethoscope, if the earpiece fits my ear well. But the binaural stethoscope is very useful for examining

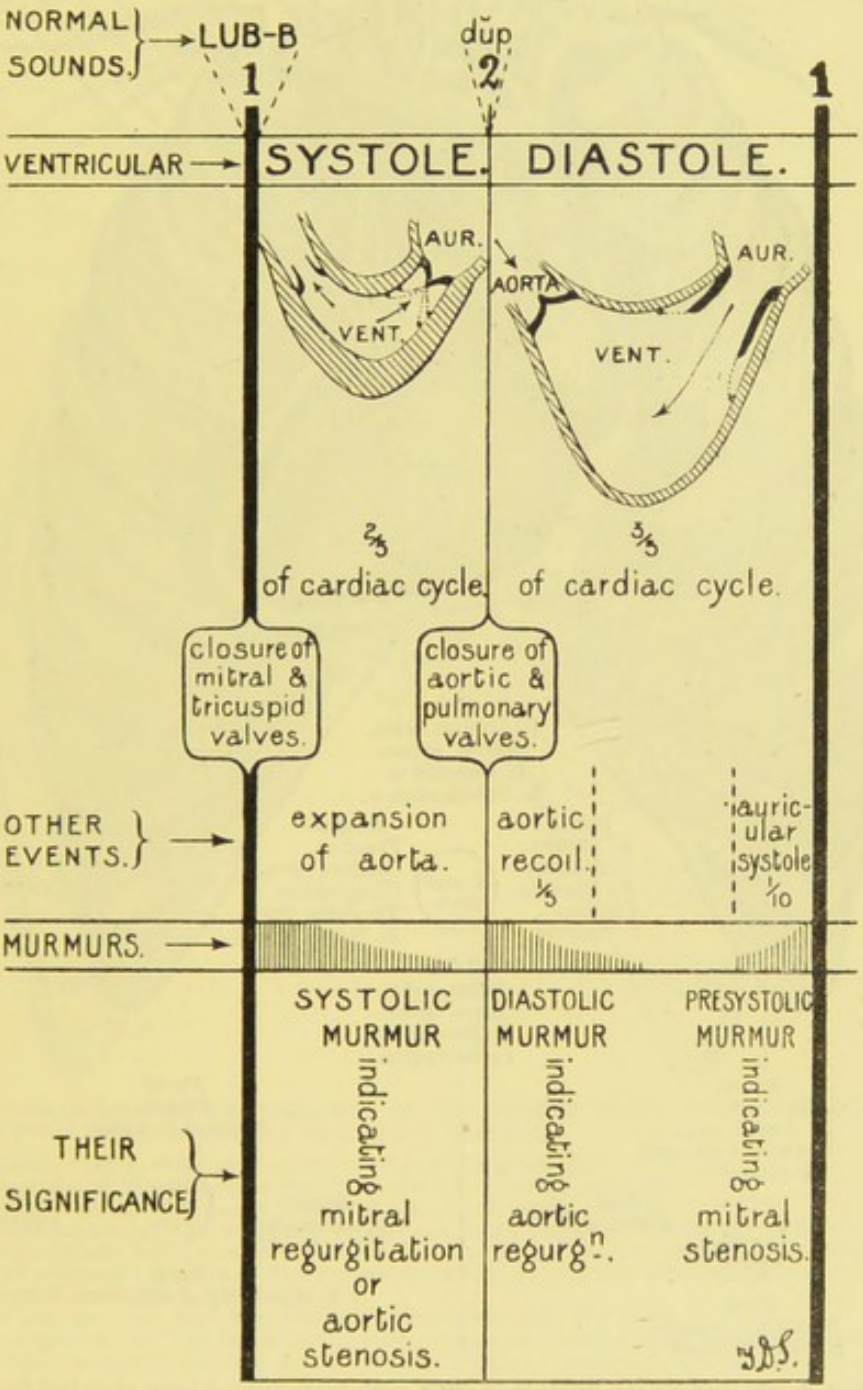

Fig. 10.—Diagram of a **Cardiac Cycle**, showing various events and their duration, how the different murmurs are produced, and their clinical significance. The student should study this and Fig. 12 very closely.

infants, or whenever there is any noise in the room or in the street, though it always has the disadvantage of giving more resonance tones (echoes arising in the cup and tubes). Place the small end of the stethoscope on the chest over the apex-beat, *so that the rim touches all round*, and then adjust *your ear to the stethoscope*, not the stethoscope to your ear. The common fault is to allow one side of

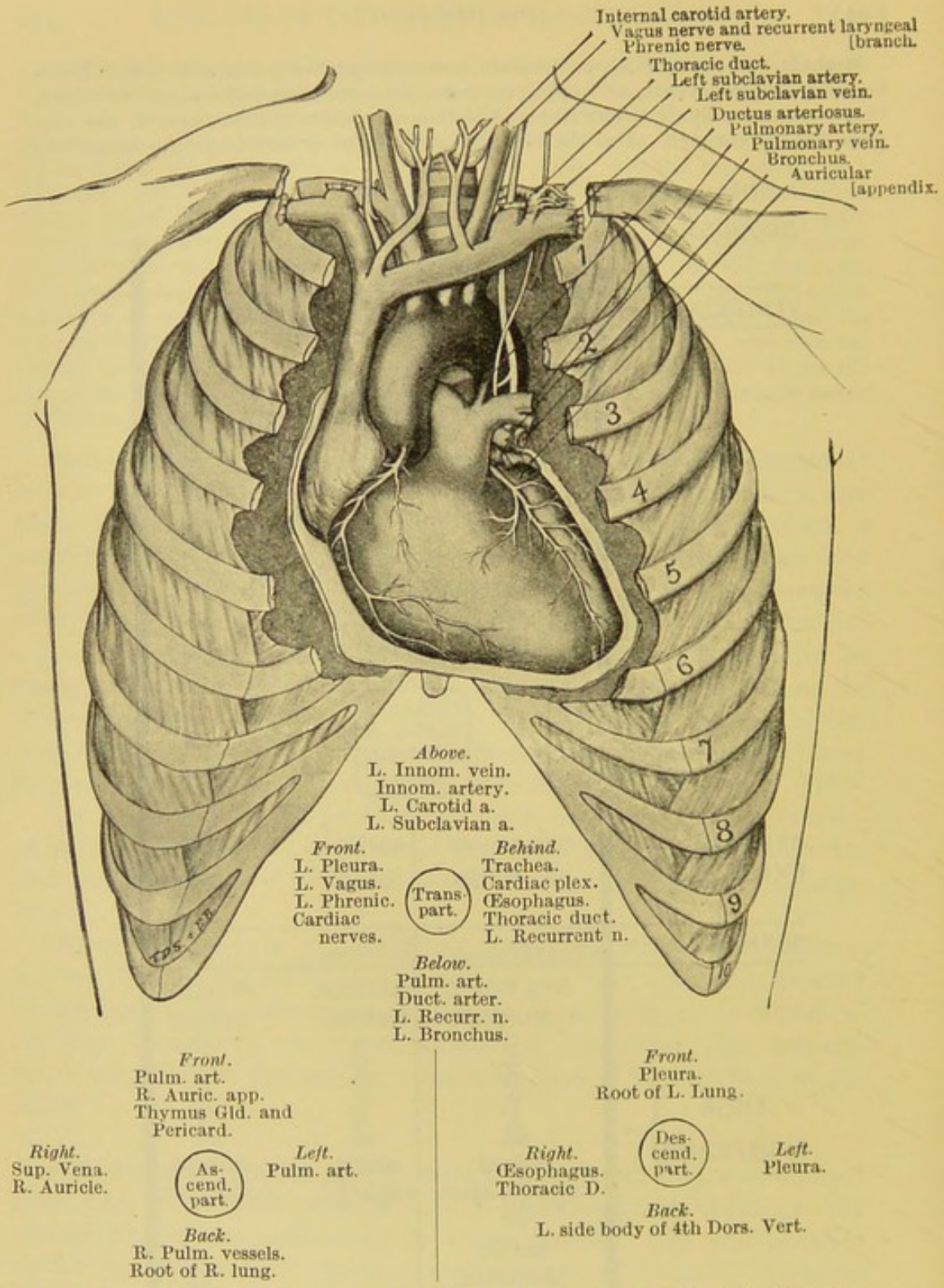

Fig. 11.—**The Heart and Great Vessels in Situ**, with lungs turned back, sketched from the cadaver. Right ventricle forms greater part of the anterior surface of the heart. Above and to right of this is the right auricle, into which the superior vena cava opens, which collects the blood from the two innominate veins. Passing out from and above the right ventricle is the pulmonary artery, above which again is the remains of the ductus arteriosus, connecting it with the arch of the aorta. Just to the left of the pulmonary artery the left auricular appendix peeps round the corner. The arch of the aorta is seen coming forward from the left ventricle (which is at back, and therefore only seen at left margin of heart), and from its upper convexity arise in order the innominate artery, left carotid, and left subclavian. The trachea is seen behind the vessels, and the phrenic and vagi nerves are seen at the sides, those on the left passing down in front of the aorta behind the root of the left lung. The relations of the ascending, descending, and transverse portion of the aorta are given diagrammatically above.

the chest end to be raised off the chest in the process of adjusting the stethoscope to the ear. Do not allow the weight of your head to rest on the stethoscope; it pains the patient, and you do not hear as well. It is a good plan to keep your fingers on the pulse (wrist or carotid) whilst auscultating.

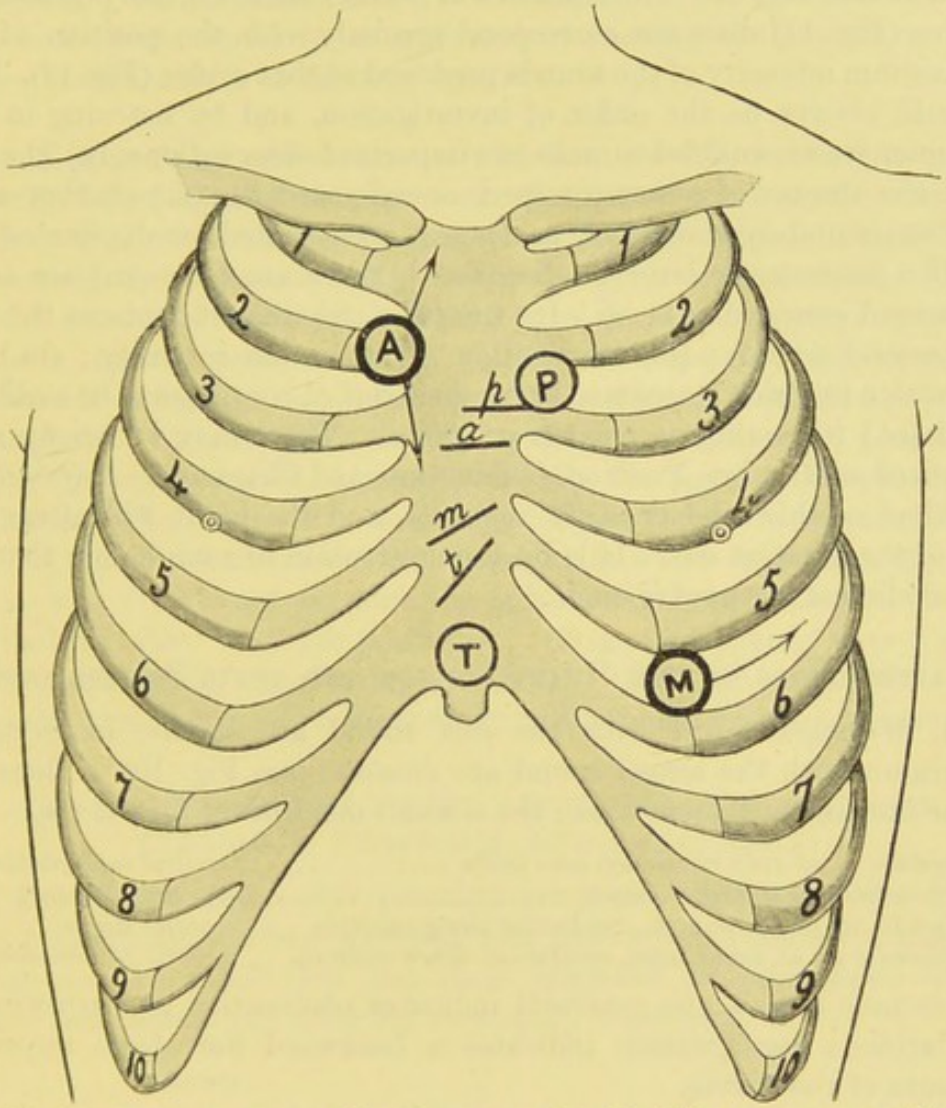

Fig. 12.—Diagram showing the **situation** of the **Cardiac Valves** and the **position** in which the several **murmurs** are heard loudest.

p = Pulmonary orifice, at level of upper border of third left costal cartilage.
a = Aortic orifice at level of lower border of third left costal cartilage.
m = Mitral orifice at level of lower border of fourth left costal cartilage.
t = Tricuspid orifice at level of fourth interspace, lying obliquely behind the sternum.
The positions where the sounds produced at the various orifices are best heard are indicated by the letters enclosed in circles. The arrows mark the direction in which murmurs produced at the corresponding orifices are conducted.
M, Mitral murmurs are best heard at the mitral area—*i.e.*, the apex.
A, Aortic murmurs are best heart at the aortic area—*i.e.*, second right costal cartilage.
P, Pulmonary murmurs are best heard at the pulmonary area—*i.e.*, second left intercostal space.
T, Tricuspid murmurs are best heard at the tricuspid area—*i.e.*, at lower end of sternum.

Listen, *first*, to the sounds at the *apex* and all round its neighbourhood. Notice that the first sound is normally longer and duller than the second,[1] and that the two sounds somewhat resemble lubb

[1] The first and second sounds correspond to G and B-flat respectively, below the middle octave.

($\frac{4}{10}$ second), dup ($\frac{2}{10}$ second). Then, *secondly*, listen at the base; place the instrument over the second right intercostal space, close to the sternum, over the aorta; listen at that spot, and in other places around, as indicated in Fig. 12. The student will notice that the actual position of a valve (Fig. 11) does not correspond precisely with the position of the maximum intensity of the sounds produced at that orifice (Fig. 12). This should always be the order of investigation, and by listening in this manner, we are enabled to make two important observations: (i.) The presence or absence of a MURMUR (peri- or endo-cardial); (ii.) whether either SOUND is unduly shortened, prolonged, accentuated, or duplicated.

If a murmur or *bruit* be discovered, there are four qualities to be observed concerning it: (i.) Its time (*i.e.*, whether it replaces the first or second sound); (ii.) its position of maximum intensity; (iii.) the direction in which it seems to be conducted (*i.e.*, continues to be audible); and (iv.) its quality as regards roughness. These may be briefly summarised as Rhythm, Position, Conduction, and Character. A systematic method of this kind is easily acquired, and the habit will afterwards be of the greatest use. It is by the cultivation of such habits that the good clinical observer is made.

ALTERATIONS OF THE HEART SOUNDS AND THEIR SIGNIFICANCE.

1. *Murmurs* heard with the *first* sound are *systolic* in rhythm; murmurs with the *second* sound are *diastolic* (see Fig. 10). There are four fundamental facts which the student must never forget, viz.:

Systolic m. at apex continued into axilla ..... = *mitral regurgitation;*
Presystolic m. limited to apex ..... = *mitral stenosis;*
Systolic m. in aortic area, conducted along carotids .... = *aortic stenosis;*
Diastolic m. at aortic area, conducted down sternum .. = *aortic regurgitation.*

Stenosis (στενοω, to contract) indicates obstruction or narrowing of an orifice; regurgitation indicates a backward flow from imperfect closure of the valves.

2. *Accentuated second* sound in the *aortic* area, at the base of the heart, is met with when the arterial tension is high (§ 61), or in aortic aneurysm. In the *pulmonary* area an accentuated second is due to increased blood-pressure in the lungs, as in mitral valve disease.

3. A *reduplicated second* sound at the *base* of the heart is found when the aortic and pulmonary valves do not close synchronously, as when the pressure in either the arterial or the pulmonary system is unduly high (as in 2). When heard a little to the right of the *apex* it is very characteristic of mitral stenosis. A *reduplicated first* at the apex is sometimes found with high arterial tension.

4. *Short, clear, sharp sounds* are found with cardiac dilatation, while *feeble* sounds occur with fatty heart. Emphysema, excess of adipose tissue, and pericardial effusion, obscure the sounds, and give the impression of feebleness.

5. A *prolongation* of the first or second heart sound is sometimes spoken of, but it is difficult to know where to draw the line between a prolongation and a murmur. A "booming" first is found with hypertrophy.

FALLACIES.—Under ordinary circumstances the respiratory do not interfere with the cardiac sounds, but if they do, the patient should be

asked to stop breathing for a few seconds. It is often wise to do this in any case, because sounds originating in the bronchi or pleura may be mistaken for cardiac murmurs, but *they cease when the patient stops breathing*. On the other hand, if a murmur be very feeble and doubtful, the heart sounds may be exaggerated by causing the patient to take some exertion, such as running upstairs.

§ **35. The Pulse** affords one of the best possible indications as to how the heart is performing its work, as to what remedies are indicated, and as to how the therapeutic measures we have already adopted are answering their purpose. The pulse also gives an important clue as to the nature of the cardiac lesion ; thus, in mitral regurgitation the pulse is usually rapid, irregular, and of low tension, whilst in mitral stenosis it is regular, but small, thready, and incompressible ; in aortic regurgitation it presents a collapsing (" water hammer ") character, whilst in aortic stenosis it presents exactly the opposite feature, being sustained. The pulse will be dealt with fully in a separate chapter (Chapter V.), but there are three important hints which may be mentioned here—viz., (1) Do not examine the pulse until the nervousness at first excited by your visit has passed off ; (2) generally keep your fingers on the pulse while auscultating the heart ; (3) in all cardiac cases the rate, rhythm, force, and tension of the pulse should be frequently noted.

C. DISEASES OF THE HEART AND PERICARDIUM : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **36. Classification.**—For practical purposes, diseases of the heart and pericardium are best grouped into ACUTE and CHRONIC ; and the latter may be subdivided into those attended by *enlargement of the præcordial dulness* and those *not necessarily so attended*.

Acute.	**Chronic.**
	AREA OF DULNESS INCREASED.
I. Acute pericarditis.	I. Cardiac hypertrophy.
II. Acute endocarditis.	II. Cardiac dilatation.
III. Neuro-palpitation (paroxysmal tachycardia).	III. Hydropericardium.
IV. Angina pectoris.	IV. Congenital heart disease.
	V. Aortic aneurysm.[1]
	AREA OF DULNESS NOT NECESSARILY INCREASED.
	I. Chronic valvular disease.
	II. Fatty heart.

[1] This is not a disease of the heart proper, but is included here because its existence is often revealed by finding enlargement of the præcordial dulness, or dulness above, merging into that of the heart.

§ **37. Routine Procedure.**—First: What is the Patient's "Leading Symptom"? It may be that the patient voluntarily complains of one of the symptoms discussed in Section A, and in this way has directed our attention to his heart. If not, we must ascertain, without putting "leading questions," what is his "chief symptom." Breathlessness is the most constant symptom in cardiac disease, and in more advanced stages we meet with dropsy and cyanosis.

Secondly: Having obtained this clue we follow it up by asking a few details, in chronological order, of the History of the Illness. In this way we *ascertain whether the disease be acute or chronic*—a most important matter, because for clinical purposes diseases of the heart and pericardium may be primarily divided into acute and chronic. The Previous and Family Histories may also be inquired into. In all cardiac cases it is important to know whether the patient has ever had acute rheumatism, this being the most frequent cause of cardiac valvular disease. Ascertain also if there be any heart disease in the family.

Thirdly: The Examination of the Heart, and especially the decision as to *whether there is any enlargement or not.* The routine method consists of the following procedures:—

1. An examination of the apex-beat (by *inspection* and *palpitation*);
2. The mapping out of the area of præcordial dulness (by *percussion*) (see Fig. 9);
3. Listening to the heart sounds (*auscultation*); and
4. The examination of the *pulse.*

The chest should always be stripped and a thorough examination made. An attempt to examine a female patient should never be made without the removal of the corsets.

If the symptoms of which the patient complains point to some **Chronic Cardiac Disease**—*i.e.*, they have come on gradually and are unattended by pyrexia or other constitutional disturbance—the reader should turn to the **Chronic Diseases** (§ 42).

If, on the other hand, the disease is of an **Acute** character—*i.e.*, it has come on recently or suddenly, and is perhaps attended by pyrexia and other constitutional symptoms—it is one of four diseases: I. Acute Pericarditis; II. Acute Endocarditis; III. Paroxysmal Tachycardia; or IV. Angina Pectoris.

I. *The patient is in evident distress, and the præcordial area of* **dulness is increased,** *the shape of the dulness being* pyramidal, *with the point upwards, and the temperature is elevated. The disease is probably* Acute Pericarditis.

§ **38. Acute Pericarditis** is an acute inflammation of the pericardial sac. The disease has two stages: the *first* precedes, and the *second* follows, the effusion of fluid into the pericardial sac. It is not frequently

met with as a primary affection. It supervenes during the course of many different diseases, and the symptoms of these may mask its onset. Rheumatic fever is certainly its most common cause, and it should be remembered that it may be the first manifestation of this affection. We should always examine the heart daily in rheumatic fever, and in acute renal affections, because in these acute pericarditis may come on insidiously, without pain or tenderness, its advent being marked perhaps only by the occurrence of delirium, so rare otherwise in acute rheumatism.

Symptoms.—(1) The patient wears an anxious, troubled look, and the cheeks are flushed ; there are fever and a rapid pulse ; the breathing is rapid, and he complains of severe pain over the heart, increased by pressure, movement, or respiration. (2) *Physical Signs.*—The præcordial dulness is only slightly increased at first, but a loud, harsh *double* friction sound, " to and fro," like a saw, is heard on auscultation. This may be distinguished from a murmur produced *within* the heart by (i.) always being double (*i.e.*, accompanying the movements of the heart) ; (ii.) the second part of the rub is continuous with the first, without any diastolic pause ; (iii.) it is often loudest at the root of the great vessels, over the third left costal cartilage ; (iv.) it varies in its character from time to time, and is increased by gentle pressure ; (v.) pressure will also elicit another differential character—viz., that the disease is usually accompanied by tenderness, as well as pain. The differentiation between peri- and endo-cardial murmurs is so important that it is also given in a tabular form below (p. 57). To distinguish pericardial from pleuritic friction is very easy, because the latter ceases if the patient holds his breath. Note that as the effusion occurs the murmur becomes less distinct, but it is again intensified as the effusion clears up.

(3) *Second Stage*, or stage of pericardial effusion. The inflammation may subside, but more frequently, in the course of a day or two, effusion of fluid occurs, and the pain and tenderness diminish. The rub becomes less audible, though it can still be heard at the root of the heart. The temperature may fall a little, but the breathlessness and other symptoms continue. A troublesome cough is frequently added, and vomiting sometimes occurs. *The increased area of dulness*, due to pericardial effusion, may be greater than the enlargement from any other cause. (i.) It is of *triangular shape*, with apex upwards, reaching to the third, or even second, costal cartilage. (ii.) As the root of the heart is fixed to the pericardium, when the sac fills the whole heart becomes raised, and, therefore, *the apex beats above and to the left of its normal position.* (iii.) The *dulness extends to the left* of the apex-beat. The heart sounds may be diminished at this time, because they are transmitted through fluid. It is possible by the height of the dulness along the sternum, which should be watched each day, to determine the amount of fluid present.

Etiology.—Pericarditis may attack any age and either sex, but is almost always preceded and caused by some other disease. It is doubtful if idiopathic pericarditis ever occurs. The causes of pericarditis may be ranged under five heads: (1) *Injury.* (2) *Certain acute infections:* acute rheumatism, pyæmia (staphylococcal, streptococcal, pneumococcal), scarlet fever, variola, typhus, typhoid, and influenza (Barlow), and some constitutional diseases—Bright's disease, scurvy, gout. (3) *Chronic infections—e.g.*, tubercle. (4) *Morbid growths—e.g.*, cancer. In the two last the process tends to be subacute, and is accompanied by a large amount of fluid. (5) *Extension* from adjacent organs, amongst which may be mentioned pleurisy or pleuro-pneumonia, especially on the left side; intrathoracic aneurysm (pericarditis

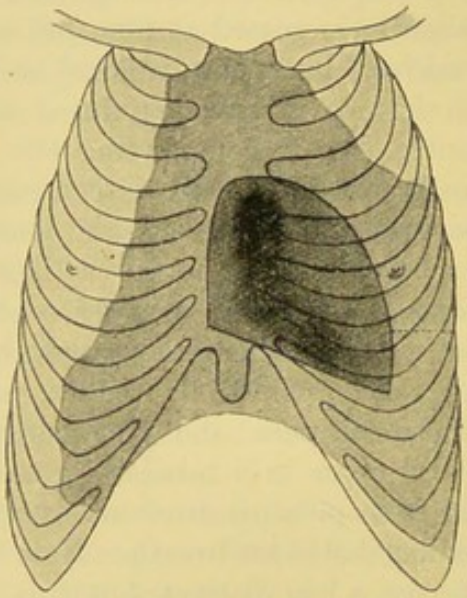

Fig. 13.—Diagram from a Case of Rheumatic Pericarditis with Effusion.—Eliza P., aged twenty-seven. The dark area indicates præcordial dulness. The larger area of lighter shading gives the area over which pericardial friction could be heard (the loudness is indicated by the depths of the shading). It is usually taught that one of the features distinguishing peri- from endo-cardial murmurs is the limitation of the former to the præcordial region; but I have many times satisfied myself that this is not so, and this case is one of several examples I have met with verified by autopsy. This case was under the care of Dr. W. M. Ord when I was his house-physician.

may be the precursor of rupture into the pericardium); solid intrathoracic tumours; perforating ulcer of the œsophagus; various diseases below the diaphragm—*e.g.*, hydatid or abscess of liver.

Course and Prognosis.—The duration of acute pericarditis with effusion varies widely, but it averages about fifteen to twenty-five days. It may undergo resolution with or without the formation of adhesions (Adherent Pericardium § 38*c* below); or result in chronic effusion (Hydropericardium, § 45); or become purulent (Pyopericardium § 38*a* below). Pericarditis with effusion is always a serious malady, but the prognosis depends much on the disease which it complicates, the general condition of the patient, and the evidences of cardiac embarrassment—namely, feebleness, rapidity, and irregularity of the pulse. Pericarditis com-

plicating rheumatism, like the other complications of that disease, tends to recover, but it may leave a weakened heart, and lead to cardiac dilatation. In renal disease it is a serious though often latent affection ; and in pyæmia, when it is generally purulent, it adds to the gravity of that serious disorder. In infancy and in debilitated patients it is also grave.

Treatment.—In the inflammatory stage the patient should be kept in bed absolutely *without movement*, on light fluid diet ; and cotton-wool, a poultice, or warm fomentation applied to the præcordium. This usually gives more relief than the ice-bag which is recommended by Dr. Lees, though this undoubtedly relieves the symptoms. If the distress is great, wonderful relief is obtained from four or five leeches over the præcordium. If cyanosis, orthopnœa, and irregular pulse are present, indicating considerable cardiac embarrassment, bleeding (4 to 6 ounces) is a prompt and efficacious measure. Opium (gr. $\frac{1}{4}$ quartis horis), or morphia hypodermically, is of great value for the pain and distress. Small doses of chloral may be given for the restlessness if the circulation is well maintained. Effervescing salines should be administered. Digitalis is given in small, frequent doses for cardiac failure, and stimulants, ether, and strychnine, according to the state of the pulse. The effect of digitalis must be very carefully watched, lest it increase the cardiac embarrassment. For hyperpyrexia and delirium some recommend the graduated bath, but the necessary movement is a grave objection ; and we have remedies quite as valuable in antifebrin, phenacetin, aconite, quinine, and opium.

Treatment for the cause of the pericarditis should be combined with the foregoing—*e.g.*, sodium salicylate combined with alkalies for acute rheumatism ; diuretics and hot-air baths for renal disease ; quinine in large doses for pyæmia. In the stage of effusion free blistering promotes absorption, but it must be remembered that renal disease is a contra-indication to blistering. If the effusion becomes chronic, potassium iodide (gr. v. t.i.d.) and diuretics may be given (F. 55). Iodine paint and other local counter-irritants are also useful.

Paracentesis Pericardii.—If, at any time, the effusion be considerable, and the cardiac embarrassment very great, as evidenced by severe dyspnœa, and a rapid, irregular, low-tension pulse ; if leeches and bleeding have failed to give relief, exploration with a hypodermic syringe, under strictly antiseptic precautions, may be practised *to ascertain the nature of the fluid*.[1] If clear fluid be found, paracentesis should be performed, and the operation may be done without fear, if rigid antiseptic precautions be employed, and the point of the trocar kept away from the viscus. Incise the integument in the fifth left interspace, close to the sixth rib, 2 to 2¼ inches to the left of the middle of the sternum (in an adult of

[1] The operation of paracentesis has now been performed sufficiently often to make it trustworthy. See cases reported by H. B. Robinson, *Brit. Med. Journ.*, 1898, vol. ii., p. 1605 ; and Foxwell, *Brit. Med. Journ.*, 1897, vol. i., p. 1350 ; and footnote, p. 56.

average size). Insert the trocar and cannula perpendicularly to the surface; withdraw the former directly it pierces the wall. Eight or twelve, or even forty ounces (in a chronic case) may be thus gradually removed.

§ 38*a*. **Pyopericarditis.**—Sometimes in debilitated children, and in the course of scarlatina, in phthisis and empyema, always in the pericarditis of pyæmia, and under some other conditions, the fluid in the pericardium takes on a purulent or sero-purulent character. This condition is sometimes revealed (as in a collection of pus in other parts of the body) by the occurrence of (1) shivering attacks, (2) profuse perspirations, and (3) a temperature with wide variations in the course of a few hours, in addition to the clinical features of acute pericarditis above described. But it is very difficult to diagnose, because the *friction sound is usually absent*. It is usually fatal.

Pyopericarditis is the form which pericarditis frequently assumes in infancy, and is then extremely difficult to diagnose. In addition to the small measurements with which we have to deal, the left lung may become adherent to the chest wall early in the disease, and so prevent the recognition of the enlargement of the præcordial dulness. It is only by the profound disturbance of the circulation, and the progressive weakness and anæmia, that we can assume the presence of pus.

Treatment.—Quinine in large doses, phenacetin, and like remedies, may be administered, but a large hypodermic syringe, rendered thoroughly antiseptic, should be very carefully introduced whenever the existence of pyopericardium is suspected. If the fluid withdrawn be of a purulent nature, paracentesis, or, better still, free drainage, should be effected.[1]

§ 38*b*. **Latent Pericarditis**—*i.e.*, pericarditis without *symptoms* (though not necessarily without physical signs). In most patients in whom we find a pericardial effusion a history of acute pericarditis is obtainable; but it is a fact not sufficiently recognised that pericarditis may have come on quite insidiously, without any acute symptoms. The effusion may be discovered when examining the heart as a matter of routine, or perhaps not until the autopsy. Nevertheless, in such cases the presence of flakes of fibrin, and the dull thickened serous surfaces, reveal its inflammatory origin. Moreover, I have, in the post-mortem room, on more than one occasion found a totally adherent pericardium in a patient in whom the most careful inquiry had failed to reveal any symptoms pointing to the heart during life. It is a latent pericarditis of this kind which ordinarily complicates Renal Disease. In Acute Rheumatism also *its advent may be indicated only by delirium or vomiting;* and Gouty persons also may be attacked by this latent disease after exposure to chill.

Pericarditis occasionally results in Adherent Pericardium.

§ 38*c*. **Adherent Pericardium** may exist in two forms: (i.) The *internal*, in which the visceral and parietal layers become joined, so that the heart is shut up, as it were, in a box, and is incapable of much hypertrophy. The symptoms are those of grave cardiac failure, which usually come on severely, and often end fatally at about puberty, when the heart should increase proportionately with the general development.

(ii.) *External*, in which the pericardium is adherent to the surrounding structures, the pleura, mediastinum, and diaphragm. The symptoms are those of cardiac embarrassment out of proportion to the signs of heart disease found. The signs are many, but not very reliable. They are (1) a systolic tug at the apex; (2) fixity of the cardiac apex with change of position and during respiration; (3) systolic recession along the attachments of the diaphragm, either in front along the lower costal border, or behind under the eleventh and twelfth ribs; (4) signs of hypertrophy, greater than can be accounted for by the severity of any valve disease which may be present; (5) pulsus paradoxus, or stopping of the pulse during inspiration.

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[1] See Discussion at Clin. Soc., the *Lancet*, December 22, 1900.

We now pass to the other acute disorders—viz., II. Acute Endocarditis ; III. Neuro-palpitation ; and IV. Angina Pectoris.

II. *The præcordial area of* **dulness is not necessarily increased,** *the patient is in evident distress, his* temperature is elevated, *and on auscultating the chest there is a* murmur *added to the heart sounds—the disease is probably* Acute Endocarditis. *It is not always easy to distinguish endocardial from pericardial murmurs* (see Table below).

Table II.—Diagnosis of Endocardial from Pericardial Murmurs.

Endocardial Murmurs.	Pericardial Murmurs.
1. May accompany first or second sound only, or both. If double, there is always a short interval of silence between the two bruits.	Always double, and can be heard throughout the diastole, as well as the systole, without any interval between the two bruits.
2. Loudest in one of the valvular areas.	Usually loudest over third left costal cartilage (root of big vessels).
3. May be conducted into the axilla, or along the aorta and carotids.	Mostly confined to the præcordium.[1]
4. Usually no pain or tenderness.	Usually accompanied by pain.

§ **39. Acute Endocarditis** is acute inflammation of the lining membrane of the heart. It is usually attended by an almost imperceptible enlargement of the præcordial dulness, because a degree of dilatation or myocarditis is associated with it. In a very large proportion of cases it complicates some other disease ; and, like pericarditis, it is very frequently associated with acute rheumatism ; it may even be the first evidence of that disease.

There are two varieties of endocarditis, commonly known as Simple and Malignant, and there are three groups of symptoms found with each.

In Simple or Benign Endocarditis, as in the other variety, (1) the characteristic feature is the *development of a murmur*, usually heard loudest at the apex because the mitral valve is the one most frequently involved in acute rheumatism ; but it may be heard in any situation, depending on the valve affected (see p. 49), and it may be single or double in rhythm. The murmur has to be diagnosed from that of pericarditis (see Table above), and, if possible, from that due to old valvular disease. In the acute disease the murmur is usually softer and heard over a more limited area ; in old valvular disease it is harsher, and is conducted in different directions (*vide* Cardiac Valvular Disease,

[1] For an exception to this, see Fig. 13, p. 54.

§ 47). The previous history, and the presence of cardiac dilatation, may also aid us considerably. The other physical signs which are present are a weak, diffuse impulse and weak cardiac sounds.

2. The *Constitutional Symptoms* may be so few and slight that at the time they may pass almost unnoticed. But since simple endocarditis usually complicates some other disease (*e.g.*, acute rheumatism), the constitutional symptoms largely depend upon the severity or mildness of the primary disease. The onset of the endocarditis in these circumstances may be suspected when there is a sudden increase in the rapidity of the heart, and an additional rise of temperature without apparent cause. Palpitation may be present, but pain and distress about the præcordium are generally absent—a feature worth bearing in mind. In the rare instances in which acute endocarditis occurs primarily, the temperature is irregularly intermittent (100° to 102° F.). The presence of such a pyrexia, and the absence of physical signs, excepting those referable to the heart, are the only data upon which we can rely for the *diagnosis* of the disease.

3. *Emboli* do not usually occur, at any rate, until very late, in *simple* endocarditis attacking a heart previously healthy. But when it attacks a heart the seat of old valvular mischief—known sometimes as Recurrent Endocarditis—the temperature may vary from 100° to 102° F. for days, weeks, or even months, and emboli may arise in various situations from the separation of the inflammatory material on the valves. Rigors, with tenderness and enlargement of the spleen, may indicate embolism of that organ; sudden hemiplegia or other nerve troubles may point to embolism in the brain; sudden occurrence of bloody albuminous urine, with a rigor, point to embolism in the kidney; sudden blindness, to embolism of the central artery of the retina; and sudden pain and tenderness in a leg or arm may indicate plugging of one of the arteries, in which case the pulsation will be absent below the blockage.

Causes of Benign Endocarditis.—A history, or evidence at the time, of the causes of endocarditis may help us in the diagnosis. (i.) Undoubtedly the most common of these is rheumatic fever, old or recent, and it should be remembered that acute endocarditis may arise quite early in the course of the disease, before the joint lesions are manifest. Exposure to cold is mentioned as a cause, but the endocarditis in such cases is probably of a rheumatic kind. (ii.) Chorea, scarlatina, typhoid, and many other bacterial infections, may give rise to endocarditis. (iii.) It is also an occasional complication of syphilitic,[1] cancerous,

[1] H. L., a lad aged fifteen, was admitted into the Croydon Hospital in 1882 with intense chlorosis, intermitting pyrexia, and a loud endocardial murmur. The cause of his illness was obscure during life, but he died gradually of asthenia. After death gummata were found involving the cranial and other bones. There were striæ in the cornea, and other evidences of syphilis, and abundant evidence of acute recent endocarditis, and a generalised endarteritis. This case is referred to at greater length in the *Clinical Journal*, December 1, 1897.

and other cachectic conditions, chronic alcoholism, and renal disease. (iv.) Valves deformed by acute or chronic endocarditis are predisposed to acute inflammation, and the recurrent endocarditis above referred to thus arises. (v.) The patient is generally young, rarely older than thirty-five or forty when attacked by endocarditis for the first time. (vi.) Heredity is an important predisposing factor.

The *Diagnosis* of benign endocarditis has been referred to above (under the constitutional symptoms), and it is not usually difficult. It is most important, however, to distinguish the two forms of endocarditis, as they differ so widely in their duration and fatality. Malignant or ulcerative endocarditis differs clinically (1) in the greater severity of the constitutional symptoms, which may present all the features of septicæmia or of the typhoid state ; (2) in the wide range of the temperature in the course of twelve or twenty-four hours, and the occurrence of severe rigors and sweats ; (3) in the invariable occurrence of systemic emboli, which may be of an infective character. When, however, malignant endocarditis supervenes on a previously damaged heart the diagnosis may become extremely difficult.

The *Prognosis*, though the malady may last for many weeks, or even months, is favourable as regards life, but the damage to the cardiac valves is generally permanent, and then the prognosis turns on many important considerations (§ 50).

Treatment should be directed primarily to the disease of which endocarditis is a complication—salicylate of soda, for instance, for rheumatic fever, though this drug is usually thought to have no control over the cardiac lesion. *Perfect rest*—hardly allowing the patient to turn in bed—is absolutely essential. This not only favours the subsidence of the inflammation, but prevents the violence of cardiac action, which separates the fragments from the valves and leads to embolism. Aconite is of great value to slow and steady the heart. In this and other respects the treatment is much like that of pericarditis (§ 38), though the local treatment has less effect in endocarditis. Stimulants and digitalis are indicated only if the heart's action is very weak and irregular, and they should be given with great caution, for fear of stimulating the heart too much and promoting embolism.

§ 39*a*. **Ulcerative** or **Malignant Endocarditis** [Synonyms—Infective Endocarditis, Multiple Systemic Embolism, Arterial Pyæmia (Wilks)]. In this form the endocardium is much more seriously affected, for there is more destruction of the valves and adjoining surfaces, so that large ulcers may be produced, and the valves may be perforated, or even completely disappear. The vegetations, too, are much larger.

It seems doubtful if the disease ever occurs as a primary affection, but it is rather a septicæmia or blood infection, in which the heart valves form a nidus for the circulating organisms. The micro-organisms most commonly found are staphylococci and streptococci, pneumococci, and, more rarely, gonococci, bacilli coli communes, typhoid and influenza bacilli. It is, therefore, usually a complication of such diseases as pneumonia, erysipelas, septic wounds, abscesses, meningitis,

gonorrhœa, dysentery, or puerperal fever. It is met with more rarely after chorea, scarlatina, and rheumatism, diseases in which simple endocarditis is so common; and with extreme rarity after tuberculosis, diphtheria, and variola. There is a marked predisposition for the disease to attack a heart which is already the seat of chronic endocarditis. (1) The *Constitutional Symptoms* vary considerably, but are usually grave, comprising intense anæmia, great prostration, and, in the (*a*) *Typhoid variety*, the early supervention of somnolence and muttering delirium. In the (*b*) *Septic variety* (such as arises with acute necrosis, the puerperium, or an external wound) the mind remains quite clear to the end, but rigors

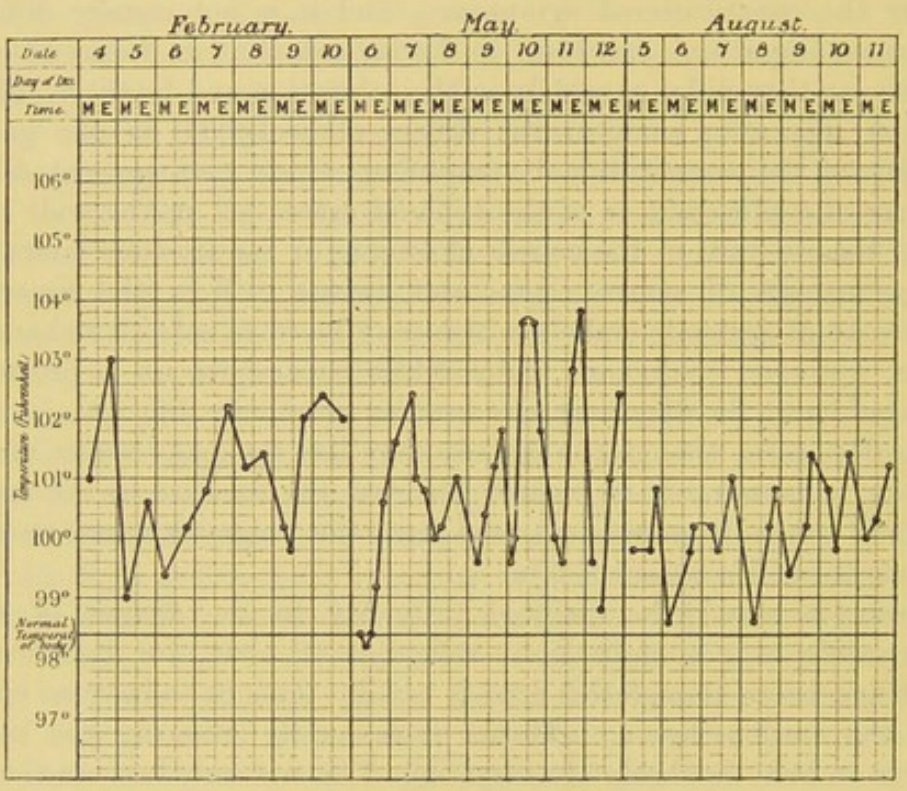

Fig. 14.—**Chart of Malignant or Ulcerative Endocarditis,** taken from a case seen with Dr. Owen Pritchard, in which exhaustive inquiry *failed to reveal any primary disease.* The patient was a single lady, aged thirty-nine, whose *previous* history pointed to chronic valve disease since the age of twenty-nine. Otherwise she had always enjoyed good health up to November, 1898, when the present illness came on gradually. At first it was mistaken for influenza, and afterwards, when the temperature did not subside, for typhoid. Extreme lassitude, great breathlessness on movement, palpitation, and pallor were the leading symptoms, with profuse and frequent perspirations at night, and at other times occasional rigors. These symptoms, with the remitting temperature, specimens of which are shown in chart, continued throughout the illness, which lasted until August, 1899, when she died. In February, 1899, after three weeks' severe headache, her doctor told her she had optic neuritis in the right eye, and about the same time the right arm became partially paralysed and completely pulseless. Later on the left arm was similarly affected. Both recovered. In June, 1899, she lost the power of speech for several weeks, and her doctor told her she had had a "stroke." In July, 1899, I saw her with Dr. Pritchard, when she had a double murmur all over the cardiac area, albuminuria, and the other symptoms above noted. No autopsy was obtained.

and sweats are prominent, simulating ague or pyæmia. The spleen is usually enlarged, and petechial rashes are fairly frequent. The fever is high, and may be continuous, but it more often runs an irregularly intermittent course, which may extend over weeks or months. (2) Generally there is a *Cardiac Murmur*, but a careful examination may be necessary, and occasionally—in cases free from old valvular mischief—there is none; so that the occurrence of embolism may be the first symptom to draw attention to the heart. (3) The *Emboli* may be simple blockings of an artery, as in simple endocarditis, but they may become abscesses, which in turn form sources of septic infection in the lung and elsewhere (Symptoms of Embolism, see p. 58).

Course and Varieties.—The severity and duration of the disease vary widely. Those cases coming on without previous cardiac mischief usually run a rapid and acute course of five or six weeks. Varieties (*a*) and (*b*) *vide supra*. (*c*) The *Cardiac group* (Bramwell)—those in which previous chronic valve disease exists—run a prolonged course of many months, up to a year or more ; rigors are often absent, and it may be very difficult from the symptoms to decide if malignant endocarditis is present or not. Between these extremes every grade is met with, but in the end the disease is almost always fatal. (*d*) There are certain aberrant forms marked by the predominance of such symptoms as jaundice, diarrhœa, parotitis, profuse sweatings, various eruptions, or pyrexia of a continued type.

The *Diagnosis* from enteric, ague, acute miliary tuberculosis, and pyæmia may be very difficult. An intermitting or remittent temperature, rigors, petechiæ, pallor of the face, and the varying character of a cardiac murmur, are points in favour of ulcerative endocarditis. In *enteric* the onset of the fever is gradual ; rigors and sweats rare or absent. The inefficacy of quinine serves to differentiate it from *ague*. Local lung symptoms aid the diagnosis of *acute miliary tuberculosis*. In *pyæmia* the cause is probably apparent, and the rigors and sweatings are more frequent ; but the resemblance is sometimes so close that Wilks proposed to call the disease " Arterial Pyæmia " (see also Chapter XV.).

Treatment must be conducted on the same general lines as that of the benign variety (*q.v.*), the precautions as to rest and stimulants applying, if possible, with greater force. On theoretical grounds it would be well to administer abundance of nutriment, and to try antiseptic remedies, such as quinine in full doses, antipyrin, antifebrin, sulphocarbolates, guaiacol, and the like. The recognition of the fact that malignant endocarditis is part of a septicæmia has led to more rational methods of treatment. Cases of recovery by the use of antistreptococcic serum have been reported,[1] but this treatment has been generally disappointing, because of the large number of different organisms which may cause the disease. The method introduced by Sir Almroth Wright promises greater success, and some encouraging results have been attained by its use.[2] A culture of the infecting micro-organism is obtained from the patient's blood, and from it a vaccine is prepared. This is injected subcutaneously, the frequency and dosage being controlled by estimating the opsonic index (*vide* Chapter XXIV.).

There are two other, *rarer*, heart disorders which arise very suddenly, and in these also the area of præcordial **dulness is not necessarily increased**—(III.) Paroxysmal Tachycardia, and (IV.) Angina Pectoris. These are paroxysmal disorders WITHOUT ELEVATION OF TEMPERATURE.

§ **40.** *The patient is suddenly seized with an attack of " palpitation," but* WITHOUT ANY DEFINITE PAIN IN THE CHEST—*the disease is probably* PAROXYSMAL TACHYCARDIA (*Neuro-palpitation*).

(III.) **Paroxysmal Tachycardia** (idiopathic tachycardia, heart hurry, neuro-palpitation, hysterical or nervous palpitation) is a term somewhat loosely applied to signify a quick pulse, but, unlike bradycardia (slow-pulse), which has no meaning in itself, the term " tachycardia " should only be employed to denote a special disease with characteristic symptoms. Tachycardia proper, or paroxysmal tachycardia, is now a recognised, though not very common, condition. It consists of a series of paroxysms, coming on at intervals, with abrupt onset, lasting for a few minutes to several hours, during which the rapidity of the pulse may amount to 200. It returns to a normal rate just as abruptly as it started. Sometimes there is no subjective distress, but it may be accompanied by a sense of constriction and suffocation, and the attack may commence with a " sinking " in the

[1] *Lancet*, August 20, 1898, p. 515. [2] *Ibid.*, November 2, 1907.

epigastrium. The face wears an anxious, terrified look, and the patient may complain of " flushing " of the general surface, with a feeling of heat or " pins and needles " all over. This is usually followed, in all the cases which I have seen, by pallor of the skin, a feeling of coldness of the extremities, with tremor, or actual shivering. Such are the symptoms of a typical attack of Paroxysmal Tachycardia, though the details may vary in different patients. The attacks return at varying intervals (days, weeks, or months), and are often determined by some emotional cause.[1]

The *Diagnosis* from Angina Pectoris is given under that affection (§ 41).

Causation.—The disease is not limited to any special period of life in women; it may occur at any time from childhood to late middle age. In men it occurs usually from fifteen to twenty-five years. No cause has been discovered, either in the heart or other organs. At present it is supposed that the disease is due to a disordered nervous condition, either in the vagus or in the sympathetic, probably the former.[2] An attack may be brought on by mental or physical strain in those who are subject to the disease. It may also be associated with hysteria; the case I have narrated in the footnote below was associated with many of the symptoms of that malady, and I have known many other cases of tachycardia similarly associated. Compare also 5, p. 64, and footnote 1 on that page. All the facts point to the disorder being an angioneurosis in which dilatation of the peripheral vessels suddenly occurs under any abnormal strain.

The *Prognosis* depends upon the frequency and duration of the attacks. The condition, it would seem, can only be temporarily cured, though patients may live for years. Dr. H. C. Wood cites a case of a physician who suffered from it for forty-three years. In the emotional variety recurrence of the attacks may be prevented by avoiding the cause.

Treatment.—For an *attack* a full dose of digitalis in brandy and water is recommended by some; others recommend that the patient should forcibly close the glottis and make a strong expiratory effort. Some patients obtain relief by strong coffee or by iced water. Ammoniated tincture of valerian is very efficacious. Tincture of sumbul, spirit of ether and of chloroform, sal volatile, and bromide of ammonium, are also useful. A seizure of this kind may frequently be cut short or averted if the patient can be induced to take some form of muscular exercise.

In the *intervals*, regular occupation, the avoidance of the various causes which are known to produce an attack, and attention to the general health, are advisable. Sodium salicylate and bromides relieved a case of Sir Clifford Allbutt's, with gouty family history. Probably bromide in some form is the most useful medicine.

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[1] The following may be quoted by way of illustration. In May, 1887, I was hastily summoned to one of the nurses in the Infirmary, who had, two hours before, witnessed for the first time in her life the death of a patient. She was a healthy young woman of twenty-five, in whom there had been previously no manifestations of hysteria. The solemnity of the scene in which she had just played her part was well calculated to have a very powerful emotional effect upon a novice, and she thereupon burst into a flood of tears. From this she recovered sufficiently to play the organ for prayers, but in the middle of the service she was seized with violent palpitation of the heart, accompanied by a pain over the apex, a sensation of " pins and needles " down the arms and legs, and a sense of impending suffocation. I found her in a state of collapse and general tremor, and unable to remember what had happened. The pulse was beating 120 per minute, respiration sighing, and the surface of the body and limbs pale, cold, and covered with a profuse cold perspiration, having previously been, I was told, suffused with marked general redness. There were no physical signs of cardiac or other visceral disease. I administered 30 grains of bromide of potash, 15 grains of chloral, and 3 ♏ liq. strychninæ. She gradually rallied, and presently sank to sleep, and the next morning she was herself again.

[2] The clinical phenomena are best explained by the hypothesis that there is a sudden lowering of the general blood-pressure, due to an equally sudden and widespread paralysis of the vaso-motor nerves of the peripheral arteries.

IV. *The patient, probably a male, at or past middle life, is suddenly seized with a severe "constricting"* PAIN IN THE CHEST, *accompanied by a sense of suffocation—the disease is* ANGINA PECTORIS.

§ **41. Angina Pectoris** is a paroxysmal affection in which the attacks consist of severe cramp-like pain in the region of the heart, attended by a sense of suffocation and impending death. The classical and severe type of this affection is, happily, very rare, but milder attacks, known as "pseudo-angina," are not uncommon.

Symptoms.—(1) An attack comes on quite suddenly, often after some exertion (at any rate, on the first occasion), and consists of acute pain in the heart, which radiates down the arms, especially the left arm. The face is expressive of the torture which the patient suffers, and at first is of a deadly pallor.[1] The limbs also are pale, benumbed, and often covered by a clammy perspiration. The patient is restless in his endeavours to assume a position of comfort. The sense of suffocation, of bodily discomfort, and of impending dissolution is extreme. The attack lasts from a few minutes to one or two hours, or more, and is liable to be aggravated if the patient ventures to move from the position of ease which he may have assumed. In a certain proportion of the cases death closes the scene. (2) The heart's action, when examined, is sometimes found to be unaltered, though palpitation may be complained of. In those cases which I have observed during the attack, the pulse was notably slow and feeble; and this is generally a marked feature in cases about to be fatal. It may be irregular, and in some cases it is increased in rapidity. There may be no murmur or physical signs of any kind referable to the heart, but more usually some form of aortic valvular mischief is present (see Etiology, p. 64). (3) The mind remains clear throughout, so that the patient appreciates fully the horror of his position. Many cases are accompanied or succeeded by a profuse flow of urine; others by profuse perspiration. Among the less frequent symptoms are tonic muscular spasms, convulsions, and vomiting. The limbs and other parts which were the seat of pain may afterwards feel "numbed." (4) In by far the larger number of cases the patients are of the male sex, and advanced in life. Out of 88 cases collected by Sir John Forbes,[2] 80 were men, and 72 of these were over fifty years of age. The disease also appears to affect by preference persons among the wealthier classes of society, and, for some inexplicable reason, as Fagge and Pye-Smith point out, persons who have been possessed of unusual mental capacity.

Varieties.—1. When discoverable cardiac lesions are present, the disease is known as *Symptomatic Angina Pectoris.*

2. *Idiopathic Angina Pectoris* is that form in which no such organic cause can be detected.

3. *Pseudo-Angina Pectoris* is a term loosely employed to designate any attack of cardiac pain and palpitation—*e.g.*, the anginoid attacks accompanying flatulent distension of the stomach. Walshe and others describe under this term a minor form of attack of frequent occurrence, consisting of more or less severe pain, referred to the region of the heart, with palpitation, "coming on either without cause or after exertion, or through overeating, or indigestion, or flatulent distension of the stomach, or a variety of other functional disturbances."[3]

4. *Hysterical Angina Pectoris* is a term sometimes employed erroneously for the condition described under Neuro-palpitation or Paroxysmal Tachycardia (§ 40).

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[1] This pallor of the surface is generally succeeded by a reddish, or sometimes cyanotic, tint of the same parts, as I have several times observed in patients at the Infirmary during the attacks. [Trousseau (Clin. Lect. New Syd. Soc., vol. iii., pp. 592, 606) and Anstie (Trans. Clin. Soc., vol. iii.) have also noted this stage.] The succeeding stage of cyanosis is due to the paralytic dilatation which sometimes follows the spasm of the arterioles.

[2] Quoted by Hilton Fagge, *loc. cit.*

[3] Walshe, "Diseases of the Heart," fourth edition, p. 209.

5. *Angina Vaso-motoria* is a term applied by Nothnagel to cases somewhat resembling true angina pectoris, excepting that the symptoms of vaso-motor disturbance—pallor followed by cyanosis, coldness, and numbness of the extremities—predominate over the symptoms referable to the chest.[1]

Diagnosis.—(1) It is important to distinguish the different forms of "anginoid attack" grouped under "*Pseudo-Angina Pectoris*" from true angina. Among the features which sometimes enable us to distinguish pseudo-angina from the graver form of disease are the following: (i.) They come on at any time of life, whereas true angina is confined to persons of the male sex over forty-five years of age; (ii.) they may occur in either sex, the hysterical form being specially liable to affect young women; (iii.) they may come on spontaneously, without previous exertion (though this is not constant); (iv.) they often appear after meals, and are nearly always associated with some gastric derangement, such as dilated stomach, flatulence; (v.) the pulse of pseudo-angina is usually rapid and regular, never slow; and the sounds and boundaries of the heart are normal. Nevertheless, pseudo-angina, like true angina, may or may not be associated with cardiac lesions. (2) Attacks of *Paroxysmal Tachycardia* are not difficult to differentiate from true angina, on account of the great rapidity and the regularity of the pulse, and the normal arterial tension. (3) *Biliary Colic* has occasionally to be diagnosed from angina, but here the patient advanced in years is usually of the female sex, and the condition is speedily followed by jaundice. (4) The diagnosis from the other causes of præcordial pain has already been given (§ 23).

Etiology.—The immediate cause of an attack is usually some undue exertion. After death it is said that no structural disease of the heart and arteries may be found, though far more frequently the heart walls are found to be degenerated, flabby, or fatty, with or without other changes in the cardio-vascular system.

The clinical and anatomical antecedents of angina are the following: (1) Fatty, or fibroid, or granular degeneration of the heart muscle is said to be the most frequent. (2) Aortic valvular disease, especially stenosis; mitral disease is rare. (3) Advanced atheroma or calcification of the aorta. (4) Aneurysm or dilatation of the aorta, especially of the root within the pericardial sac. (5) Atheroma of the coronary arteries, calcification, or some other disease of these structures; and this may in some cases lead to embolism or thrombosis, and thus to a more or less localised degeneration of the cardiac muscle (Kernig). (6) Arterial sclerosis (using that term in its widest sense to indicate any thickening and rigidity of the arterial walls). (7) Gumma of the heart wall, in which circumstances the patient may be young.[2]

Angina Pectoris has been regarded as the result of disease of the coronary arteries, but it is now generally believed to be due to a *sudden* demand for increased effort on the part of a *damaged heart.* In, at any rate, a certain proportion of cases this sudden demand consists of an abrupt increase in the peripheral resistance by *contraction of the peripheral arterioles.* It appears that for the production of the attacks of angina the combination of these two factors is necessary. Neither of these can alone produce a paroxysm; for, as Broadbent ("Heart Disease")

[1] "Angina Pectoris Vaso-motoria," *Deutsch. Arch. Klin. Med.*, iii. As Fagge and Pye-Smith point out ("Prin. and Prac. Med.," second edition, vol. i., p. 912), in these cases of Nothnagel "the state of the peripheral circulation seems to be very similar to that which in other patients leads to paroxysmal hæmoglobinuria," and this angina is brought into line with other neuro-vascular disorders.

[2] Dr. Sidney Phillips (*Lancet*, 1897, vol. i., p. 223) has collected a valuable series of cases, and from these it would appear that, if small and situated elsewhere than in the left ventricle, a gumma of the heart may give rise to no symptoms, until by its growth it produces sudden death. It is not, therefore, possible to diagnose **cardiac syphilis** with certainty. Its existence, however, may be suspected in presence of angina pectoris and a rapid, irregular pulse, especially if these occur in a person under middle age, and if no *other cause for these symptoms* can be made out. If these be present, even in a slight degree, in syphilitic persons, they should be regarded with grave apprehension; and the disappearance of these symptoms under antisyphilitic treatment renders the diagnosis highly probable.

points out, high arterial tension is extremely common, alone ; so also is a degenerated heart wall—yet angina is rare. When, however, the two are present in combination, a third or determining cause (*e.g.*, some unusual exertion, or a further increase in the blood-pressure), supervening *suddenly*, may produce an attack of angina.

Prognosis.—True angina is an extremely serious condition. The patient may die in a paroxysm. The attacks are sure to return, though this may not happen for some years. The existence of a cardio-valvular lesion, which is usual, does not materially modify the prognosis ; the condition of the cardiac wall is our best guide to the probable course of a case (Prognosis of Cardiac Valvular Disease, § 50).

Treatment.—(*a*) *For the Attacks.*—Amyl nitrite, 3 to 5 minims, inhaled, generally gives prompt relief, a method of treatment for which we are indebted to Sir Lauder Brunton. Sufferers should carry about with them glass capsules containing this quantity, which can be broken into the handkerchief.[1] The remedy hastens the advent of the second stage of arterial dilatation, and the attack passes off. For a more lasting effect, nitroglycerine may be given internally, $\frac{1}{100}$ drop every one to four hours, in tabloids, pushed by tolerance to $\frac{1}{10}$ drop. All the nitrites have a similar action in dilating the peripheral arteries ; and lately advantages have been claimed for erythrol-tetra-nitrate,[2] in that its effects are more permanent, 1 grain administered in 1 drachm of absolute alcohol, suitably diluted, being said to have effects lasting four or five hours. It is reported to have relieved cases in which other remedies have failed. A hypodermic injection of a full dose of morphia, if the last-named remedies are not at hand, generally gives some relief ; and in very severe cases chloroform, inhaled to complete anæsthesia, has been recommended. If this be combined with a dose of morphia, its effects become more prolonged. In some cases of "Angina Vaso-motoria" warm baths give great relief. I have not tried this treatment in cases of true angina, but, judging from the good effects I have observed in other conditions of vascular spasm, this method of treatment would be very efficacious in cases where movement is not harmful to the patient. Mustard plasters and warm fomentations to the epigastrium may be tried, either during or between the attacks. In two cases of somewhat severe pseudo-angina I found that the following draught, carried by the sufferer in his pocket, and taken at the outset of an attack, was attended by prompt relief : Tinct. lobeliæ æth., ℥xx. ; spiritus etheris, ℥xx. ; liquoris morphinæ, ℥xx. ; aquæ chloroformi, ℥i.

(*b*) *Between the Attacks.*—It follows from the above remarks on the etiology that the indications for treatment lie in two directions—to relieve excessive tension or any tendency to vascular spasm, and, if possible, to restore the damaged heart. If the main element of the case is cardiac enfeeblement, this should receive our special attention, on the lines mentioned elsewhere (Treatment of Cardiac Valvular Disease). If, on the other hand, the peripheral resistance is excessive, our treatment should be directed to reduce it. The pulse should be examined many times, and under different conditions, during the day, and if the tension is very high, much may be done, even though the arteries be diseased (§ 61, High Tension). Erythrol-tetra-nitrate and nitroglycerine are here again valuable remedies, not only to relieve, but to prevent the occurrence of the attacks, and these may be combined with various cardiac tonics, such as iron, nux vomica, and especially arsenic. Digitalis and stimulants may be administered on the same principles as in cardiac valvular disease.

Much may be done by regulating the mode of life, and avoiding those things which are known by experience to induce the seizures. When, as usual, attacks are brought on by exertion, complete repose of mind and body must be strictly enforced. Other determining causes met with are exposure to cold, indigestion, dilatation of the stomach by too heavy meals, and a sudden alteration of posture by the patient. Such conditions must be avoided, as also any unnecessary or sudden exertion or emotion.

[1] This remedy seems to lose its effect when preserved in the ordinary way in a bottle.

[2] Bradbury, *Brit. Med. Journ.*, 1895, vol. ii., p. 1213 ; and Geo. Oliver, *Brit. Med. Journ.*, 1896, vol. i., p. 1433.

CHRONIC AFFECTIONS OF THE HEART AND PERICARDIUM.

§ **42. Classification.**—CHRONIC Disorders of the heart and pericardium may follow an acute attack of the conditions described in the previous sections, as, for instance, when chronic valvular disease dates from an acute endocarditis which has complicated rheumatic fever or scarlatina in early life. But a considerable proportion of the disorders which affect the heart are chronic from the beginning ; they start insidiously, and are unaccompanied by any marked constitutional symptoms.

For clinical purposes, the chronic disorders of the heart and pericardium may be divided into those attended by enlargement of the area of dulness, and those *not necessarily* so attended.

(*a*) CHRONIC Diseases attended by ENLARGEMENT of the area of præcordial dulness.

I. Cardiac hypertrophy.
II. Cardiac dilatation.
III. Hydropericardium.
IV. Congenital heart disease (rare). *Mem.* Aneurysm, and other Mediastinal Tumours (see footnote to table on p. 5).

(*b*) CHRONIC Diseases NOT NECESSARILY attended by ENLARGEMENT of the area of præcordial dulness, the diagnosis of which depends mainly on auscultation.

I. Valvular disease.
II. Fatty heart.

It is important to bear in mind that valvular disease, though not *per se* giving rise to an enlarged area of præcordial dulness, is so often associated with hypertrophy or dilatation that it is usually attended by enlargement of the præcordial dulness.

Method of Procedure.—It will be remembered that the routine examination of the heart consisted of (1) inspection ; (2) palpation ; (3) percussion of the præcordial dulness ; and (4) auscultation. The student should bear in mind the various *fallacies* which may give a false impression of cardiac enlargement, and also those conditions, such as emphysema, which obscure an enlarged heart (§ 33). If the area of dulness is NOT INCREASED, turn to § 47.

GROUP A.—The patient complains of some of the subjective symptoms pointing to **chronic cardiac disorder,** and, on examination of the heart, the **area of dulness is found to be increased,** chiefly in the transverse direction—the disease is probably **Hypertrophy, Dilatation,** or **Hydropericardium.**

I. *The* APEX BEATS BELOW *its normal position ; the impulse is* FORCIBLE *and heaving ; on auscultation, the first sound is* DULL *and prolonged. There is* HYPERTROPHY OF THE HEART.

§ **43. Hypertrophy of the Heart,** and the dilatation which so frequently accompanies or follows it are certainly the commonest conditions which produce an increased area of præcordial dulness.

Cardiac Hypertrophy is an increase of the muscular substance of the heart, and its weight, which is normally about 8½ ounces in women and 9½ ounces in men, may be increased to 10 or 12 ounces, and on rare occasions to 15 or 20 ounces. Its *signs* are as follows : (1) The increase in the præcordial dulness is in a transverse direction—towards the left if the left ventricle be hypertrophied, towards the right if the right ventricle ; (2) the apex beats below its normal position ; (3) the impulse is unduly forcible, heaving, or thrusting ; (4) on auscultation, the first sound is muffled, less audible, and prolonged. The pulse is firm, strong, and bounding.

Symptoms may be altogether wanting if the hypertrophy accurately compensates for the obstruction in the circulation which has caused the hypertrophy. The patient may, indeed, be unaware of any cardiac disorder. But generally, on inquiry, he will complain of a " thumping " in his chest and " throbbing " in his head, occasionally of breathlessness and præcordial distress.

Etiology.—Cardiac hypertrophy—and the dilatation which often accompanies it—is always the result of some obstruction in the circulation, either in the lungs (such as bronchitis and emphysema) or in the general circulation (such as cardiac valvular disease, or arterial thickening). It is an illustration of the physiological law that increased use leads to increased growth. The part of the heart which chiefly undergoes hypertrophy will depend on the position of the obstruction ; and the signs met with in addition to those above mentioned will vary accordingly. Thus, there will be three sets of symptoms : (*a*) Signs of hypertrophy of the heart ; (*b*) signs of enlargement of the cavity specially involved ; and (*c*) signs and symptoms of the cause. The following causes will be more readily understood by consulting Fig. 15 (p. 76), and it must be remembered that the enlargement is never in actual practice strictly limited to one chamber of the heart.

(*a*) Hypertrophy of the Left Ventricle is indicated by enlargement of the area of dulness, chiefly towards the left ; the apex beats *below* and to the left of its normal position ; the pulse is strong unless modified by the presence of a valvular lesion, and the carotids may be seen to pulsate. This condition may arise under ten different causes :

(i.) *Mitral regurgitation*, in which case there would be a systolic apical murmur, and the other features given in § 47.

(ii.) *Aortic stenosis* or *regurgitation*, which may be recognised by a basal murmur of systolic or diastolic rhythm, and other characters given in § 47. The hypertrophy resulting from regurgitation may be greater than that due to any other cause (*cor bovinum* of the ancients). The heart may weigh in these cases 20 to 30 ounces, or more. In regurgitant lesions a certain amount of dilatation always accompanies hypertrophy, and the condition is then known as " eccentric " hypertrophy. In these cases the dilatation is compensatory, and produces no

untoward symptoms. True, or, as it is called, "concentric," hypertrophy, unaccompanied by any dilatation, is only met with in stenosis, especially aortic stenosis.

(iii.) *Aneurysm of the aortic arch*, if unattended by valvular disease or renal mischief, does not *per se* cause cardiac hypertrophy; but it is nearly always so attended, and thus becomes a fairly frequent cause of hypertrophy of the left ventricle. If the aneurysm involves the first half of the arch, it produces marked physical signs; if the second or third part, *pressure* symptoms arise without signs (§ 53).

(iv.) Prolonged *high arterial tension*—and thus its numerous causes (§ 61)—may lead to hypertrophy of the left ventricle. It is probably in this way that chronic Bright's disease is so frequently accompanied by hypertrophy of the left ventricle.

(v.) Widespread *thickening of the peripheral arterioles* is invariably followed sooner or later by a certain degree of hypertrophy of the left ventricle (arterial sclerosis, § 67).

(vi.) *Disturbed innervation*, such as attends the "irritable heart," Graves' disease, and neuro-palpitation, in time results in moderate hypertrophy, chiefly of the left ventricle.

(vii.) *Pericardial adhesions*, unless they are few or filamentous, or unless there is universal internal adhesion, so that the heart cannot expand.

(viii.) *Excessive muscular exercise*, whether athletic or laborious, may produce hypertrophy, and in support of this statement it may be mentioned that the normal increase with age is more noticeable in men than in women.

(ix.) *Cardiac fibrosis* (sclerotic myocarditis) is a rare condition, which may be associated with a certain amount of hypertrophy.

(x.) *Increased density of the blood* is a cause of hypertrophy which is not mentioned among the lists usually given in textbooks, but which, nevertheless, must be of considerable potency. It is estimated that 99 per cent. of the work done by the heart is employed in overcoming the resistance due to capillarity in the arteries and capillaries, and it follows, almost of necessity, that if the density of their fluid contents be increased, the resistance will be increased proportionately.

(*b*) Hypertrophy of the Right Ventricle is indicated by enlargement of the area of dulness to the right; throbbing and pulsation in the lower left intercostal spaces and epigastrium (and if accompanied by dilatation, pulsation also in the veins of the neck); a violent but more *diffuse* apex-beat; and, on auscultation, accentuation of the second sound over the pulmonary valves. *The degree of hypertrophy present may be measured by the degree of the second and fourth of these symptoms:* in this way we measure the efficiency of compensation (§ 50).

It may be due to three important causes:

(i.) *Pulmonary diseases* attended by obstruction in the pulmonary circulation, of which *bronchitis and emphysema* are certainly the most frequent. This combination, a very common one, is identified by a history or evidence of lung mischief (§ 93).

(ii.) *Mitral stenosis* is the next most common cause, and should be borne in mind even in the absence of a presystolic murmur (§ 47).

(iii.) *Mitral regurgitation* is followed by hypertrophy of the right ventricle, due to the congestion of the pulmonary circulation.

(*c*) Hypertrophy of the Left Auricle is always attended by dilatation. It is a difficult condition to detect, because the palpable and visible pulsation in the third left interspace, when present, though due to this cause, may admit of other explanations.

It may arise in *mitral regurgitation*, but its chief cause is *mitral stenosis*. In the latter condition, palpation generally reveals a thrill over the apex, and careful auscultation may detect the presystolic or mid-diastolic murmur (§ 47).

Hypertrophy of the Right Auricle is not recognisable clinically.

(*d*) Extreme HYPERTROPHY OF BOTH AURICLES AND VENTRICLES arises in congenital heart disease, but may be confined to the right side, since the valvular lesions are mostly on that side.

Obscure Causes.—If in a given case of cardiac hypertrophy careful examination reveals no valvular mischief, and no obvious cause can be made out, the physician should always suspect OBSCURE AORTIC ANEURYSM, OBSCURE RENAL DISEASE, or widespread ARTERIAL THICKENING.

Prognosis and Treatment.—Cardiac Hypertrophy is in itself essentially a conservative process for some condition which causes obstruction in the circulation. It is Nature's method of compensating for the obstruction, and it is well to promote it up to a certain point.

1. *If the cause be removable*, the prognosis is favourable. Our treatment in such cases should therefore be directed to the removal of the cause—*e.g.*, high arterial tension, which can be reduced.

2. *If the cause be not removable*, the prognosis of the case depends on our being able first to maintain the compensatory hypertrophy, and, secondly, to relieve the heart of part of its work, so that the hypertrophy does not go beyond what is necessary. To accomplish the first, we should endeavour to promote the general nutrition by tonics and hygienic measures, and by regulating the emunctories with mild saline laxatives, and similar remedies. In order to relieve the heart of part of its work, and to aid the systemic circulation, baths, massage, passive and active movements, are of the greatest use (see § 51).

3. The *existence of cardiac hypertrophy* adds an element of risk to a person's life in three ways. In the first place, hypertrophy infallibly indicates that there is obstruction somewhere in the circulation, and this, whatever it be, is in itself an injury to health, and may shorten life. Secondly, a far more important consideration is the increased liability to cerebral hæmorrhage, and vascular rupture elsewhere, which cardiac hypertrophy entails. Thirdly, high arterial tension is an almost invariable accompaniment of cardiac hypertrophy, and this causes a continual strain upon the peripheral vessels, which results first in arterial hypermyotrophy and later in arterial sclerosis, the serious consequences of which are indicated elsewhere (§ 67).

II. *The area is increased; the position of the* APEX IS INDEFINITE; *the impulse is diffuse and wavy; on auscultation, the first sound is short and sharp. The disease is* CARDIAC DILATATION.

§ **44. Cardiac Dilatation** (one form of "Cardiac failure") is an indication that the heart is giving way, or "failing" to keep pace with the extra demand made upon it by reason of some obstruction in the circula-

tion. In ordinary circumstances the heart first hypertrophies, then dilates, and that is why hypertrophy and dilatation are usually associated. But if the nutrition of the body or of the heart is faulty, the heart begins to dilate from the outset, without any preliminary hypertrophy.

The *Physical Signs* of cardiac dilatation resemble those of hypertrophy in several ways, and, like it, (1) the area of dulness is increased chiefly in a transverse direction, to the right or to the left, according to the side of the heart which is dilated. But there are three important features specially belonging to dilatation : (2) The apex-beat is wavy and diffuse, and is displaced outwards rather than downwards ; it may be so feeble as to be hardly perceptible. (3) On auscultation, the first sound at the apex is clear and sharp, resembling the normal second sound in character. Murmurs may be present from co-existing valvular disease, but *a systolic murmur*—the "murmur of dilatation"—may sometimes be heard apart from actual valvular disease, because the auriculo-ventricular orifices, by reason of the dilatation, allow a reflux of the blood. (4) The pulse is feeble, rapid, irregular, and sometimes intermittent (see also Table III.).

TABLE III.—DIAGNOSIS OF TYPICAL CARDIAC HYPERTROPHY FROM TYPICAL DILATATION.

Apex-Beat and Impulse : Displaced in Both.	Percussion.	Auscultation.	General Symptoms.
Hypertrophy. Forcible, heaving, thrusting ; below and to l. of normal (l. ventricle) ; in epigastrium (r. ventricle).	L. V. : Area increased transversely to the l. R. V. : Area increased transversely to the r.	Sounds muffled, prolonged, and forcible.	May be absent ; or symptoms of high arterial tension.
Dilatation. Feeble, irregular, undulatory diffuse. If r. ventricle—pulsation in the epigastrium and veins of the neck.	L. V. : Area increased transversely to the l. R. V. : Area increased transversely to the r.	Systolic murmur at apex, at one stage. Systolic murmur in tricuspid area, at one stage. Sounds sharp and clear.	Dyspnœa, cough, cyanosis, and other signs of lung congestion. Dropsy, scanty high-coloured albuminous urine, enlarged liver, ascites, and other signs of congestion of organs.

Towards the end, when cardiac failure is extreme, fœtal rhythm, gallop rhythm, and "delirium cordis" may occur. In fœtal rhythm the long and short pauses are almost identical, so that the first and second sounds can scarcely be distinguished ; in gallop rhythm there is rapidity of action, together with a distinctly reduplicated second sound. In delirium cordis the heart is so rapid and so irregular that it is practically impossible to make out the relations of sounds and murmurs.

It is, however, by the presence of certain *symptoms* that the existence of cardiac dilatation (or failure of compensation) generally becomes manifest. In hypertrophy, as we have seen, there may be no symptoms at all ; but with dilatation the patient complains of: (1) *Heart symptoms*,

such as breathlessness on little or no exertion, palpitation, and præcordial distress. (2) There may also arise a number of *symptoms referable to other parts*, in consequence of the delay in the circulation, such as anasarca, ascites, and symptoms of congestion of the lungs, liver, and kidneys. These will be described under Cardiac Valvular Disease, where the means of detecting which cavity is chiefly involved is also given (see also Table III.).

The *Causes of Cardiac Dilatation* are of extreme importance as bearing on the prognosis and treatment of cardiac valvular disease and other circulatory disorders. The *clinical conditions* which produce dilatation are practically identical with those which produce cardiac hypertrophy (§ 43), when they are persistent and *are associated with some condition which impairs the nutrition of the heart* (see below). Undoubtedly the two commonest causes of cardiac hypertrophy and dilatation are Cardiac Valvular Disease and Chronic Bronchitis with Emphysema, and these are the possibilities which should first suggest themselves to the mind in a case where dilatation is evident. The former will be fully discussed in the following section.

Chronic Bronchitis, with its usual accompaniment of Emphysema, produces in time a dilated *Right Ventricle*. This latter is recognised by two very characteristic local signs, in addition to the breathlessness, etc., above mentioned—viz. : (i.) epigastric pulsation ; and (ii.) pulsation in the jugular veins. The clinical picture presented by this frequent pathological combination is very characteristic—the florid face and plethoric build ; the easily excited breathlessness and constantly recurring cough, enable us to recognise the condition almost at a glance. The subject will be more fully discussed under Cardiac Valvular Disease (§ 47).

The *essential or pathological causes* of Dilatation may be arranged under four headings :

(*a*) Any condition which persistently *prevents the complete emptying of the cavities of the heart* (see Causes of Hypertrophy) will produce compensatory hypertrophy with dilatation, which will be *exactly proportional* to the increased resistance in the circulation, provided none of the circumstances mentioned under (*b*), (*c*), or (*d*) below are also present. If any of these circumstances are in operation, dilatation or failure may be initiated without previous or accompanying hypertrophy. Moreover, the supervention of any of these in the course of a cardiac case may at once disturb a well-balanced compensatory hypertrophy, and serious symptoms may immediately appear.

(*b*) Any *failure of general nutrition*, or vitality, may entail a weakened cardiac wall, which will perhaps yield even under normal circulatory conditions. Such, for instance, may be caused by exposure, insufficient food, alcoholic excesses, old age, various fevers (especially rheumatic fever, typhus, typhoid, and malaria), various blood conditions (such as pernicious anæmia, scurvy, chlorosis, leukæmia, etc.), and cachectic conditions (such as syphilis, tubercle, and cancer). See Causes of Pyrexia and of Anæmia (Chapters XV. and XVI.).

(*c*) *Local impairment of the nutrition of the heart wall* may result in dilatation without hypertrophy, even with normal circulatory resistance. Myocarditis, for instance, and the conditions which accompany peri- and endo-carditis (which lead sometimes to acute dilatation) ; or the more gradual degenerations which

ensue on sclerosis and other diseases of the coronary arteries; or fibroid and other degenerations of the cardiac wall (see Fatty Heart). Prolonged fatigue may also act locally by overtaxing the heart muscle. Any of these may upset the balance of a well-adjusted hypertrophy.

(*d*) Any *sudden strain on an apparently normal heart* may produce acute dilatation. Thus, severe and sudden grief, fright, or anxiety may damage the heart through its nervous apparatus, and severe muscular exertion in athletes or others who have not had any previous training may cause the heart to give way and dilate. Instances of the latter are met with in hill-climbers who are "out of form," and others who take sudden and unaccustomed exercise. Breathlessness may date from incidents of this kind, from which the patient may never, or only with difficulty, recover.

The *Prognosis* and *Treatment* of Cardiac Dilatation are fully dealt with under Cardiac Valvular Disease (§ 47).

III. *The area of dulness is* INCREASED UPWARDS, *and its shape is pyramidal, with the point upwards; the apex-beat is raised, and the impulse is weak and undulatory; on auscultation, the sounds are feeble. The disease is* HYDROPERICARDIUM.

§ **45. Hydropericardium** is a chronic effusion of fluid into the pericardium. (1) The shape of the dulness is very characteristic, being pyramidal, with the narrow end upwards. (2) The apex of the heart is *raised*, and *to the right* of its normal position, because the roof of the pericardium is raised by the fluid, and takes the heart with it. (3) For the same reason, the left margin of præcordial dulness extends *beyond* the apex-beat. (4) On auscultation, the heart sounds are distant and muffled. There may be irregularity and rapidity of the pulse, and difficulty of breathing from the impeded action of the heart and lungs.

Etiology.—Chronic effusion into the pericardium may originate in one of three ways. (1) As the result of Acute Pericarditis (§ 38), of which a history is generally obtainable, but by no means always (see Latent Pericarditis, § 38*b*). Most authors draw a distinction between chronic pericardial effusion of inflammatory origin and simple dropsy of the pericardium (hydropericardium proper). But the physical signs are practically indistinguishable; for the diagnosis of the former we depend mainly on the history of it having commenced as an acute affection, and on the absence of anasarca. (2) True hydropericardium seldom occurs excepting as part of a general dropsy due to renal or cardiac disease, and therefore the urine should be carefully examined. In these circumstances the effusion gives relatively *little inconvenience to the patient*, because it takes place so gradually that the parts have time to adapt themselves to the stretching they undergo. (3) If hydropericardium be not preceded by pericarditis, or be not part of a general dropsy, new growth or tubercle, although rare, should always be suspected. In these circumstances, if a little fluid be withdrawn by a hypodermic syringe (p. 56), it may be blood-stained (cancer), or contain bacilli (tubercle).

The *Diagnosis* from Cardiac Dilatation should be readily accomplished by the shape of the dulness, which is square instead of pyramidal in dilatation; and by the heart sounds, which are clear and sharp in dilatation, muffled in effusion. Pleuritic effusion is attended by pulmonary symptoms.

The *Prognosis* of hydropericardium depends on its causation, being favourable in Cause 1, adding only a little to the gravity of the primary malady in 2, and being almost necessarily fatal in 3.

Treatment.—The treatment of inflammatory effusion is dealt with in § 38. If part of a general dropsy, our efforts must be directed to this. Counter-irritants are sometimes useful. Paracentesis should not be considered unless the cardiac embarrassment is very urgent, because of the danger of withdrawing a large amount of fluid suddenly from the pericardial sac, and because the risks of the operation are not small.

IV. *The præcordial percussion area is* **considerably** *and* **irregularly increased** ; *the impulse is forcible and heaving.*

(*a*) *The area is* DISTORTED *and somewhat* SQUARE *; on auscultation, there is a loud murmur, probably loudest in the pulmonary area. The disease is* CONGENITAL HEART DISEASE (see below).

(*b*) *The* UPPER PART *of the area is increased transversely, and there is dulness* OVER THE STERNUM—*it is probably an* INTRATHORACIC TUMOUR. *If, on auscultation, the second sound at the base is reinforced and sharp, or replaced by a diastolic murmur, it is probably* ANEURYSM *of the first part of the* ARCH OF THE AORTA (§ 53).

§ **46. Congenital Heart Disease** is another chronic form of cardiac disorder attended by increased præcordial dulness, but it is comparatively rare. There are three cardinal signs produced by it: (1) The præcordial dulness is very considerably *increased*, the normal shape is *distorted*, and it may extend considerably beyond the right border of the sternum, because the commonest form of the disease results in immense hypertrophy and dilatation of the right ventricle. (2) Palpable and sometimes visible pulsation over almost the whole of the cardiac area may often be detected for the same reason. (3) A loud, rough systolic murmur can generally be heard, loudest in the third or fourth interspace, close to the left of the sternum, and it is often easily heard in the back. These signs in a child who has a tendency to cyanosis are almost certainly due to cardiac malformation. (4) Dyspnœa is also fairly common, and may be either persistent or paroxysmal. The condition, however, may remain latent for many years, until exertion or some illness reveals its existence. The diagnosis is sometimes a matter of difficulty. Other symptoms arise as the disease progresses—thus, general cyanosis, reaching a very extreme degree, greater than in any other condition ; coldness of the extremities ; a low temperature of the surface generally (because the blood is poor in oxygen), although not of the interior of the body (Peacock) ; dropsy occasionally ; hæmorrhages from the lungs ; and symptoms of congestion of the other viscera. Dilatation of the conjunctival vessels is often observed, and clubbing of the toe and finger ends. Headache is often present, and convulsions are not unknown. Backwardness or precocity, and sometimes more grave mental defects, become evident as the child grows older. It is important to remember that very loud bruits may mean but slight abnormality, whilst very severe disease may be unaccompanied by even a slight murmur.

Etiology.—Congenital disease of the heart arises under two conditions : (1) *Inflammatory affections* attacking the fœtal heart *in utero* may lead to stenosis of the orifices, almost invariably on the right side of the heart, which is in contradistinction to the left-sided lesions of extra-uterine life. (2) An *arrest of the closure*, which normally takes place shortly after birth, of either the ductus arteriosus, foramen ovale, or the ventricular septum. Whatever the cause, the commonest *lesion* is a narrowing of the pulmonary artery or pulmonary valves,[1] which probably results in the venous blood making its way through the foramen ovale or ventricular septum from the right to the left side of the heart, the septum also deviating to the left. Stenosis of the aortic orifice is much rarer, but in that case the arterial blood finds its way through the same orifices from the left to the right side of the heart. In either case three events happen : (1) Deficient oxygenation, and probably admixture of venous and arterial blood ; (2) the right ventricle takes an *equal*

[1] Some observers maintain that the primary mischief is always the non-closure of the ductus arteriosus or foramen ovale or ventricular septum, narrowing of the aorta and pulmonary arteries being secondary. But the view above stated is the more probable, because a small leakage through one of these orifices is a by no means infrequent occurrence without symptoms during life. Some observers also maintain that there is *no admixture of the venous and arterial blood*, the cyanosis being due to deficient oxygenation.

share with the left in the work of the heart, and consequently it hypertrophies and dilates; and (3) the ductus arteriosus remains patent, to compensate for the insufficient delivery of blood into the aorta or pulmonary artery, as the case may be. It is only occasionally possible to suggest the precise nature of the lesion during life, but this, although it is a matter of great interest, is not always of great moment.

Prognosis.—The condition may remain latent for many years, though few marked cases survive to adult life. The prognosis is serious in proportion to the degree of dyspnœa and cyanosis, pointing to deficient aeration of the blood, and in proportion to the other symptoms of "cardiac failure" (§ 50).

The *Treatment* is the same as that of Cardiac Dilatation, bearing in mind that rest is of primary importance (§ 51).

We now turn to those Chronic Heart Diseases in which the **area of dulness is not necessarily increased,** and which depend mainly on AUSCULTATION for their diagnosis (Table IV., p. 75).

V. *On auscultation, one or both of the heart sounds is heard to be accompanied by a* MURMUR, *or bruit. Pericardial friction having been excluded, the* VALVES *of the heart are diseased—with certain exceptions or fallacies (such as hæmic murmurs), which will be described.*

§ **47. Chronic Endocarditis — Cardiac Valvular Disease — Cardiac Murmurs.**—Disease of the valves of the heart is the commonest of all cardiac disorders, and it is revealed on auscultation by the presence of a bruit or murmur which is added to, or replaces, one or both of the heart sounds.

Method of Procedure.—In order to arrive at a diagnosis, it will be remembered that four features must be carefully investigated in any given murmur—namely, its RHYTHM, POSITION, CONDUCTION and CHARACTER (§ 34). The last named is relatively least important. In order to be quite sure of the rhythm of a bruit, it is often convenient to place the fingers on the carotid artery whilst auscultating the chest.

A cardiac murmur may arise in three ways. It may arise outside the heart—*e.g.*, from roughness of the PERICARDIUM; it may be of HÆMIC OR FUNCTIONAL ORIGIN; or it may arise within the heart from ORGANIC DISEASE OF THE VALVES (which concerns us now).

The characters of PERICARDIAL MURMURS have already been given (§ 39); and their diagnosis from endocardial murmurs (Table, p. 57).

HÆMIC, or functional, MURMURS are frequently heard in anæmia and in some other blood conditions (see Chapter XVI.). They are usually systolic in time. They are rarely double, and never diastolic alone. They are usually heard loudest in the pulmonary area. A single murmur of presystolic or diastolic time is a certain indication of organic disease at one of the cardiac orifices.

Organic Murmurs are those which are produced by organic disease of the valves (cardio-valvular disease). Valvular disease may be due to

several lesions (§ 49), but the commonest one in early life is endocarditis (acute or chronic), and in older persons chronic degenerative change. The effect is a thickening or puckering of the valves and ring, which results in one or both of two conditions: (*a*) *Stenosis*—*i.e.*, a narrowing (στενοω, to contract) of the orifice, which prevents the blood flowing freely through it; or (*b*) *Regurgitation*, in which the valves are incompetent and allow a reflux of the blood to take place from imperfect meeting and closure of the cusps. The remote effect of these two conditions is practically the same—viz., a retardation or obstruction to the circulation of blood through that orifice.

It simplifies matters very much that cardio-valvular disease arising *after* birth is practically *confined to the left side of the heart*—*i.e.*, to the mitral and aortic orifices. Thus it happens that there are four principal cardiac lesions—Mitral Regurgitation, Mitral Stenosis, Aortic Regurgitation, and Aortic Stenosis.

Table IV.—Differentiation of Cardiac Valvular Diseases.

			Auscultation.	Appearance of Patient.	Pulse.	Other Symptoms special to the Disease.	
C. V. D.	**Mitral** (apical murmurs).	*Regurgitation.*	*Systolic* murmur conducted into axilla.	Florid.	Irregular, rapid, and compressible.	Dropsy, enlarged liver and ascites, etc.,	with signs of congestion of organs.
		Stenosis.	*Presystolic* murmur.	Patient young.	Regular, small, and moderately firm.	Hæmoptysis; emboli,	
	Aortic (basal murmurs).	*Regurgitation.*	*Diastolic* murmur conducted down sternum.	Sallow.	"Water-hammer," rapid and compressible.	Throbbing of arteries of neck,	with symptoms of cerebral anæmia and anginoid attacks.
		Stenosis.[1]	*Systolic* murmur conducted into vessels of the neck.	Heart lesion of the aged.	Slow, regular, small and hard.	No special symptoms,	

The student should study Fig. 10, p. 47, so as to thoroughly comprehend the various events which occur during one complete contraction and dilatation of the heart (a cardiac cycle). He should also bear in mind that the left side of the heart is behind the right, and that the left ventricle comes nearest to the surface only at the apex, immediately behind or just below the fifth rib (Figs. 11 and 12, p. 49). He should also remember that a cardiac murmur is not produced *in* a diseased orifice, but by the eddies in the blood-stream beyond. For these reasons a murmur is not always heard loudest directly over the orifice diseased. The student may also consult the diagram of the circulation on the next page.

Diagnosis of Cardiac Murmurs.—The first thing to determine is whether a given murmur is related to the first or second sound of the heart—*i.e.*, whether its rhythm is systolic or diastolic—and this will form a convenient basis of classification of cardiac murmurs.

[1] Real aortic stenosis is very rare, but atheromatous roughening is very common.

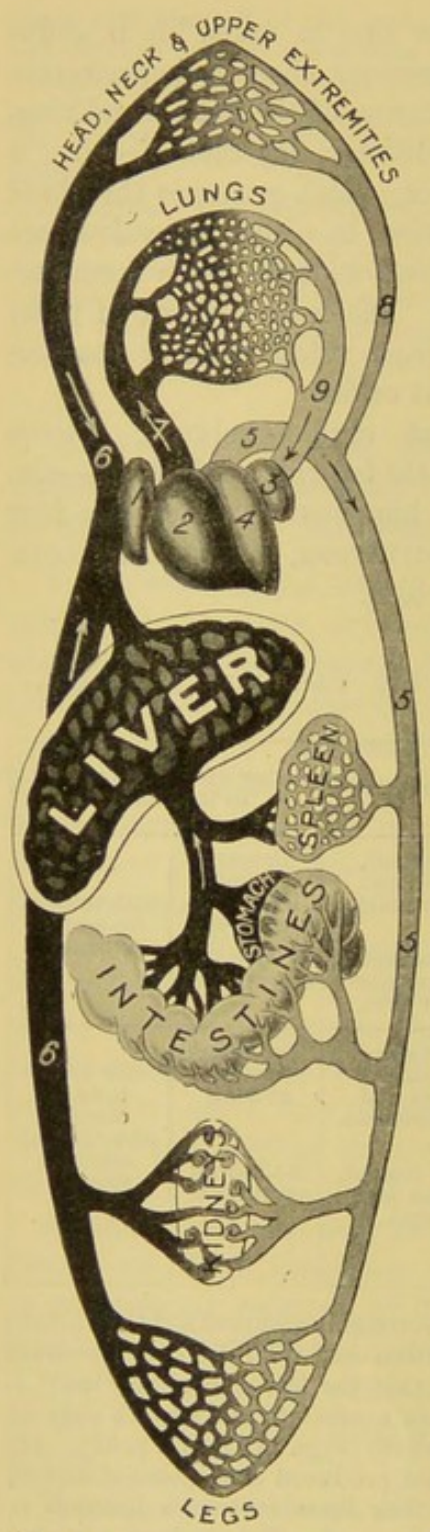

Fig. 15.—Scheme of the Circulation of the Blood.—The superior and inferior venæ cavæ (6) bring the blood back from the organs and tissues into the right auricle (1). Thence it passes into the right ventricle (2), through the pulmonary artery (7) into the lungs. Returning from the lungs by the pulmonary veins (9), it passes through the left auricle (3) and left ventricle (4), and is distributed by means of the aorta (5, 5) and the carotids (8) to the organs and tissues of the body. Notice that the blood from the stomach and intestines passes through the liver before joining the general circulation. (From Huxley's "Physiology," modified.)

A. **Systolic Murmurs**[1] — *i.e.*, bruits added to or replacing the first sound —may be produced by the following causes, which are mentioned more or less in order of frequency: Hæmic conditions (see above, and Anæmia, § 402), mitral regurgitation, aortic stenosis, aortic aneurysm, tricuspid regurgitation, pulmonary stenosis, congenital heart disease, and cardio-pulmonary conditions.

I. In **Mitral Regurgitation** the systolic murmur is characterised by (i.) being loudest at the apex; (ii.) being conducted to the axilla, and also audible behind, at the angle of the scapula; and (iii.) owing to the resulting hypertrophy of the left ventricle, the apex is displaced downwards and outwards. There is accentuation of the second sound in the pulmonary area, due to the congestion in the pulmonary circulation. The pulse is soft, there is a characteristic florid physiognomy, and a tendency to dropsy.

General Symptoms of Mitral Regurgitation arise when there is failing compensation; and two events take place, which, in order of occurrence, are (1) *dilatation of the left ventricle* and *pulmonary congestion;* and (2) *dilatation of the right ventricle.*

1. The symptoms of *dilatation of the left ventricle* have been already mentioned (p. 70). *Pulmonary congestion* is revealed by laboured breathing, cough, expectoration of mucus, sometimes tinged with blood, or actual hæmoptysis. The physical signs are abundant mucous râles, and sometimes scattered patches of dulness at one or both bases.

[1] Systolic murmurs are sometimes spoken of as Ventricular Systolic, or V. S., murmurs, being produced by the systole of the ventricle.

Pulmonary apoplexy may be suspected by sudden increase of dyspnœa, accompanied by continuous hæmoptysis.

2. *Dilatation of the right ventricle*, consequent on the congestion of the pulmonary circulation, sooner or later produces the following symptoms and conditions :

(i.) A tricuspid *bruit* is sometimes heard (see below, p. 78).

(ii.) *Pulsation* in the epigastrium and in the veins of the neck.

(iii.) *Dropsy*, which indicates congestion of the whole venous system. Cardiac dropsy *starts and predominates in the legs* or *the back*, whichever may happen to have been in the most dependent position. The skin is tense, and is very liable to be attacked by erythematous, erysipelatous, and inflammatory conditions (cellulitis, ulcer, etc.). *Ascites* in varying amount is generally present. It is often an early and prominent sign in mitral stenosis. *Cyanosis* and a general lividity of the surface are consequences of the same venous stasis. A case of mitral disease, therefore, presents a marked contrast to one of aortic disease, where the countenance is pale and sallow.

(iv.) *Engorgement of the liver* is evidenced by pain and tenderness in that region, and jaundice of the skin and conjunctivæ. The organ is enlarged, and it may extend even to the umbilicus. Sometimes pulsation of the liver may be made out by placing one hand on the epigastrium, and pressing the other beneath the back in the dorsal region. In cases of *dropsy with albuminuria*, when we are in doubt whether the dropsy is of renal or cardiac origin, hepatic enlargement is a valuable diagnostic aid, for its presence is very usual in cardiac cases, but it is not one of the consequences of renal disease.

(v.) *Indigestion—i.e.*, want of appetite, a sense of discomfort in the stomach after meals, nausea or actual vomiting, with streaks of blood, indicate congestion of that organ.

(vi.) *Albuminuria*, with high-coloured scanty urine of high specific gravity (and possibly casts in long-standing cases), points to congestion of the kidney.

(vii.) *Splenic enlargement* and tenderness are the only indications of congestion of that organ.

Ia. A Murmur of Dilatation, systolic in rhythm, having all the above characters, and, like it, due to mitral regurgitation, may occur without definite disease of the valve, when the *left ventricle becomes dilated*, and the muscular ring around the valve *fails to complete* the closure of the mitral valve. This condition is especially apt to occur in the aged when dilatation of the left ventricle supervenes on hypertrophy.

II. **Aortic Stenosis** is another lesion producing a systolic bruit. True stenosis of the aortic orifice is not common, but a roughness, or the presence of vegetations on the inner surface of the valves, may produce the same bruit. The latter is mostly found in old people on account of the degenerative changes, and a systolic murmur thus produced is of relatively less serious import.

This murmur is characterised by (i.) being loudest at the second right interspace ; (ii.) it is conducted up to the vessels in the neck, and is audible in some cases also at the apex (Fig. 16) ; (iii.) it is usually harsh, sometimes musical, but in many cases it is barely audible ; (iv.) the pulse is small, sustained, and slow, sometimes anacrotic (§ 59). A systolic thrill is sometimes felt over the base of the heart.

General Symptoms are almost wanting in aortic stenosis—other than occasional pain, pallor of the face, and faintness or giddiness—until perhaps the mitral valve, owing to backward pressure, gives way (see Mitral Regurgitation, p. 76).

The detection of aortic stenosis is sometimes as difficult as mitral stenosis, and the characteristic murmur may be absent. It may then be suspected when the patient, generally an elderly man, presents persistent dyspnœa, bradycardia, nervousness, and occasionally anginoid attacks, which are not otherwise accounted for.

III. In Aortic Aneurysm a systolic murmur is the most common one heard. Accentuation of the aortic second sound is the most constant physical sign (§ 53).

IV. Tricuspid Regurgitation takes place when that orifice is diseased or dilated. Some maintain that if the valve be healthy, though dilated, no bruit can be heard, but it is certain that a murmur is often present in cases of confirmed bronchitis, which comes and goes under treatment, and which is not found to be attended with any marked changes in the tricuspid valve after death. The murmur is characterised by (i.) being heard best at the tricuspid area—*i.e.*, on the left side of the lower part of the sternum ; (ii.) it may be heard as far out as the right nipple ; (iii.) the pulse is of low tension, often irregular ; (iv.) owing to the accompanying hypertrophy or dilatation of the right ventricle, the area of dulness extends to the right, and there is epigastric pulsation ; and (v.) there is also pulsation of the veins of the neck, due to regurgitation into them, which is distinguished from the undulation seen in simple engorgement by the fact that the pulsation is not obliterated, but is rendered more distinct when the finger is placed on the external jugular vein, emptying the vein by the finger, and noting that it fills from below.

General Symptoms, as above indicated (p. 77) result from tricuspid regurgitation. By far the commonest cause is Chronic Bronchitis, which thus presents a clinical picture (p. 71) readily recognised.

V. Pulmonary Stenosis is practically unknown, unless it be part of congenital malformation of the heart. This murmur is systolic in rhythm, loud and harsh, and is heard over a very wide area, but most distinctly in the second left interspace.

Fallacies in the Diagnosis of Systolic Murmurs.—1. *Hæmic* murmurs (§ 402) are undoubtedly extremely common, and sometimes very difficult to distinguish from those of cardiac valvular disease.

2. A systolic murmur audible in the *aortic area*, and having all the characters of II. above, is due not so often to aortic stenosis as to (i.) roughening of the valve in old people from *atheroma* or calcareous deposit ; (ii.) simple *incompetence* of the aortic valves may produce both a systolic and a diastolic murmur ; (iii.) atheroma, with *dilatation* of the aorta, may also produce a systolic or a double bruit : then there is a ringing second sound in addition.

3. A systolic murmur heard best at the second left interspace is sometimes present in *mitral regurgitation* with a hypertrophied left auricle. It must not be mistaken for pulmonary stenosis.

4. The "*milk-spot*" murmur is due to a localised thickening of the visceral pericardium, appearing as a glistening white spot near the centre of the anterior

surface of the heart. Usually it is unattended by symptoms, but it may be of importance clinically, for it is apt to be mistaken for valvular disease. The "milk-spot" murmur (based on twenty-three observations, verified by autopsy, at the Paddington Infirmary) is generally a prolonged rough bruit, systolic in time, though occasionally double; it is *strictly localised* to a circle of 1 or 1½ inches radius, whose centre is situated in the third left interspace, close to the sternum, which is also its position of maximum intensity. Another important feature is that *at one time it is very rough and loud*, and a day or so later it may have completely disappeared. These features, and the absence of the concomitant symptoms of cardiac valvular disease, or of chlorosis, enable us to differentiate the milk-spot murmur from other conditions. It was found more often in hypertrophied hearts than in those of normal size. It has been variously attributed to tight-lacing, the soldier's shoulder straps, and other less probable causes. The condition is more frequently met with in adult or advanced life. A history of pericarditis was obtainable in only one of the twenty-three cases.[1]

5. A *congenital* murmur, usually systolic and localised to the base, has been known—in rare cases—to persist throughout life in some persons who have never

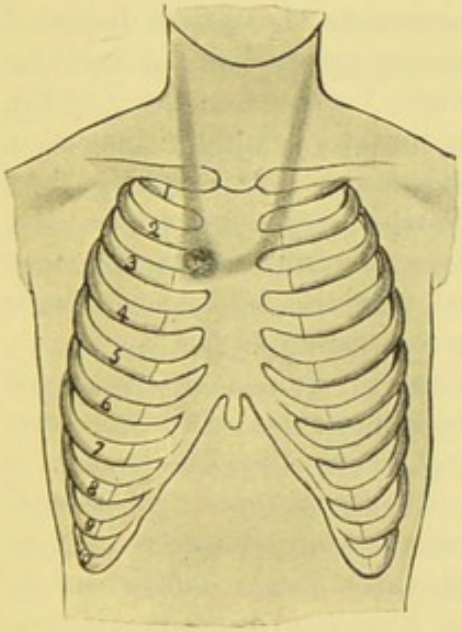

Fig. 16.—The systolic murmur of **aortic stenosis**. Depth of shading indicates intensity of murmur.

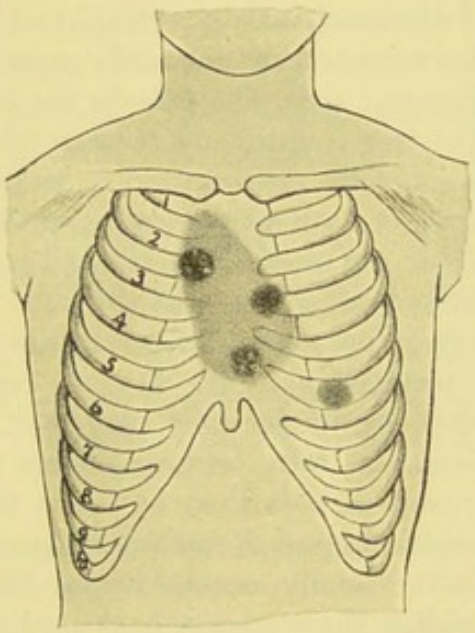

Fig. 17.—The diastolic murmur of **aortic regurgitation**. Depth of shading indicates intensity of murmur.

experienced any other manifestation of cardiac disease, although they have lived to a good old age.

6. *Cardio-pulmonary or Cardio-respiratory* murmurs are also rare, and are probably produced by the expulsion of air from the adjacent lung tissue by the movements of the heart. They do not indicate any cardiac lesion, and the lung may also be healthy; but they are sometimes associated with phthisis when the cavity is near the heart. They are heard in various parts of the antero-lateral region of the chest. They have a blowing, whiffing, or "sipping" character, are usually systolic in rhythm, and in rare cases double, though the systolic element is always loudest. Sometimes these murmurs disappear when the patient alters his position or posture. When he stops breathing, they may be weakened, abolished, or unaltered.

[1] The reader is also referred to an article by Professor Gairdner, in the *Edinburgh Med. Jour.*, vol. iv., part 2, p. 909 *et seq.*, and *Monthly Jour. Med. Sci.*, vol. xii. (1851), p. 103.

B. **Murmurs** heard in the **diastolic interval** may occupy either (*a*) the first half of that interval, replacing, accompanying, or following the second heart sound (*Diastolic* murmurs); or (*b*) they may occupy the second half of the interval, preceding and leading up to the first heart sound (*Presystolic* murmurs) (see Fig. 10).

Murmurs of the *first* kind are produced, in order of frequency, by aortic regurgitation, aneurysm, and pulmonary regurgitation; murmurs of the *second* kind are mostly due to mitral stenosis, very rarely to tricuspid stenosis.

I. In **Aortic Regurgitation** the murmur is *diastolic* (Ventricular Diastolic),[1] and is (i.) audible in the aortic area (second right space), but it is often loudest at the third left intercostal space; (ii.) it is conducted down the sternum, and audible at the apex (Fig. 17). It is, therefore, one of the loudest and most widespread of murmurs. (iii.) Owing to the amount of dilatation and hypertrophy of the left ventricle, the apex is displaced downwards and outwards more than in any other form of valvular disease. (iv.) The pulse is the characteristic "water-hammer" (§ 62). The face is pale, and the carotids visibly pulsate. Capillary pulsation is generally present, and is detected by drawing a line across the forehead, or by lightly pressing on the finger-nail or on the lips with a glass slide; the alternate blush and pallor due to the pulsation in the capillaries is thus well brought out. So great may this be that a pulse is sometimes communicated to the veins on the dorsum of the hand.

General Symptoms in aortic regurgitation: (i.) Pain about the chest, often of an anginoid character, may be complained of before compensation fails, or true angina may be present. (ii.) The anæmia is greater in this than in aortic stenosis. Faintness, giddiness, frontal headache, and disturbed sleep are common. (iii.) Dropsy is rare, as death usually occurs before the mitral valve yields sufficiently to produce the necessary backward pressure. Embolism sometimes occurs, though not so often as in mitral stenosis.

II. In **Mitral Stenosis** the murmur is *presystolic* in time. It occurs during the contraction of the auricle, and is known, therefore, as the auricular systolic or A. S. murmur. It is heard (i.) at the apex, and (ii.) over a very limited area. (iii.) The murmur is rough or rumbling, and *crescendo* in character, running up to a loudly accentuated first sound. (iv.) A reduplicated second sound is heard best just to the right of the apex.[2] (v.) On palpation, a characteristic sign in this heart

[1] *Diastolic* murmurs are sometimes spoken of as V. D. murmurs, being produced during the ventricular diastole. Similarly, *presystolic* murmurs are spoken of as A. S. murmurs, being produced during the auricular systole.

[2] This so-called "reduplicated second" is supposed to be due to a short diastolic murmur following the second sound, because it is heard at the apex, where the pulmonary second is said not to be audible; and because, if due to asynchronous closure of the aortic and pulmonary valves, from the high pressure in the pulmonary circulation, one would expect it to be heard in mitral regurgitation, and in the later stages of mitral stenosis, in both of which it is absent.

lesion is the presystolic *thrill*. It may be *felt* even before the murmur can be *heard—i.e.*, before the number of vibrations per second are sufficient to produce a musical note. In the later stages it disappears altogether. (vi.) The apex-beat is not displaced to the left *unless regurgitation be also present;* but the præcordial area of dulness is increased *to the right*, owing to the hypertrophy and dilatation of the right ventricle. (vii.) The pulse is small and of fair tension, until compensation fails.[1]

In the later stages of mitral stenosis the presystolic murmur disappears, and sometimes a mid-diastolic or an early diastolic murmur is heard (Fig. 18). Sometimes the second sound is absent at the apex.

Later still, there may be *no murmur at all*, and then the characteristic "slapping" (short, sharp) first sound and irregular rhythm of the heart form the sole auscultatory signs. This form of cardiac valvular disease is commoner in women, and, in my experience, in children.

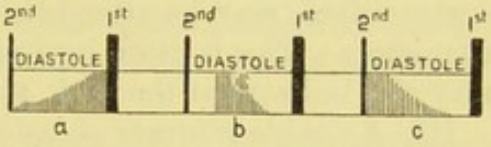

Fig. 18.—Three murmurs may be met with in MITRAL STENOSIS, which, as Dr. Bristowe has pointed out, may occupy different parts of the diastolic interval, and may therefore be called the (*a*) LATE, (*b*) MID, and (*c*) EARLY DIASTOLIC MURMURS. The late diastolic — *i.e.*, presystolic — murmur is the commonest; the early diastolic is least frequent. The reduplication of the second sound has been omitted for the sake of clearness.

General Symptoms. — (i.) Pulmonary congestion (p. 76, *ante*) is especially common; consequently hæmoptysis is more frequent in this than in other forms of valve disease; (ii.) emboli are also more frequent; (iii.) dropsy is rare until the end, but, on the failure of the right ventricle, all the symptoms of right ventricular dilatation appear. Liver enlargement is more common, but cyanosis and dropsy are less common in mitral stenosis than in regurgitation.

III. In AORTIC ANEURYSM a *diastolic* murmur is sometimes heard, but the murmur is usually systolic in time.

IV. TRICUSPID STENOSIS is a rare condition, but it is occasionally met with in young women, and is recognised by (i.) a presystolic murmur, heard loudest over the fifth right costal cartilage, close to the sternum. (ii.) Dropsy precedes the pulmonary engorgement, but in other respects the consequences are the same as those of regurgitation through this orifice.

V. REGURGITATION through the PULMONARY ARTERY is practically never met with, excepting either as an accompaniment of congenital malformation of the heart, or as part of a general valvular inflammation in acute ulcerative endocarditis.

FALLACIES IN THE DIAGNOSIS OF DIASTOLIC MURMURS.—1. A diastolic murmur due to *aortic regurgitation* may be heard at the *apex*. It must not be mistaken for mitral stenosis. In addition to the fact that the aortic murmur is heard louder at the base than at the apex, it has a uniform character, whereas a mitral diastolic murmur is broken, of varying intensity, and the pulse and other symptoms are different.

[1] Some (*e.g.*, Broadbent) say the pulse is regular in mitral stenosis, and that any irregularity infallibly indicates concurrent regurgitation. Others (*e.g.*, Guttman) hold that of all lesions, mitral stenosis alone can give rise to irregularity while compensation is good.

2. A *presystolic apical* murmur is occasionally heard with aortic regurgitation (Austin Flint murmur). It is diagnosed from that due to mitral stenosis by its not being followed by an accentuated first sound, and by the absence of the other signs of mitral stenosis.

3. *Mitral stenosis* is the most difficult form of valvular disease to detect in the second or third stages, when the characteristic murmur may be *altogether absent.* It may, then, be strongly suspected when there is—(i.) a loud, clear, sharp first sound at the apex, with marked accentuation of the pulmonic second sound; or (ii.) hypertrophy of the right ventricle, chronic pulmonary catarrh, and hæmoptysis, especially if the second sound is reduplicated or absent at the apex.

C. **Double Murmurs** may be produced by a combination of any of the above systolic and diastolic murmurs.

(*a*) Double murmurs most audible at the **base** (other than hæmic):

I. Combined Aortic Obstruction and Regurgitation is the most common condition, and causes a loud double see-saw murmur, heard best in the second right interspace.

II. Aneurysm of the Aorta, even in the absence of disease of the aortic valves, may be attended by a double murmur having the same characters. This is heard loudest in the second right interspace, but it may also be heard at the back, to the left of the fourth dorsal vertebra.

III. A double murmur occasionally occurs in the dilated aorta of the aged, but with less marked features.

IV. A double murmur, loudest in the pulmonary area, usually indicates Congenital Heart Disease.

(*b*) A double murmur most audible at the **apex** may be heard when both Mitral Regurgitation and Stenosis are present. It consists of a systolic bruit followed by a long diastolic murmur almost filling up the diastole.

Fallacies in the Diagnosis of Double Murmurs.—1. When a double murmur can be heard both *at the base and apex*, do not imagine that mitral regurgitation exists, as well as aortic disease. Remember that a systolic mitral and a systolic aortic may be alike in character, and that aortic murmurs can often be heard at the apex, as well as the base. To arrive at a conclusion is often very difficult, but one must rely on the position in which the murmur is loudest, and on the other features which distinguish mitral and aortic lesions.

2. When a *double aortic* murmur is present, the lesion may be regurgitation, or stenosis, or both together. A diagnosis is made by examining the pulse (p. 115), the rhythm of the thrill, if one is present, and the position of the apex-beat. In regurgitation the apex is displaced farther downwards and outwards than in any other form of valve disease. In aortic stenosis the left ventricular wall is hypertrophied, with but little enlargement of the cavity, and as emphysema is so often associated with it, the apex may be hard to find.

3. Murmurs of *pericardial friction* may easily be mistaken for a double aortic murmur.

4. *Hæmic, cardio-pulmonary,* and *milk-spot* murmurs are occasionally double.

§ **48.** General Symptoms of Cardiac Valvular Disease.—The first effect of valvular disease is *hypertrophy* of the heart, as already mentioned, and so long as there is adequate compensatory hypertrophy there may be no concomitant symptoms at all.

But, sooner or later, in most cases hypertrophy gives way to *dilatation*, and then a series of characteristic symptoms ensue. Those special to each form of valvular lesion have been referred to in the preceding section. Certain *general symptoms are common to all forms of chronic valvular disease.*

1. *Breathlessness* on walking uphill, or even on very slight exertion, is a very constant feature. No serious enfeeblement of the heart wall or disturbance of its function can exist without this symptom ; and it cannot be too much insisted on that breathlessness is not only a symptom, but, in general terms, is a measure of the extent of the cardiac failure.

2. *Dropsy* occurs early in mitral, late in aortic, disease.

3. *Palpitation* is of less diagnostic import, for it may occur without any organic heart change, and is not always present with valvular disease.

4. *Pain* is by no means always present in cardiac dilatation, but few cases run their entire course without considerable præcordial discomfort. Pain is a fairly common feature of aortic disease, and sometimes amounts to angina.

5. *Insomnia*, in advanced cases, is frequently a very troublesome symptom. Sometimes the patient, when dropping off to sleep, suddenly starts with the terror of suffocation, and gasps for breath. It is probably due to the disturbed and irregular circulation through the brain and medulla. *Headache* and *delirium* are also met with in advanced cardiac disease. The former is occasionally due to temporary high tension, but both are more often due either to pyrexia or to a toxic condition of the blood from failure of the emunctories. In either case free purgation is indicated.

6. *Embolism* may occur, having all the features described under Acute Endocarditis (§ 39). It is most frequent in mitral stenosis, and next in aortic disease.

§ **49.** The chief **Cause** of cardiac valvular disease in *youth* is acute endocarditis, which has a special tendency to attack the *mitral* valve, and in *advancing years* the chronic degenerative changes, which attack the *aortic* orifice.

1. *Acute Endocarditis* of rheumatic origin is by far the most frequent cause, and a large majority of " heart cases " date their symptoms from an attack of that disease in youth or early adult life. Scarlatina and the other acute specific fevers, and all causes of acute endocarditis (§ 39), play their part, but the other specific fevers are infrequent relatively to acute rheumatism and scarlatina.

2. Chronic Endocarditis may come on insidiously, especially under the influence of *certain poisons*, chief among which are alcohol, syphilis, and gout, and especially if these be combined with hard labour. Under such circumstances, the lesion usually affects the aortic orifice. But

chronic endocarditis more often supervenes upon acute endocarditis—an attack of which may have been overlooked.

Dr. C. O. Hawthorne[1] has done good service in drawing attention to the fact that endocarditis may start with an apparently trivial attack of subacute rheumatism, the child complaining of nothing but slight pains in the limbs, accompanied perhaps with a slight sore throat, not of sufficient gravity for him to be kept in bed. Parents of children whose antecedents are rheumatic should be warned not to treat such symptoms lightly.

3. *Degenerative* changes (*e.g.*, atheroma) are the lesions chiefly met with after middle life. They essentially affect the aortic orifice, either by injuring the valves or by causing dilatation of the aorta, which, extending to the situation of the valves, prevents them from meeting during the diastole.

4. Any prolonged *high tension*—*e.g.*, that which accompanies arterial sclerosis—may lead to valvular strain, usually aortic. Persistent obstruction in the lungs (*e.g.*, chronic bronchitis), or in the general systemic circulation, may have the same effect as persistent high tension on the right or the left side of the heart respectively.

5. Extensive or prolonged *muscular exertion* may, it is believed, lead to valvular mischief—at least, there is no other mode of explaining the fact that a large number of athletes have sclerosis of the aortic valves. In rare circumstances a sudden strain may lead to rupture of a valve.

6. *Congenital* conditions are referred to in § 46.

§ **50.** The **Prognosis** of chronic heart disease is but ill-understood if the conclusions are based only on hospital cases. They need to be followed from beginning to end as in private practice or infirmary work. It is quite certain that many patients have disease of the heart for years without knowing it. It is also certain that the first symptoms very often date from the patient knowing that he has cardiac disease, and unless there are special reasons to the contrary, a patient should never be informed of its presence.

Cardiac disease may terminate life in three ways: (i.) By sudden death—this may result either from syncope, or from rupture of the heart, or, as some say, from cardiac anæmia, due to non-filling of the coronary arteries; (ii.) by the occurrence of complications, especially bronchitis, and other pulmonary affections; or (iii.) by asphyxia, from dropsy of the pleura, often combined with congestion of the lungs.

The probable course and duration depend upon many considerations, but on nothing more than the condition of the *cardiac wall* (No. 3 below), and this should be the object of the most thorough investigation.

1. The presence of certain *Cardiac Symptoms* is in itself an indication that compensatory hypertrophy is giving place to dilatation—*e.g.*, palpitation, dyspnœa, increased by emotion or exertion, cardiac pain, syncopal and anginoid attacks. In actual practice the prognosis is good in proportion to *the amount of exercise a patient can take without producing breathlessness.* Syncope and anginoid attacks usually indicate serious cardiac embarrassment. Palpitation and cardiac pain are less serious indications; "Delirium cordis" and Cheyne-Stokes breathing are very grave.

[1] *Lancet*, 1900, vol. i., p. 1169.

2. The *Condition of the Pulse* is of considerable value in prognosis, but it has to be judged in connection with the valvular lesion. (i.) Irregularity is a grave indication except in mitral regurgitation. In aortic disease it is very serious ; and at all times intermittency or dropped beats is a symptom of most serious import. (ii.) Persistent rapidity is always a bad sign.

3. The *Physical Signs* of Cardiac *Hypertrophy* and *Dilatation* given in Table III., p. 70, will help us to gauge the amount of dilatation—*i.e.*, failure—or hypertrophy—*i.e.*, compensation—which is present, by means of a careful examination of the apex-beat, by percussion, and auscultation. If emphysema or other condition prevents us obtaining reliable conclusions, it is worth remembering that *a regularly acting heart with an apex in the normal situation*, justifies (with possible exceptions in mitral stenosis) a fairly good prognosis.

For purposes of prognosis—and, indeed, for treatment also—cases of cardiac valvular disease are best divided into three groups or stages. In the *first* stage there is *efficient hypertrophy*, with or without compensatory dilatation (in regurgitant lesions dilatation is also a compensatory process and aids the heart). In this stage the patient may not come under notice at all ; both the valvular mischief and the hypertrophy may be discovered accidentally.

In the *second* stage the *dilatation exceeds* that which is necessary, and some of the symptoms referable to the heart, above mentioned, are sure to be present. The dilatation, however, is not sufficient to produce pulmonary or systemic engorgement.

In the *third* stage there is *advanced dilatation*, with thinning of the wall and congestion of the lungs (in aortic lesions), of the general venous system (in mitral lesions), and finally of both.

4. The presence of *signs of venous obstruction* as a measure of backward pressure—viz., pulmonary congestion, dropsy, lividity of the lips and fingers, enlargement of the liver and spleen, and albuminuria—is unfavourable. But the gravity is very different in mitral and aortic lesions respectively. In *mitral* cases a moderate degree of these symptoms indicates only moderate cardiac failure, and it by no means follows that the heart is beyond redemption. But if they occur in *aortic* disease they show that the final stage is reached, and that the patient will probably not live many months. When general venous congestion exists, the relative *amount of urine* passed day by day is a good measure of the strength of the heart and the improvement made—a fact which is not generally mentioned.

5. Concerning the *Nature of the Valvular Lesion* as bearing on the prognosis, some difference of opinion is expressed as to the relative importance of aortic and mitral lesions.[1] My own experience is that

[1] Peacock and Bristowe place *aortic obstruction* as the most favourable, *mitral stenosis* next, *mitral regurgitation* next, and *aortic regurgitation* as by far the most

a moderate degree of aortic stenosis is the most favourable form, and if well compensated may give rise to little or no inconvenience; the patient generally dying of some intercurrent malady. Next in order comes mitral regurgitation, then mitral stenosis; the most serious being aortic regurgitation, the valvular disease which most frequently ends in sudden death. Combined lesions of stenosis and regurgitation are naturally more serious than single ones, and the gravest of all valvular lesions is double aortic disease.

In *Aortic Regurgitation*, the *measure of the amount* of regurgitation, and therefore the prognosis, depends upon the clearness with which one can hear the aortic second sound (as distinct from the murmur) in the carotid arteries, and on the degree to which the pulse collapses. In *Mitral Regurgitation* a loud murmur *following* the first sound is more favourable than a weak murmur, or than one which replaces or accompanies the first sound. In *Mitral Stenosis* a faint or absent second sound is a grave sign. Apical murmurs due to *dilatation* can generally be made to disappear under treatment.

In *Double Aortic Disease* it is important to note which is the louder, the first or the second of the two bruits. If the first be the louder, it indicates considerable compensating hypertrophy of the left ventricle, and the prognosis is more favourable; but if the second (the regurgitant) bruit be the louder, it probably indicates a weakened ventricle, which allows a large reflux of blood, and the prognosis is as grave as well can be.

6. The *Primary Cause* of the valvular mischief influences the prognosis to some extent. Injury and congenital mischief, both happily rare, are very serious. Rheumatism is grave in proportion to its tendency to recur. In general terms cases due to acute endocarditis in early life are much more favourable than the degenerative changes (accompanied perhaps by an alcoholic or syphilitic taint) supervening during middle life.

7. *Age* is not a very important factor. Valvular lesions in childhood are more readily compensated, but at the same time advance more rapidly. *Mitral stenosis* coming on in childhood is much graver than when it supervenes in the adult, and generally terminates fatally before the age of twenty-one. On the other hand, *aortic regurgitation* due to endocarditis in youth is compatible with a long and useful life; but when coming on in middle or advanced life, it is generally due to degeneration and dilatation of the aortic orifice—a condition of far graver import.

8. The *Temperament*, *Habits*, and *Means* of the patient will naturally influence his future. The prognosis is bad in the intemperate, and those who lead irregular lives. It is also unfavourable in the destitute, and in those who are compelled to work hard for their daily bread. Nevertheless, complete idleness is equally bad, and a patient should be encouraged to do as much as he is able without fatigue.

9. Finally, before hazarding a prognosis in any given case, the *Effects of Treatment* should always be watched, for it is sometimes truly wonderful how the skilful

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fatal. Sir Dominic Corrigan, on the other hand, and some other observers, regarded this lesion as consistent with unlimited longevity; but Dr. George Balfour states that cases of aortic incompetence generally "die within four years of admission to hospital." As regards this last statement, it depends really on the age at which it supervenes (see p. 86, par. 7).

administration of digitalis, and the application of modern methods of treatment, will sometimes seem to snatch the patient from the very jaws of death. The existence of an organic murmur without change, and not requiring active treatment for two years, justifies a favourable prognosis.

§ **51. The Treatment of Chronic Heart Disease** (including Cardiac Dilatation or Failure and Valvular Disease) may be considered under three heads: (*a*) When compensation is fully established; (*b*) when compensation begins to fail; (*c*) when compensation has broken down.

(*a*) When there is efficient compensation, no symptoms are present and no active treatment is needed, but much may be done to prolong the patient's life, and to avoid the supervention of cardiac failure. Subjects of chronic valvular disease should be enjoined to lead quiet, regular, and orderly lives. They should be warned particularly against the dangers of any *sudden*, unusual exertion, such as running to catch a train. With regard to exercise, it may be said, in general terms, that the patient himself is the best judge, provided always that he does not exert himself sufficiently to cause palpitation, severe dyspnœa, or præcordial pain. Some sports are more permissible than others; thus cricket, tennis, and golf may usually be enjoyed, whilst football, racing, and rowing must generally be forbidden. Climbing, especially to high altitudes, must be disallowed. Alcohol, tobacco, and tea are all myocardial poisons if taken to excess, and should be used only in strict moderation. The skin should be kept active by the daily bath, and the bowels regular by means of purgatives if necessary. Whenever possible, a means of livelihood should be chosen in which the heart is subjected to but little strain. A sedentary occupation with moderate exercise in the intervals, is more suitable than that which entails earning a living literally by the sweat of the brow. Lifting or carrying heavy weights, climbing ladders, wielding heavy hammers, and physical labour in constrained positions, are liable to overtax the powers for compensation of the cardiac muscle. Meals should be regular, and heavy meals should be avoided. The diet should be easily assimilable, and contain only a moderate amount of fluid. Anything requiring prolonged digestion disturbs the night's rest, so that it is sometimes a good rule to allow nothing solid after 2 p.m. in those who have a tendency to indigestion or gastric disturbances. A small quantity of stimulant with meals may be called for, but should not be used unnecessarily, because of the reaction afterwards, and of the tendency to excess, which exists in cardiac cases.

(*b*) When compensation is beginning to fail, the condition of the heart should be noted frequently, rest, drugs, and exercises being prescribed in accordance with the variations in the circulation and the capability of response to treatment by the cardiac muscle.

Drugs.—In cardiac failure, especially when the pulse becomes feeble, rapid, and irregular, digitalis is *par excellence* the remedy. It is especi-

ally indicated in failure of the right heart and in mitral regurgitation, whether primary or secondary to aortic lesions. It is contra-indicated when there is full compensatory hypertrophy, and the pulse is fairly strong, regular, and slow, or if vomiting is present. It is best to begin with 5 or 10 minim doses of the tincture twice or thice daily, and subsequently to reduce it. It may be continued for a considerable time in smaller doses in the form of a tonic. Its action may be promoted by the addition of spt. ammoniæ aromat. and senega. Digitalis not only affects the heart, but relieves the engorgement of the viscera. Strophanthus has much the same action as digitalis on the heart; it acts more rapidly, and does not constrict the arteries. The action of digitalis and many other cardiac remedies is expedited by an occasional dose of calomel. F. 54, 57, 59, 67, and 84 are useful. In *aortic valvular disease* and in *mitral stenosis*, where the pulse has other characters, digitalis is not so valuable a drug.[1] But in the later stages even of these affections, when compensation begins to fail, small doses of digitalis give relief. In aortic cases, where the blood-pressure is high, or where angina is present, the vaso-dilators are often of most use, such as nitroglycerine (in the form of liquor trinitrini ♏ i. t.i.d.), or erythrol-tetra-nitrite, sodium nitrite, and sal volatile. Belladonna is sometimes useful if there is relaxation of the vessels. Among the cardiac tonics strychnine, nux vomica, iron, and arsenic are the most valuable in the order mentioned.

The various symptoms may be met by appropriate remedies. For the *pulmonary congestion*, squills and stimulating expectorants are indicated. For *breathlessness*, spirits of ether or of chloroform and ammonia are useful. Nitroglycerine is useful where breathlessness is associated with high tension, which may accompany cardiac hypertrophy; and at the same time it cures the headache and sleeplessness due to the same cause. Ether ♏ xx. to xl., or strych. sulph. gr. $\frac{1}{60}$ hypodermically, are useful for the paroxysms of dyspnœa. *Cough* is relieved by drinks of hot milk, and drugs such as codeia, small doses of opium, and chloroform or ether. For *palpitation* alcohol is a most valuable cardiac stimulant, and relieves the breathlessness as well. Unfortunately, patients soon find this out for themselves, and thus cardiac valvular disease is a not infrequent cause of chronic alcoholism, especially among women, who take it secretly, during the night, when the palpitation is most apt to come on. The exact dose should therefore be carefully prescribed, and the quantity should always be moderate. Other causes of palpitation which may be present should be treated (§ 22). For *sleeplessness* opium or morphia hypodermically is useful; in mitral disease, however, where the liver is congested, opium is better avoided, and other drugs employed, such as potassium

[1] In mitral stenosis especially digitalis may cause irregularity, when strophanthus must be substituted.

bromide, sulphonal, trional, and paraldehyde. I have not found small doses of chloral do harm, as some maintain. The *hæmoptysis* of heart disease is best left alone, as it relieves the congestion. The *gastric* symptoms may be relieved by acting on the congested liver with calomel, ½ to 1 grain every night, with sodium sulphate and sodium bicarbonate (30 grains in 2 ounces of hot water) in the mornings. Digitalis must be stopped if it causes sickness. It may be necessary to give predigested food. For the treatment of *syncopal attacks* and *pain, vide* §§ 24 and 23 ; F. 56 is useful.

Massage and Systematised Exercises.—At one time rest was regarded as imperative for all forms of cardiac disease. But the advance of physiological knowledge has shown what an important part the skeletal muscles play in the circulation of the blood, by squeezing the fluids out of the soft-walled veins and lymphatics, while they cannot compress the lumen of the firm-walled arteries. There are three varieties of this treatment, which are invaluable for different degrees of cardiac failure. *First*, for the worst cases, *gentle massage*, combined perhaps with *passive movements*. These are available where any kind of voluntary movement on the part of the patient is attended with breathlessness. The great value of properly regulated massage in cardiac failure is well seen in the case from which the tracings in Fig. 20 were taken. *Secondly*, slow *voluntary movements* of flexion and extension on the part of the patient while standing or sitting. In the Nauheim system these voluntary movements are gently resisted by the operator—" resistance gymnastics "—see F. 114. These movements, combined with *baths* (see below), constitute the essence of the Schott system. *Thirdly*, Oërtel's method, which consists of three parts : First, reducing the amount of fluid taken to 31 ounces per diem (to include the amount contained in the solid food) and promoting perspiration ; secondly, a diet largely consisting of proteids[1] ; and thirdly, graduated exercise in the form of walking uphill, each day a little farther. Cases attended by plethora and obesity are the most suitable.

Baths, such as those in use at Nauheim, may be usefully added to the preceding. They act by relaxing the arterioles of the skin directly, and the arterioles of other parts reflexly. By these means blood is transferred from the venous to the arterial system, and its flow accelerated (F. 113).

(*c*) When compensation has broken down and marked cardiac failure is present, absolute rest is necessary. The patient is usually

[1] Oërtel's dietary is as follows :—*Morning :* 6 ounces of coffee, 3 ounces of bread. *Noon :* 3 to 4 ounces of soup, 7 to 8 ounces of roast meat or poultry, salad or green vegetable, a little fish, 1 ounce of bread or farinaceous pudding, 3 to 6 ounces of fruit ; no liquid (excepting in hot weather, 6 ounces of light wine). *Afternoon :* 6 ounces of tea or coffee (1 ounce of bread occasionally). *Evening :* one or two lightly boiled eggs, 1 ounce of bread, salad, fruit, sometimes a small piece of cheese, 6 to 8 ounces of light wine, with 4 to 5 ounces of water.

unable to lie down, but has to be propped up with pillows, and in severe cases sleep can be obtained only when the legs are hanging down. In severe failure of the right heart, as indicated by orthopnœa. lividity, distended jugular veins, the liver dulness extending well below the costal margin and the cardiac dulness extending far to the right, *venesection* is called for, and brings prompt relief. The abstraction of from 5 to 10 ounces of blood is usually sufficient; its efficacy is shown in Fig. 19, below. Three to six leeches may be applied to the right lower ribs in children, in whom venesection is more difficult to perform. The dropsy may require special treatment, such as draining the legs by Southey's tubes (§ 21) or multiple superficial incisions. Aspiration of a pleural effusion or paracentesis abdominis may be necessary. Diaphoretics are of little use in cardiac dropsy. Cardiac tonics, such as digitalis and caffein, should be employed in conjunction with diuretics, calomel, and hydragogue cathartics, such as pulv. jalapæ co. and cream of tartar. F. 55 is useful. Diuretin is a valuable drug

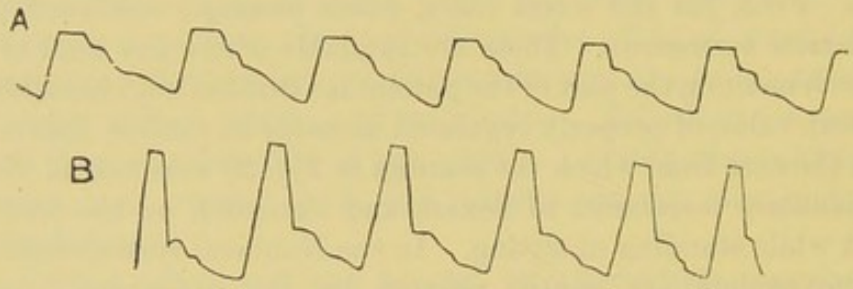

Fig. 19.—Tracings A and B show the efficiency of **bleeding**. A shows the flat top of high tension. B was taken immediately after 5 ounces of blood were removed, and shows the reduction thus effected, and also the senile character of virtual tension. The patient was about fifty-seven years of age, and suffered from cardiac valvular disease with recurrent high tension (with headache, etc.). The urine was always normal. Some years later he was brought in with apoplexy and died.

at this stage. The readily diffusible stimulants such as spirits of nitrous ether, alcohol and sal volatile are of great value.

VI. *On auscultation,* NO MURMUR *can be heard, and the heart sounds are very feeble; the* IMPULSE AT THE APEX IS SO WEAK *that it cannot be localised.* FATTY DEGENERATION *of the heart wall may be strongly suspected.*

§ **52. Fatty Heart** in its clinical sense indicates enfeeblement of the cardiac wall. A better term for it would be CARDIAC ENFEEBLEMENT, or Primary Cardiac Failure. Examples have now been given of all the various physical signs liable to be found in the heart by means of inspection, palpation, percussion, and auscultation. But, supposing the most careful examination reveals *no physical signs*, although, by reason of certain *subjective symptoms*, we believe the patient to be suffering from cardiac disease, DEGENERATION OF THE CARDIAC WALL should be borne in mind, the diagnosis of which often rests, as I have previously mentioned, on a process of exclusion.

Its detection is often a matter of some difficulty, but the disease may be *suspected* (i.) when the pulse and heart impulse are feeble, and the heart sounds perhaps inaudible; (ii.) if the patient be subject to attacks of fainting or of dizziness; and (iii.) if he be subject to palpitation and breathlessness. The pulse may be either very quick or very slow, intermittent, or irregular. The disease is more frequently met with in persons past middle age, and if unaccountable fainting attacks occur for the first time at this age period, fatty heart is the most probable cause. Some œdema of the ankles *may* also be present. (iv.) The heart sounds are not usually accompanied by a murmur, for even if valvular mischief exist, the force of the heart may not be sufficient to produce a bruit. The area of præcordial dulness may or may not be increased, but in any case it is often obscured by emphysema of the lungs, which is itself one of the causes of fatty degeneration. Later on, anginoid and epileptiform attacks are not uncommon, or the syndrome known as Stokes-Adams disease (§ 58).

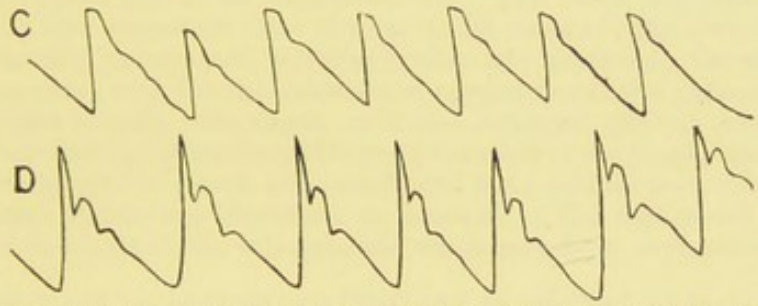

Fig. 20.—Tracings C and D, taken by a Marey's sphygmograph (in which the momentum of the lever is greater and the excursion larger, than those taken by Dudgeon's). C (which shows simply high tension) was taken before, and D (which shows the reduction of tension) was taken directly after massage—massage and passive movements. The patient was a man aged sixty-five, under care for arterial sclerosis, and these tracings show the efficacy of massage in relieving the heart.

The *Prognosis* is extremely grave. Fatty heart is one of the commonest causes of sudden death. The earlier stages of the malady are so insidious, that by the time pronounced symptoms appear great and irreparable mischief may be done. The patient may die in one of the syncopal attacks, or, if not, he will rarely live for more than six to twelve months after definite symptoms have become manifest.

Diagnosis.—In the early stages it may be impossible to distinguish Fatty Heart from Cardiac Dilatation, especially when emphysema is present, preventing accurate percussion. In *dilatation* (i.) the dyspnœa has often the peculiar character of being improved by using the voice (Broadbent). (ii.) The syncopal attacks of dilatation, when present, are sudden, with complete unconsciousness, and soon recovered from; those of fatty heart are prolonged, but less intense. (iii.) In dilatation there is nearly always backward pressure, leading to anasarca, fluid in the serous cavities, and congestion of organs.

Treatment consists of (i.) perfect rest, both of body and mind, and avoidance of anything like excitement; (ii.) stimulants in small and frequent doses—ammonia, alcohol, ether, combined with plenty of

easily assimilated nourishment, as in the form of mistura vini Gallici; (iii.) cardiac tonics, and especially strychnine, arsenic, and quinine, combined with plenty of fresh air. Digitalis should not be given if there be no signs of dilatation and the pulse be slow. It is, however, of great value in the opposite conditions. (iv.) The heart may be relieved of some of its work by passive movements, massage, and other measures described under Cardiac Valvular Disease, due care being exercised.

CHAPTER IV

ANEURYSM OF THE AORTA AND OTHER INTRATHORACIC TUMOURS

Anatomy.—The mediastinum is the irregular space in the chest which lies between the two pleural sacs. For descriptive purposes it is divided into four parts—viz., the *middle mediastinum*, which is occupied by the heart and pericardial sac; the *anterior*, which is the space in front; the *posterior*, the space behind; and the *superior*, the space above the pericardial sac. The most important structures contained in these spaces are: The thymus or its remains; the arch of the aorta with its branches (innominate, left subclavian, and carotids); the superior and inferior venæ cavæ, with the innominate and azygos veins; the pulmonary vessels, the trachea and bronchi; the vagus, recurrent laryngeal, phrenic, and splanchnic nerves; the cardiac and pulmonary plexuses; the roots of the lungs; the œsophagus, thoracic duct, lymphatic glands and vessels, and loose cellular tissue (Fig. 11, p. 48). The lymphatic glands are important on account of the occurrence of lympho-sarcoma and other glandular enlargements which may form mediastinal tumours.

Dulness with an Irregular Outline is referred to in the *italicised* remarks on diagnosis preceding § 46.

If, on percussing over the sternum,[1] or just beside it, the præcordial dulness is found to be **increased irregularly upwards**—the morbid condition may be Pericardial Effusion, Enlargement of the Left Auricle, Retraction of the Lung, an Abdominal Swelling pushing up the Heart as a Whole, or Aortic Aneurysm and some other Mediastinal Tumours. The two last-named are generally to be distinguished sooner or later by the presence of pressure symptoms (p. 96). If possible, a skiagram should be made.[2]

If, on auscultation over the **abnormal dulness** *near the base of the heart, there is a* reinforced or ringing second heart sound—*perhaps a systolic or diastolic murmur—the disease is probably* Aneurysm of the Aorta.

§ **53. Intrathoracic Aneurysm.**—Aneurysm of the aorta is undoubtedly the commonest of intrathoracic tumours. In regard to the anatomy of this serious and important malady, the student should study Fig. 11 (p. 48).

[1] Remember, in percussing over the sternum, the note elicited is of a much higher pitch than that just beside the sternum.

[2] Dr. Hugh Walsham, the *Lancet*, November 3, 1900.

The arch of the aorta is the favourite seat for aneurysmal dilatation. Its shape and the fact that it is subject to continuous strain make it surprising that the malady is not even more frequent. Any part of it may be affected—the ascending, transverse, or descending part of the arch. The dilatation may assume either a fusiform or saccular shape, the former being the more frequent. It may make its way in various directions, and it is extraordinary how bones, cartilages, and other hard structures, may become eroded and absorbed under its pressure. One of the earliest results of aneurysm near the root of the aorta is *cardiac hypertrophy*, but this may not occur at all when it involves other parts. According to its position, aneurysm of the aorta may be either very easy or very difficult to detect. If it involves the first part of the aorta, near the *front* of the chest, it is soon revealed by definite *physical signs*. If the second or third parts of the arch are involved, and the tumour extends backwards, there may be no physical signs, and even the *pressure symptoms* may be obscure. Thus the clinical manifestations belong to two categories—physical signs and pressure symptoms; and we have two varieties of aneurysm: (*a*) The *aneurysm of physical signs*, when the FIRST HALF of the arch is involved; (*b*) The *aneurysm of symptoms* (that is, pressure symptoms), when the SECOND HALF of the arch is involved.

The *Symptoms Common* to aortic aneurysm in all positions will be considered first, because these are the symptoms which will probably first attract our notice. Then we will turn to certain others *special* to the first, second, and third parts of the arch respectively.

Symptoms COMMON TO ALL POSITIONS:

1. Dyspnœa is often one of the earliest complaints which the patient makes. When it is due to pressure on the trachea, as in aneurysm affecting the transverse portion of the arch, it is persistent and stridulous in character. When it is due to pressure on the anterior pulmonary plexus, as in aneurysm of the first part of the arch, it is often paroxysmal.

2. Cough is generally present and has a characteristic brassy sound (gander cough). Pressure upon the recurrent laryngeal nerve is common, with consequent paralysis of the left vocal cord, and there may be hoarseness or even aphonia from the same cause. *Paralysis of the left vocal cord*, in the absence of central nerve lesions, practically always means aortic aneurysm. Laryngoscopic examination should be a matter of routine in all suspicious cases, because abductor paralysis occurs before complete paralysis, and the former may be unattended by any alteration of voice.

3. Pain in the chest is another common symptom. It may occur in attacks of an anginoid character, shooting down one or both arms, usually the left, especially in aneurysm of the first part of the arch. The pain may be neuralgic when there is pressure on nerves; or it may be

of a dull boring character when due to erosion of bone, such as occurs in connection with aneurysm of the descending arch.[1] Short of definite anginoid attacks of this kind, patients with aortic aneurysm are liable to feelings of suffocation, constriction, or "spasm" in the chest, and nameless dreads come over them from time to time without cause. Such attacks may in many cases be brought on by bending the head backwards, or by any movement which stretches the neck. I have known patients with dilated and rigid aorta suffer from the same symptoms.

4. A reinforcement of the aortic second sound is the most constant of the auscultatory signs of aortic aneurysm. It is sometimes spoken of as a "ringing second" sound, and is due to the increase in the aortic tension (Broadbent).

5. The diastolic shock or thud is an equally important sign. It is felt by the hand or the stethoscope, and is synchronous with the second sound.

6. Inequality of the radial pulses is a fairly frequent symptom. It is present whenever the aneurysm is so placed as to cause a difference in the arterial tension in the great vessels which spring from the aorta. The typical aneurysmal pulse occurs in the one just beyond the sac, and its characteristic is a loss of the pulse-wave, the blood flowing in one continuous stream.

7. Inequality of the pupils occurs from pressure on the sympathetic. In the early stage the irritation of the nerve causes dilatation of the pupil on the same side. Later on there is paralysis, with contraction of the pupil, accompanied sometimes by vascular dilatation and unilateral sweating of the face and neck.

8. The heart may be displaced when the aneurysm is large, usually to the left. It is sometimes hypertrophied.

(*a*) Symptoms peculiar to aneurysm of the **ascending** or **first part of the arch.** Aneurysm of this part of the arch is usually easy of detection, and in marked cases the *Physical Signs* are unmistakable. (i.) On auscultation, an accentuated second sound is usually to be heard; and in a large number of cases, where the dilatation involves the valvular orifice, a diastolic murmur is also heard. Over the site of the aneurysm a systolic murmur is always present, and this is frequently present also at the aortic area. Thus, a double murmur at the aortic area is found in many cases. (ii.) Any percussion dulness present is continuous with that of the heart. It usually extends to the right of the sternum, but this depends upon whether the aneurysm makes its way forwards or not. The left heart gradually hypertrophies. (iii.) On palpation, the diastolic shock is very characteristic. Sometimes there is a thrill felt also in the suprasternal notch. (iv.) When the aneurysm is so large

[1] A case is mentioned in the footnote to p. 37 in which this was almost the only symptom.

as to form a tumour, the swelling expands laterally with each systole of the heart. (v.) The right bronchus may be pressed upon, leading to diminished or absent respiratory murmur (R. M.) of the right lung. In severe cases there may be pressure on the superior vena cava, with œdema of the neck and arms. (vi.) The dyspnœa is paroxysmal ; and the right recurrent laryngeal nerve may be involved, with right laryngeal paralysis.

(*b*) The symptoms of aneurysm of the second or **transverse part of the arch** may be equally easy to detect when it makes its way forwards. But when the posterior part is affected it may present considerable difficulty in diagnosis, especially from other intrathoracic tumours. (i.) The dyspnœa may be either paroxysmal or continuous, with inspiratory stridor, owing to the pressure upon the trachea. (ii.) Pressure upon the left bronchus may lead to diminished breath sounds in the left lung, and symptoms (2) and (5) above are specially marked in aneurysm of the transverse arch. (iii.) Tracheal tugging is a very characteristic sign of aneurysm in this situation. Standing behind the patient, hold the cricoid between the finger and thumb, and press gently upwards, the patient sitting in a chair erect with the chin up (see Fig. 21). In this way the pulsation is transmitted by the trachea to the hand. (iv.) The physical signs—which are in this situation less marked, or may be absent—consist of a thrill felt on palpating the suprasternal notch ; dulness on percussion over the manubrium, continuous with that of the heart, and extending from the middle line to the left of the sternum ; and auscultatory signs as described above—(*a*) (i.).

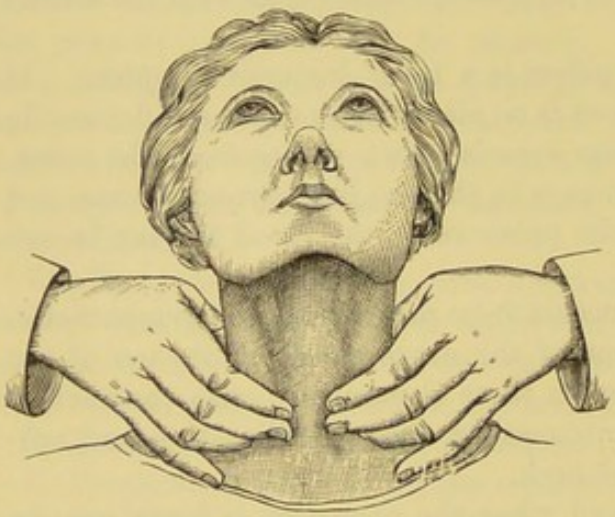

Fig. 21.—**Tracheal tugging**, showing position of hands in order to elicit this symptom.

(*c*) The symptoms of aneurysm affecting the **descending aorta** may be very obscure. (i.) Intense pain in the back is the most common symptom, and there may be no other for a long time (*case* in footnote, p. 37). The pain may pass to the side, following the course of an intercostal nerve. (ii.) Other pressure symptoms, such as dysphagia, from pressure upon the œsophagus ; wasting, from pressure upon the thoracic duct ; disease of the left lung, from pressure upon its bronchi ; and any of the other symptoms mentioned on p. 94. (iii.) If the swelling enlarges, physical signs to auscultation and percussion may become apparent in the left (occasionally the right) scapular region ;

and in advanced cases there may even be a pulsating swelling without the knowledge of the patient. Osler found that in such cases there is absence of pulsation in the femoral arteries.[1]

Etiology.—(1) Aortic aneurysm is far more frequent in men than in women, especially those in the prime of life—namely, between the ages of thirty-five and fifty. (2) It is especially frequent among soldiers and those who do laborious work. This liability has been attributed to the wearing of belts and the like, but it is probably due to the fact that these classes are subjected to sudden and severe muscular exertion and heart-strain at certain times. It also occurs among blacksmiths for the same reason.

(3) Both syphilis and alcohol are potent agencies in the production of arterial degeneration. Alcohol acts probably in two ways—partly by predisposing to degeneration of the aortic walls, and partly by over-stimulating the heart from time to time.

(4) As an exciting cause some cases of aneurysm date from a period of overexertion, exposure, and destitution, or from an injury.

Diagnosis.—The diagnosis of a deep-seated aneurysm is sometimes as difficult in the early stages as it is easy when the aneurysm is situated superficially. The diagnosis from *cardiac valvular disease* and other causes of cardiac hypertrophy (§ 43) is made by the occurrence of the pressure symptoms. Many of the local signs of aortic aneurysm may be produced by a *dilated and rigid aorta*, but here, again, the pressure symptoms are wanting. The *throbbing aorta* of aortic regurgitation is apt to be mistaken for aortic aneurysm, and it is sometimes impossible to differentiate these conditions. The throbbing aorta in Graves' disease and severe cases of anæmia may also give rise to difficulty. *Mediastinal growths*, on the other hand, may have the same pressure symptoms as aneurysm, and can only be diagnosed by the absence of the physical signs referable to the heart. There is no murmur on auscultation over the dull region, and the area of dulness is usually not so limited or defined. Finally, the course of mediastinal tumours rarely lasts longer than eighteen months. Of late, radiography has been employed in the diagnosis of the presence and nature of intrathoracic tumours.

Prognosis.—By treatment much can be done to prolong life, and the patient may live a good many years if his occupation does not necessitate much exertion. Death may occur in four ways—from rupture, exhaustion, cardiac failure, or complications. The rupture usually leads to a sudden and copious hæmorrhage, which terminates life; but sometimes there is a slight leakage, which may recur at intervals of a few days. With aneurysm of the *ascending* aorta rupture usually takes place into the pericardium, pulmonary artery, or superior vena

[1] Osler, "Prin. and Pract. of Med."

cava ; with aneurysm of the *transverse* arch, into the trachea (a very frequent situation) or into the bronchi ; and, when the *descending* aorta is involved, the blood usually finds its way into the pleura or œsophagus. The process may be so gradual that there is no sudden onset of symptoms, such as dyspnœa, or cyanosis, or bleeding, and death may not take place for some time. But generally, as in the cases just narrated, it is copious and sudden, death speedily ensuing. The severity of any case is measured to some extent by the amount of dyspnœa present and the rapidity of the evolution of symptoms. Other consequences or complications are due, for the most part, to the effects of pressure—such as collapse or a low form of pneumonia of the lung, hydrothorax, and œdema of the head and neck.

Treatment.—The indications are three in number : (*a*) To lessen arterial tension ; (*b*) to slow and steady the heart ; and (*c*) to increase the blood coagulability in the hope that laminated clot will form in the sac. Absolute rest in bed must be enjoined. This alone may accomplish very considerable relief, and there is no doubt that some of the extraordinary results claimed for certain remedies have been due to rest. Much can be accomplished by diet. It should be of the smallest quantity consistent with life. Tufnell's dietary[1] is based on this fact, and in it only 8 ounces of fluid and 10 ounces of solid are allowed per diem. It must be persevered in for three to six months. The good derived from this dietary mainly depends on the reduction of fluid. Drugs should be employed to steady the heart and reduce the tension (see Pulse). There is, however, one remedy which is undoubtedly capable of materially improving thoracic aneurysm—viz., iodide of potassium in large and gradually increasing doses, commencing with 20 grains, three or four times a day.

Calcium chloride in large doses, in view of its known power to increase coagulation, is worthy of trial. The digestive organs often need attention. For the pain, morphia, atropine, or belladonna, internally or in the form of a plaster, are used ; if of anginoid character, nitroglycerine. Even if the dyspnœa is very urgent, do not perform tracheotomy unless it is due to bilateral laryngeal paralysis. If there be an external swelling, some elastic support is needed. Calomel is valuable for high arterial tension ; aconite for palpitation. For venous distension or severe dyspnœa venesection may be performed. Surgical measures have been adopted from time to time in the treatment of superficial aneurysms, but they are not free from danger. Of such we may mention acupuncture, galvanopuncture, and the injection of coagulating fluids such as perchloride of iron (a dangerous procedure). Distal

[1] The solids may consist of well-cooked meat or fish and biscuit, and for the fluid 10 ounces of milk are permitted per day. From 12 ounces to 18 ounces solid may be permitted, but the fluid must not exceed 16 ounces. It must be combined with absolute rest, and drugs are better avoided.

ligature of one of the great vessels sometimes leads to improvement, especially if it is involved in the aneurysm.

Lancereaux introduced a novel method for the treatment of aneurysm by the hypodermic or intramuscular injections of a solution of gelatin, and Dr. Robert Maguire informs me he has had considerable success with this plan[1]; but Dr. Golubinin does not think this plan is successful.[2]

Other Mediastinal Tumours.

§ 54. The **Symptoms of Mediastinal Tumour** belong to three categories—namely, (*a*) the signs of displacement of organs; (*b*) the physical signs of tumour; (*c*) the symptoms of pressure. There are also (*d*) certain symptoms special to the different kinds of tumour.

(*a*) The **displacement** of organs is sometimes the first intimation we receive. The liver is rarely displaced, but the lungs and heart are often pushed to one side, and the apex-beat may be found in the axilla.

(*b*) The physical **signs of tumour** appear sooner or later on the anterior or posterior aspects of the chest, and consist of: (1) Dulness on percussion, corresponding to the position of the tumour; (2) auscultatory signs, which differ somewhat with the position and nature of the tumour. If it be solid, the breath sounds will be tubular, and there will be an increased conduction of the heart sounds. If it contain fluid (such as aneurysm or, more rarely, hydatid) there will be a diminished respiratory murmur, and in the case of aneurysm a characteristic murmur (§ 53). (3) Ausculto-percussion will aid in defining the boundaries of the tumour. (4 Radiography is now used for defining the nature and position of mediastinal growths.[3]

(*c*) The symptoms of mediastinal tumour which are due to **pressure** on the various structures around are as follows:

(1) Dyspnœa always appears sooner or later, and may be of a type peculiar to mediastinal tumours when there is pressure upon the trachea; it has a stridulous character, which resembles tubular breathing heard without the aid of the stethoscope. The breathlessness is often paroxysmal or asthmatic when there is pressure upon the heart and cardiac plexuses; or it may be of a Cheyne-Stokes nature. But the character of the dyspnœa depends upon whether it is the heart, the great vessels, the bronchi, or the nervous apparatus of the heart, lungs, or larynx, which is pressed upon by the growth of the tumour.

(2) Cough, sometimes of a laryngeal brassy character, is also present, and it is accompanied by expectoration if, as is usual, there is also bronchitis or congestion of the lungs. There may be laryngeal paralysis from pressure upon the recurrent branch of the vagus, and hoarseness, or even aphonia, may result.

(3) Cardiac and circulatory symptoms, such as palpitation, or a difference in the pulses of the two sides in the neck or radial arteries.

(4) Dysphagia, from pressure on the gullet, is present chiefly with posterior mediastinal growths.

(5) Inequality of the pupils may appear, owing to pressure on the sympathetic. Usually the pupil on the affected side is contracted from paralysis of the sympathetic, but it may be dilated during the stage of irritation.

(6) Pleuritic effusion occurs if there be pressure on the thoracic veins.

(7) The inferior vena cava is rarely compressed, but lividity or œdema of the head, neck, and arms may occur from pressure on the superior vena cava.

(8) In suspected tumour of the anterior mediastinum, it is well to remember that when the head is thrown back, the veins of the neck become distended, owing to the increased thoracic pressure producing venous obstruction.

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[1] See Discussion, Medical Society, the *Lancet*, December 1, 1900.
[2] *Brit. Med. Journ.*, October 13, 1900, p. 60 (Epitome).
[3] Dr. Hugh Walsham, *loc. cit.*

(9) Pain down the arms and in the back occurs when there is pressure on the spinal nerve trunks.

(*d*) **Causes.**—There are certain symptoms which are **special to the nature and situation of the tumour.** There are five clinical groups of tumours, in addition to aortic aneurysm.

I. Malignant Tumours, which may be primary or secondary. If, in addition to the above physical signs, the expectoration present a constant prune-juice character, and if on paracentesis a bloody fluid is drawn off, the presumption is strongly in favour of malignant tumour. Out of 520 cases of mediastinal tumour, Hare[1] found 134 were cancerous. Cancer of the mediastinum is the commonest mediastinal tumour, because it is usually secondary to cancer of the lung or œsophagus. In the latter case it is situated in the posterior mediastinum. Primary cancer, as of a bronchus, is rare, and tends to affect the anterior mediastinum. *Sarcoma*, especially lymphosarcoma, may start in the mediastinum as a primary growth, or originate from the pleura and from the thymus remains. Primary sarcoma is most frequent in the anterior mediastinum. If secondary in origin (as when the abdominal viscera are the seat of the primary tumour), it invades chiefly the posterior mediastinum. In primary mediastinal sarcoma enlargement of the glands in the neck and elsewhere may occur.

II. Innocent Mediastinal Tumours, though more rare than the foregoing, are sometimes found in the mediastinum—*e.g.*, fibroma, dermoid cyst, hydatid. Lipoma, gumma, and enchondroma, the latter growing from the sternum, are also occasionally met with.

III. Enlargement of the Mediastinal Glands.—With these there is often dulness posteriorly in the upper half of the interscapular space, but occasionally there is dulness over the sternum. Paroxysms of coughing, "croupy" or like whooping-cough, may be present, together with stridulous breathing from pressure upon the trachea. The *causes* of enlarged bronchial glands are :

(*a*) *Tubercle*, which is generally secondary to tubercle of the lungs. It is more common in children than in adults. The condition may be suspected when concurrent disease of the lungs is present, and symptoms such as the above arise. If the glands suppurate, sweatings and intermittent temperature become more pronounced than when the lung only is diseased. An abscess may form and open into a bronchus (compare IV. below).

(*b*) *Lymphadenoma* (*Hodgkin's disease*) may start in the anterior mediastinal glands. Hare mentions twenty-one cases which he included under the names "lymphoma" (lymphosarcoma) and "lymphadenoma." Lymphadenoma cannot be diagnosed with certainty from lymphoma unless the ordinary symptoms of Hodgkin's disease be also present—viz., (i.) enlargement of the glands in other parts of the body ; (ii.) attacks of pyrexia.

(*c*) *Bronchitis* and the *pneumonia* which complicates measles, influenza, and whooping-cough, are often attended by enlargement of the bronchial glands, which may occasionally be recognised behind the sternum in children.

(*d*) *Whooping-cough*, without bronchitis or other disease of the lungs, may give rise to swelling of the bronchial glands, although the condition may be hard to make out. Some observers consider that it is the pressure of these glands which causes the paroxysms of whooping-cough.

IV. Suppurative Mediastinitis (abscess of mediastinum) is a rare condition which may affect the anterior or posterior mediastinum, or both, but more often the anterior. (i.) The most prominent symptom is pain, in the site of the inflammation, or passing down the nerves pressed upon. (ii.) Dulness, with œdema and redness, may be present over the upper part of the sternum if the disease be in the anterior region, or over the dorsal spines if in the posterior mediastinum. Pulsation communicated from the aorta may be present, and lead to a diagnosis of aneurysm, but the pulsation is not expansile, and fluctuation may be felt.

[1] Hare ("Mediastinal Tumours," Philadelphia, 1889) found out of 520 cases, 134 were cancer, 98 sarcoma, 21 lymphoma, 7 fibroma, 11 dermoid, 8 hydatid, 115 suppurative mediastinitis.

(iii.) Pyrexia is present, usually of a hectic type, with the rigors, sweats and weakness which attend all deep-seated inflammations. The causes of the acute form of mediastinitis are trauma, erysipelas, and the eruptive fevers. The chronic form is usually due to tubercular disease. It may rupture in various directions.

V. Enlargement of the Thymus.—A certain degree of enlargement is normal to childhood, and this may give rise to a dulness over the manubrium. In the rare cases in which enlargement is met with in after-life it is sometimes due to hyperplasia, as in Graves' disease, but generally to carcinoma, either primary or secondary.

Prognosis.—In all cases of intrathoracic tumour which are large enough to produce symptoms the prognosis is unfavourable. Moreover, all of these conditions entail much suffering to the patient. Malignant tumours are fatal in six to twelve months, depending upon the site and progress of the growth. Innocent tumours may last for a long time. Syphilitic, tuberculous, and simple inflammatory glandular enlargements may recover under treatment, but even in these no confident prognosis of recovery can be given in any case. Suppurative mediastinitis may open externally, and run a course of a few days or weeks only ; other cases are chronic, and last for years, or lead to pulmonary gangrene and other serious complications when the pus burrows into adjoining organs.

Treatment in intrathoracic tumour is almost wholly palliative. For aneurysm, see § 53. Abscesses, hydatids, or growths connected with the sternum may be dealt with by the surgeon in some cases.

CHAPTER V

THE PULSE AND ARTERIES

§ 55. The Meaning of "The Pulse."—By the term "pulse" is understood the expansile sensation communicated to the finger by the alteration in the shape of the artery, due to the momentary increase of blood-pressure which takes place during the systole of the heart, and which is transmitted to the periphery *in the form of a wave*. It has been shown that there is no dilatation of the artery; the increased output of blood for the moment raises the blood-pressure, and alters the shape of the channel from an oval to a circle. The examination of the pulse is of extreme importance, not because it has a set of diseases of its own, but because it affords us so many valuable practical hints about the diseases of other organs, and about the general condition of the patient. "Many of the indications obtained from the pulse do not depend upon a comprehension of the circulatory conditions which the varieties of the pulse denote, or, indeed, upon a knowledge of the circulation at all. Observant physicians before the time of Harvey could gauge thoroughly the state of the patient in fever from the pulse, and it is not for the purpose of estimating the movement of the blood that we ourselves, in a case of fever, count the beats and note their force and volume. We calculate from the data thus obtained the strength of the sufferer, and the effect upon him of the disease. On the other hand, it is only through a knowledge of the conditions which govern the circulation that such facts as the connection between kidney disease and cerebral hæmorrhage can be understood, and that the prognostic significance of the hard pulse, which betrays this connection, can be appreciated."[1]

For the production of the pulse three factors are requisite: (i.) The contractions of the ventricle, which determine the frequency and rhythm of the pulse, and to a large extent its force; (ii.) the elasticity of the large vessels; (iii.) the peripheral resistance found in the arterioles and capillaries. These three factors must always be considered in studying the pulse.

§ 56. Clinical Investigation.—It is preferable not to examine the pulse until the preliminary excitement occasioned by the doctor's visit has subsided; and in all accurate records the pulse should be noted under similar conditions as regards the posture of the patient, time of day, relation to meals, etc.

The radial pulse is the one usually selected for examination, since it is easily accessible and lies against a bone. But the pulse can be observed in other situations—*e.g.*, the temporal, dorsalis pedis, or

[1] Sir William Broadbent, "The Pulse," first edition, p. 75. London, 1890.

popliteal arteries. Three fingers should be placed along the course of the artery, the index finger next the heart, and allowance should be made for much adipose tissue. The different means of eliciting the several features will be dealt with below. When feeling the pulse, its special features may often be brought out more fully by holding up both wrists with the fingers on the pulses. Only experience and comparison between all types of pulse can give to the physician the necessary aptitude for observation and correct inference. It is impossible here to enter upon all the complex data of the circulation, but the leading practical points which are of use in clinical work will be indicated.

A complete observation of the pulse should comprise six features, the first four being the most important.

1. *Rate and Rhythm* (*i.e.*, regularity).—The rate of the pulse per minute is easily calculated by the watch, and in making this observation it should be remembered that a physiological acceleration occurs after any exertion, excitement, or after a meal, or even by the visit of the doctor. The pulse is faster in the evening than in the morning, and it is faster by about eight beats per minute in an upright than in a recumbent posture.[1] If the pulse be irregular, the type of the irregularity (*vide infra*) must be noted.

2. The *Force* or strength of the pulse depends largely upon the force of the heart beats, and is best measured by its *compressibility*—the finger next the heart presses the vessel until the wave is no longer appreciable to the other fingers. By the amount of pressure required to obliterate the wave, the force with which the blood is propelled from the heart can be estimated. A " full bounding pulse " is one which has strong pulsations, but it is not necessarily one of high tension. Indeed, a full bounding pulse may occur in fevers where the tension is generally low. The STRENGTH of the pulse is measured by the force or *strength of the pulse wave ;* the TENSION of an artery (*i.e.*, the blood-pressure) is measured by its condition *between the waves.*

3. The *Character of Each Beat* is observed by noting (i.) whether the pulse wave *rises* suddenly or gradually ; (ii.) the *duration* of the beat, whether long or short ; and (iii.) whether the *decline* is abrupt or gradual. It is important to note the presence or absence of *dicrotism*, which is a marked feature in low tension (see § 62).

4. The state of the *Arterial Tension* or blood-pressure is perhaps the most important pathological feature of the pulse, and it is estimated

[1] The pulse is faster in the female than in the male, and it varies considerably at different ages, thus :

In the fœtus and new-born infant its average rate is				140	per minute.
Under 1 year	,,	,,	,,	120	,,
Under 3 years	,,	,,	,,	100	,,
From 7 to 14	,,	,,	,,	90	,,
From 14 to 21	,,	,,	,,	80	,,
From 21 to 65	,,	,,	,,	70	,,
In old age	,,	,,	,,	80	,,

by the degree of fulness of the artery *between the pulsations*. Normally the vessel is hardly felt between the beats if the wall is healthy. The vessel should be rolled transversely under the fingers, and, if the arterial tension is high, it stands out like a cord *between* the beats.

5. The *Size of the Artery* and the *State of its Walls* will require fuller consideration later on, but it is important to note these features, because an artery of small size may give the impression of a weak pulse. The thickness of the wall must also be noted, because a thick-walled artery may give the impression of high arterial tension.

6. The pulse of *both Radial Arteries* should be compared, so that any abnormalities may be detected. It should be part of the routine to examine both pulses, as by this procedure we may detect the existence of unsuspected disease,such as aneurysm or other intrathoracic tumours.

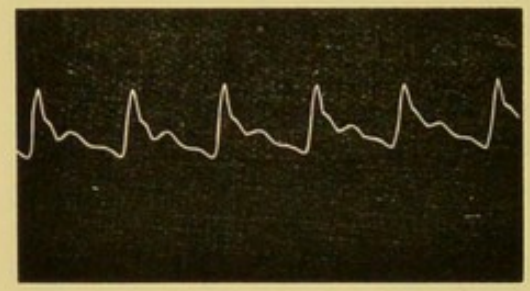

FIG. 22.

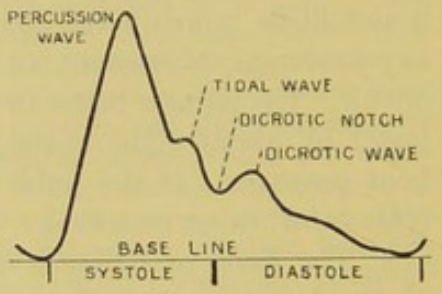

FIG. 23.

Fig. 22.—NORMAL PULSE TRACING, taken with the author's modification of Dudgeon's Sphygmograph. Rate 68; pressure about 2 ounces. Fig. 23.—NORMAL PULSE TRACING (Fig. 22) magnified, with the names of the principal parts. The dicrotic (or aortic) notch indicates the closure of the sigmoid valves, and therefore the termination of the ventricular systole and the commencement of the ventricular diastole. The diastolic line is that part of the tracing from the dicrotic notch to the next percussion wave.

Abnormalities such as a more or less superficial position of the radial on one side or the other exist more frequently than is supposed.

It will be advisable to consider the Pulse under six headings: I. Infrequency; II. Rapidity; III. Irregularity; IV. Intermittency; V. High Arterial Tension (or Blood-Pressure); VI. Low Arterial Tension. They are relatively of very different importance. Alterations of tension are of the greatest moment, and irregularity comes next. The **causes** of these will be considered; their treatment, with the exception of high and low tension, belongs to the causal conditions.

The SPHYGMOGRAPH is an instrument employed to obtain a record on paper of the characters of the pulse. The first one used was that of Marey. In this instrument a pad placed over the pulse is connected with the short arm of a lever; the long arm, which magnifies the pulse wave, is sharpened to a point, and makes a tracing on smoked paper.

The handiest instrument, however, is that of Dudgeon. This is a little instrument which is strapped on to the wrist. Some years ago Weiss made for me a modification of the latter which can be used without a strap—an appendage which I regard as unnecessary, since the instrument can more readily, with a little practice, be steadied and adjusted by the hand of the operator. The manipulation of any of these instruments is easily acquired by experience. The chief precautions are: (i.) To place the pad *exactly* over the artery, and it is of great

assistance if the course of the vessel has been previously marked by an aniline pencil ; (ii.) the *amount of pressure used*, and the adjustment of the instrument, should be such as to obtain the most graphic record.

The SPHYGMOGRAM or sphygmographic tracing is very useful as a graphic record of the pulse, and to show the progress of the case from day to day ; but it does not tell us as much as the educated finger, and its readings can never be quite accurate because the exact amount of pressure exercised by the pad upon the artery cannot be known. Figs. 22 and 23 represent a normal pulse tracing, the principal named parts of which it consists being indicated in the latter. (1) The *percussion wave* is abrupt

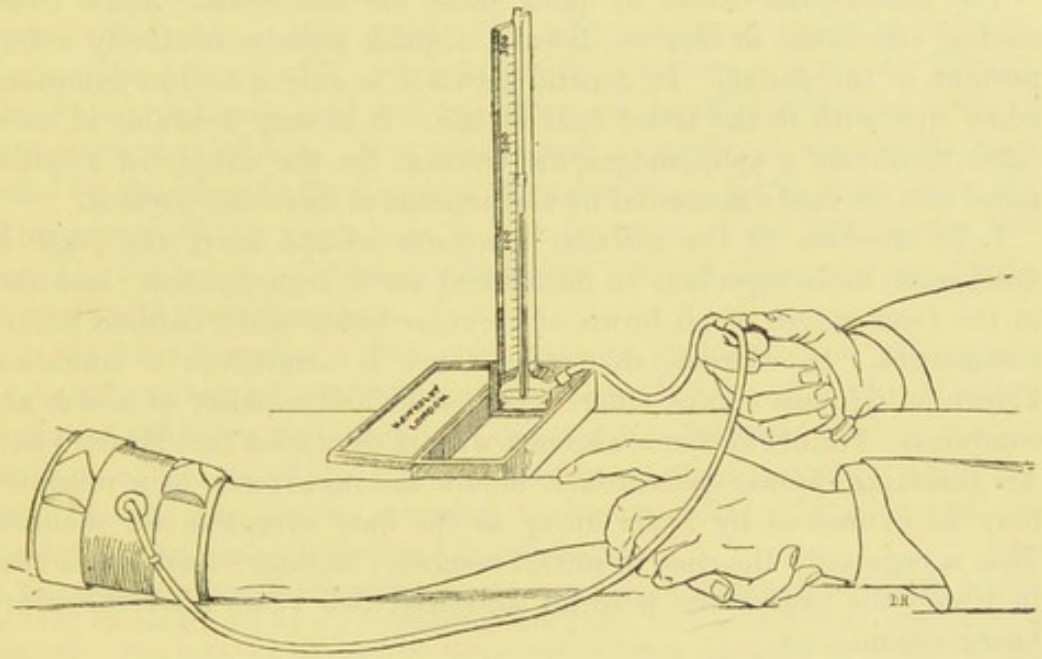

FIG. 24.—RIVA ROCCI'S SPHYGMOMANOMETER.

The armlet should be wrapped round the patient's arm above the elbow (the arm should either be bare or at most covered only by a thin shirt) ; the armlet should be strapped on, and it is important to see that it fits snugly. One end of the rubber tube should be attached to the armlet, and the other end to the cistern of the manometer, the inflator and liberating valve being in the middle. The left hand should be used for feeling the pulse, and with the inflating bulb in the right hand, air should be pumped into the apparatus until *the pulse at the wrist is felt to stop*. The pressure, as indicated by the manometer at which this occurs, should be read off, and then (by a very slight turning of the milled head of the valve) allowed to decrease slowly, and the exact pressure at which the pulse returns at the wrist should be noted. This will be the maximum *systolic* pressure in the brachial artery. After each observation the pressure in the apparatus should be released. The maximum systolic pressure for an ordinary healthy adult is between 120 and 130 millimetres of Hg *approximately* under fifty years of age, and between 130 and 145 millimetres in later years. To obtain the so-called *diastolic* pressure with this instrument, after the pulse at the wrist has been stopped, the pressure in the apparatus should be slowly released by a turn of the valve, and the pressure at which the maximum pulsation of the surface of the mercury in the manometer occurs should be noted. In taking repeated observations in the same patient, it is important that the same arm should be used each time, and that the patient should always be in the same position—*i.e.*, either sitting or lying down.

and vertical in proportion to the force of the ventricular contraction and inversely proportional to the peripheral resistance. (2) The *tidal wave* is prominent in proportion to the amount of peripheral resistance and the force of the ventricular contraction—*i.e.*, when the arterial tension is *high*. When the peripheral resistance is very low there may be no tidal wave. (3) The *dicrotic wave* is pronounced and the *aortic notch* more marked in proportion as the peripheral resistance and the heart force (*i.e.*, the arterial tension) are *low*.

Many instruments have been devised for the MEASUREMENT OF THE BLOOD-PRESSURE, the most accurate of them would seem to be RIVA ROCCI'S SPHYGMOMANOMETER, a modification of which is shown in the accompanying figure (Fig. 24). Hill and Barnard's sphygmomanometer, used with a broad armlet, is also very good.

§ **57. Quick Pulse.**—The rapidity of the heart-beat varies considerably within the range of *health*, and in many persons the heart may at times beat 150 a minute for a short time without inconvenience. In infancy the normal rate is 130, and this may continue in after-life.[1] The pulse is normally more rapid during the menstrual period and menopause, in the evenings and after meals. After a severe illness the pulse more easily becomes rapid.

The *pathological* causes of quick pulse are numerous. Apart from cardiac affections, or Graves' disease, a quick pulse is relatively unimportant in the young. In general terms it is only a serious symptom when met with in the latter half of life. It is very desirable in such cases to obtain a sphygmographic tracing, for the danger of a quick pulse may be fairly measured by the amount of dicrotism present.

1. In diseases of the *valvular structures* of the heart the pulse is quickened, more especially in mitral and aortic regurgitation ; and also in the later stages of all forms of valvular lesion when cardiac failure commences. In extreme dilatation there is sometimes a condition known as *delirium cordis*, where irregularity and rapidity of action are combined. Fœtal rhythm is also met with in dilatation (see Heart, § 44). An *insidious chronic endocarditis*, before the occurrence of a murmur, may be evidenced by heart hurry as the only symptom for months. This is especially the case in mitral stenosis ; Balfour[2] mentions a case in which the presystolic murmur appeared two years after the heart hurry commenced.

2. In *cardiac dilatation* (apart from valvular disease) the pulse is quickened, especially if accompanied by arterial atheroma or sclerosis. In the latter half of life heart hurry is nearly always associated with dilatation of the heart, the result of a degenerative change. Thus, one of the earliest signs of the failure to compensate for the obstruction caused by arterial disease is a persistent tachycardia. Quick pulse, indeed, is regarded by some (*e.g.*, Balfour, *loc. cit.*) as the leading feature of both " irritable " and " senile heart."

3. Various *toxic conditions* increase the heart rate. The heart hurry which attends chronic alcoholism is very serious, as indicating fibroid or fatty degeneration of the heart wall, or neuritis of the vagus. Tobacco first slows the heart, but in large doses paralyses the vagus ; hence excessive smoking may induce paroxysmal tachycardia. Digitalis in large doses also paralyses the vagus (Balfour). Belladonna increases the rate, fulness, and force of the beat, and increases the blood-pressure (Balfour), but in toxic doses it paralyses the vagus and produces tachycardia. Tea and coffee may produce temporary heart hurry.

4. Many *affections of the nerves*, functional and organic, are attended by heart hurry, usually transient, but sometimes persistent. Emotional rapidity of the pulse is familiar to everyone. Paroxysmal tachycardia is described elsewhere (§ 40). Tumours pressing on the vagus may be evidenced by heart hurry, even if the tumour be small ; a rapid pulse may, indeed, be the only symptom.

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[1] See footnote, p. 103.
[2] " The Senile Heart," 1894.

5. In *Graves' disease* heart hurry is often the earliest symptom, and in this disease there may be throbbing in the whole arterial system, the heart sounds being clear and distinct.

§ 58. Infrequent Pulse, Slow Pulse, or Bradycardia (*i.e.*, slow heart), is met with under several conditions. In *health* a slow pulse is normal to certain individuals without any very obvious explanation, and without any other symptoms. Napoleon is said to have rarely had a pulse-rate over forty. I have known a gentleman for twenty years, whose age is now eighty-two, who has never, to my knowledge, had a pulse-rate over fifty when in health : its average is forty. He has always enjoyed very good health. In some families it is met with as a hereditary condition. It is always well to verify an apparently slow pulse by listening to the heart at the same time, for in some cases the infrequency of the pulse may be due to some of the heart-beats not reaching the wrist. *Pathologically* a slow pulse is of importance chiefly in heart and brain disorders.

1. *In heart disorders* a slow pulse without irregularity is uncommon. It may, however, be met with in connection with the senile heart, where it always indicates some degree of dilatation. Sclerosis of the coronary arteries, atheroma of the aorta, and fatty or other degeneration of the cardiac wall, may also be attended by bradycardia. It is one of the cardinal symptoms of Stokes-Adams disease.

Stokes-Adams Disease (Heart-Block) is a rare disease, first described by R. Adams in 1827, characterised by a permanently slow pulse, with syncopal and epileptiform attacks. The patients are usually advanced in years, complain of dyspnœa, and have marked bradycardia, the pulse-rate ranging from 20 to 40. Any mental excitement is liable to bring on an attack. The breathing becomes stertorous, the face cyanosed ; there is dilatation of both pupils, rigidity of the body, accompanied by clonic movements of the arms ; the pulse beats more and more slowly, and finally ceases ; the jaw drops, and for forty to eighty seconds the patient is to all appearance dead. No pulse is felt in either wrist, and on auscultation the cardiac sounds are inaudible. Then a feeble sound is heard, followed by a stronger, and a second later the pulse begins beating (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Many such fits may occur in succession, from six to ten in a single night. There is no albuminuria.

Etiology.—This symptom-complex occurs in association with arterio-sclerosis and myocardial degeneration in old people ; in young people with syphilis and coronary disease. In heart-block there is a blocking of the normal stimulus from the auricles to the ventricles, due to disease affecting the muscle bundle of His. During an attack there is cessation of the ventricular systole, whilst the auricles continue to contract.

Treatment.—Inhalations of strong ammonia may avert an attack. The general condition requires attention. Strychnine and bromides are recommended.

2. If a slow pulse is associated with marked high tension, *arterial sclerosis* should be suspected, even when no sign of arterial disease can be discovered.

3. Various *gastric derangements* are frequently associated with a slow pulse, probably in a reflex manner. This is, perhaps, the commonest cause of slow pulse in children. In adults it may be associated with gastric ulcer and carcinoma. In chronic dyspepsia a slowness of the pulse is usually associated with some amount of irregularity and intermission.

4. Many *nervous disorders* may be associated with slow pulse. Thus :

(i.) If the temperature is raised, the arteries contracted, and the pulse slower than normal, and if with this there be some irregularity in rhythm, and perhaps a reduplicated second sound, in a child, we probably have to do with an early stage of meningitis.

(ii.) Cerebral tumour is another nervous condition associated not infrequently with slow pulse. Here, however, it probably only occurs in the late stages, and as a pressure symptom. Halberton mentions a case in which a violent blow on the head was followed by a permanently slow pulse, with syncopal attacks, succeeded by epilepsy. The post-mortem revealed narrowing of the foramen magnum.

(iii.) Various psychical disorders, such as melancholia, general paralysis, and epilepsy, may also be attended by slow pulse.

(iv.) An abnormally slow pulse has been observed in association with spinal injuries, especially affecting the medulla and cervical regions.

5. *Drugs*, such as digitalis and strophanthus, slow the rate of the heart, and, if irregular, steady its rhythm. Belladonna and tobacco at first slow the heart.

6. Slow pulse is also met with in *toxic* conditions, such as diabetes, jaundice, uræmia, and poisoning by carbon monoxide.

7. In states of prolonged *exhaustion* and anæmia, and in *convalescence* from acute illness, bradycardia occurs.

§ **59. Irregular Pulse (Arrhythmia).**—In *health* irregularity is rare, except in old age and under the influence of transient emotion (mental or bodily shock). In nervous subjects it may occur, as the result of tea, coffee, or tobacco in excess. In *diseased* states the pulse may be irregular in rhythm, in force, and in volume. Irregularity of volume or force is of more importance than irregularity of rhythm. The prognosis of irregularity is less favourable than that of intermission. In acute diseases irregularity of the pulse is a bad omen, because it indicates cardiac exhaustion.

1. Irregularity is chiefly found with *cardiac valvular disease*, and constitutes one of the leading indications of *failing compensation*. In mitral stenosis it sometimes occurs even while compensation is perfect ; but irregularity of the pulse is especially marked in the failing compensation of mitral regurgitation. As a point of distinction we may remember that the tension in mitral stenosis is relatively high, in regurgitation it is low.

2. Irregular pulse, in the *absence of all cardiac lesions*, is usually the result of some reflex disturbance of gastric origin. This may occur after a heavy meal, or in association with a distended colon, either of which may cause irritation of the vagus terminations, or produce a direct pressure upon the heart. Any mechanical impediment to the heart may produce irregularity, and this may be the explanation of the frequency with which irregularity attends emphysema. In this case the expiratory may differ from the inspiratory pulse rate, the former being the more rapid. Sometimes chronic gout, and sometimes atheroma, are attended by an irregularity of the pulse.

3. *Fatty* or *fibroid heart*, without valvular disease, is often evidenced by irregularity. If the pulse becomes regular after a little exercise

there is not much amiss with the heart wall, but if the irregularity is increased, it indicates that there is serious damage.

In *pulsus paradoxus* there is complete, or almost complete, disappearance of the pulse during inspiration. It has been shown by Dr. Maguire[1] to be due to either (1) an increase of the "negative" intrathoracic pressure which normally takes place at the end of inspiration, or (2) an extreme weakness in the left ventricle, or to both. Maguire has shown that it can be produced in even healthy persons at the end of inspiration by so contriving that the negative intrathoracic pressure can be *suddenly* increased. It is met with in intrathoracic tumours, mediastinitis, and adherent pericardium.

The *pulsus bisferiens* is a rare and peculiar type of pulse occurring in some cases of aortic disease (stenosis or regurgitation, and especially double). It is apt to be confused with the dicrotic pulse, though it indicates neither increase nor diminution of tension. A double beat is felt because the ventricle makes two distinct efforts during the systole. It is distinguished from dicrotism by becoming more evident by pressure, whereas the dicrotic pulse is obliterated by pressure.

The *anacrotic* pulse is a somewhat rare condition, resulting from high arterial tension. In it the tidal wave is higher than the percussion wave. It is found in some cases of aortic stenosis and aneurysm, where the ventricle has to overcome abnormal resistance, and the blood cannot rush out in full volume during the systole.

The following special kinds of irregularity are found chiefly in mitral disease, and especially in mitral disease under treatment by digitalis.

(*a*) In the *pulsus myurus*, a rare form, there is one strong beat followed by a succession of beats of diminishing volume.

(*b*) In the *pulsus alternans* there are two beats of the heart to one of the pulse, or it may be that the second is perceptible at the wrist, but only with difficulty.

(*c*) In the *pulsus bigeminus* the beats occur in couples, a strong beat being followed quickly by a somewhat weaker one, after which there is a pause. On auscultation the heart's action may be found to correspond with this irregularity, but sometimes an abortive contraction occupies the position of the missed beat. Examination of the heart in *pulsus bigeminus* gives the impression of a contraction only of the right side of the heart during the abortive beat (Broadbent, *loc. cit.*). In the *pulsus trigeminus* there are three beats and a pause.

§ **60.** In the **Intermittent Pulse,** which is of less importance than irregularity, a beat is missed at intervals, after several (say 5 to 20 or 30) regular beats. On auscultation the heart is found sometimes to pause entirely as the pulse does, but more frequently it makes a rapid and imperfect contraction, corresponding with the missed beat at the wrist. No symptoms may be experienced by the patient, but sometimes there is cardiac discomfort, or palpitation. If intermission be due to a gastric or nervous cause, the patient is, as a rule, conscious of it; if, on the other hand, it be associated with a cardiac condition, he is more often unconscious of it. Intermission is of minor importance if it can be made to disappear with exercise.

In *health* intermission may be found either as a constant feature, or after meals, and in these circumstances the pulse is restored to normal during exercise, fatigue, excitement, and pyrexia. In some people tea, coffee, or tobacco will produce intermission.

(1) Intermission of the pulse may attend *cardiac valvular disease*,

[1] "On the Dynamics of the Thorax in Disease," Dr. Robert Maguire, *Clin. Journ.*, August, 1898.

but it is rare unless associated with irregularity (*vide supra*). (2) It also occurs with fatty heart and in other *lesions of the wall* (see Irregularity); but (3) intermission is due more frequently to gastric derangement, and occurs most often after a heavy meal. It is fairly common in chronic gout.

§ **61. High Arterial Tension**[1] (*i.e.*, HIGH BLOOD PRESSURE) when habitual or recurrent, is undoubtedly the most serious of the pathological conditions of the pulse, because of the continuous strain it produces upon the cardio-vascular system. Normally the artery collapses completely beneath the finger between the beats; but if the arterial tension or blood-pressure be too high *the artery remains full between the beats*, and sometimes stands out like a cord, so that it can be rolled under the finger. In applying this test we must beware of the fallacy of a thickened artery, also the "recurrent pulse"—*i.e.*, a wave flowing back from ulnar to radial artery. The pulse beat or wave of high tension *may* appear small, because when the artery is constantly and fully distended, it cannot be much more distended by the pulse wave. High blood pressure is revealed in a sphygmogram (Fig. 25), by the small extent of the excursion made by the lever. In high tension the upstroke is short and sloping, and the percussion wave, it will be seen, tends to be flat-topped, and the dicrotic wave is almost absent. The blood pressure may now be measured with more scientific accuracy by the instruments described on p. 105, those of Riva Rocci, or Hill and Barnard. By means of these instruments great advances have been made in our knowledge of blood pressure and its variations.

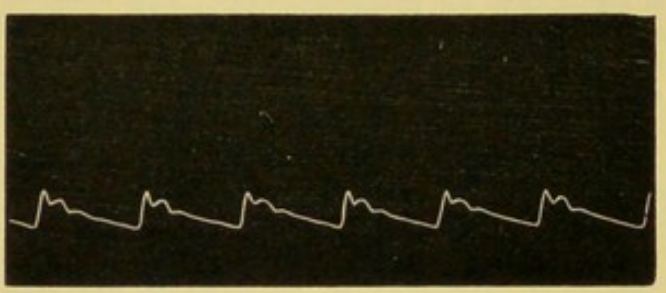

Fig. 25.—HIGH TENSION in a case of Chronic Bright's Disease, with Albuminuric Retinitis, under the care of Dr. J. S. Bristowe. High tension is indicated graphically by (1) prominence of the tidal wave (unless the arteries are very atheromatous); (2) smallness of the dicrotic wave, and its occurrence high up on the diastolic line; (3) *gradual* sloping of the diastolic line towards the next upstroke.

VIRTUAL TENSION is a term which was applied by Sir William Broadbent to a condition of pulse due to *rigidity of the aorta*, which simulates high tension. In consequence of this rigidity, the systolic shock is transmitted unmodified to the periphery, and the distension of the radial artery is *at first* maintained, as in true high tension, though only for a fraction of a second; then it suddenly collapses. It will be best understood by consulting the sphygmogram (Fig. 19, B, p. 90; and Fig. 31, p. 119), where the percussion wave is seen to be flat, or rather square-topped. The first half only, or one-third, of each beat is in a state of high or sustained tension; the remainder is low tension. Virtual tension is the leading feature of the *senile pulse*, since in the aged a rigid aorta is usual.

[1] It has become a common error to speak of "pulse tension," "high-tension pulse," etc. It is not correct to say that a wave has pressure or tension, but the terms have become sanctioned by custom.

The *Symptoms* which accompany high arterial tension, and which lead us to suspect that condition, are very important, though somewhat variable. They consist of (i.) headache, which may be frontal, occipital, or vertical, accompanied by vertigo from time to time, and a constant feeling of fulness about the head. Sometimes the headache assumes the character of migraine, sometimes of neuralgia. (ii.) There may be some lassitude, disinclination for exercise, and depression of spirits. (iii.) Breathlessness on exertion is common ; very often it is paroxysmal, and the patient thinks he has asthma. (iv.) Wakefulness, or sleeping by dozes, is not uncommon.

High tension is rare in children, but these symptoms occurring in adults, especially in those past middle life, are suspicious, and are confirmed if, upon examination, we find the following physical signs : (i.) The pulse revealing the above characters ; (ii.) on auscultation an accentuated aortic second sound (sometimes accompanied by a reduplicated first sound at the apex) ; (iii.) later on, if the condition persist or frequently return, cardio-vascular hypertrophy supervenes.

The *Causes* of high arterial tension are numerous, and of considerable importance. Among the *predisposing causes* heredity undoubtedly plays a most important part. " No condition, indeed, runs more strongly in families than high arterial tension, and it is the explanation of a family liability to apoplexy and paralysis, or to death from heart disease " (Broadbent, *loc. cit.*). No age is exempt from high arterial tension, but it is found far more frequently at and past middle life. As regards sex, males are more subject to the disease, since they are more exposed to the dietetic and other influences which raise arterial tension.

Exciting causes bring into operation one or more of three pathological factors—increased peripheral resistance, increased cardiac force, or increase in the volume of the blood. The exciting causes are as follows, arranged fairly in order of importance:

1. Anything in the mode of life which leads to *deficient oxygenation* and *elimination of nitrogenous waste*, such as : (i.) Excess of nitrogenous food—meat, and even poultry and game, soups, beef-tea, and other meat extracts and preparations containing extractives. The condition is believed to be more common among meat-eating races. (ii.) Alcoholic drinks of any kind, even in moderation. (iii.) Sedentary habits, or any mode of life leading to deficient exercise in the open air. (iv.) Constipation is another very important cause. All these four causes lead to increased resistance in the capillaries, in the opinion of some observers [1] by producing a toxic condition of the blood, which acts either directly on the capillary walls, so provoking their contraction, or by causing cohesion of the blood to the capillary wall. Constipation may act also in another way—viz., through its effect upon the abdominal circulation, for it drives the blood out of the large abdominal veins into the general circulation, and so raises the general blood-pressure. This is the current explanation, but it seems to me that the toxæmia which results is far more likely to act by irritating, and so constricting, the peripheral arterioles.

2. Several *toxic conditions* of the blood act probably in the same way. (i.) Renal disease is a very familiar antecedent and accompaniment of high arterial tension. The renal diseases here referred to are those which result in inflammation or degeneration of the parenchyma—acute and chronic desquamative nephritis, and granular kidney—*i.e.*, those which lead to the imperfect elimination of nitrogenous waste. (ii.) Gout, where there is probably excessive retention of nitrogenous waste. High arterial tension so frequently accompanies this condition

[1] *E.g.*, Sir William Broadbent, *loc. cit.*

that it is known sometimes as the "gouty pulse." (iii.) The diabetes which occurs in persons past middle life is attended by high tension, but not that more fatal form of diabetes which occurs in younger subjects. (iv.) Plumbism, which is intimately connected with gout and renal disease. (v.) Emphysema, and sometimes other lung conditions (probably by deficient oxygenation). (vi.) Anæmia sometimes. (vii.) Pregnancy is invariably accompanied by increased tension, probably on account of the hyperinotic condition of the blood.

3. Any condition leading to a *persistent contraction of the arterioles* results in high arterial tension. It is highly probable that some of the above toxic conditions may act in this way. Migraine, which is attended by constantly recurring spasm of the peripheral vessels, thus comes to be attended by high arterial tension. One of the results of high tension is an increase in the muscular coat of the vessels, and this in turn tends to perpetuate constant high tension.

4. *Plethora*, by increasing the volume of the blood, necessarily results in increased arterial tension.

5. *Cardiac valvular disease* is more often attended by a diminution than an increase of blood-pressure. Mitral stenosis is almost the only variety with high tension, though we may get all the features of high tension in aortic stenosis. It is, however, in *cardiac hypertrophy* that we have the greatest tendency to high tension. Whenever the heart beats more rapidly and more powerfully—as, for instance, during excitement or during exertion—more blood passes into the vessels in a given time, and there must necessarily be a rise of pressure. This high tension is usually transient, but if frequently repeated, as in athletes, it may be a forerunner of a persistent high arterial tension.

6. In certain *neuro-vascular* diseases there is a tendency to dilatation, and in others to spasm, of the peripheral vessels. In the latter (*e.g.*, Raynaud's disease) there is a frequently recurring tendency to high tension.

Pathological Effects of High Tension and Prognosis.—Temporary high arterial tension is not serious, but when it constantly recurs or continues over many months or years, it has very grave ulterior results. By the physiological law that increased function results in increased growth, there is hypertrophy of the muscular tissues of the whole of the vascular system—that is to say, hypertrophy of the heart and of the muscular tissues of the arteries (arterial hypermyotrophy, § 68). This may be said to constitute the first stage. If the increased tension continue, a degeneration occurs in the muscular tissue of the heart and of the arteries. The heart muscle gradually degenerates and its cavities dilate. The muscular tissues of the vessels undergo first a granular degeneration (the early stage of sclerosis), which is attended by hardness, loss of elasticity and of contractility. The second stage is manifested chiefly by the failure of the heart to compensate for the increased peripheral resistance caused by the rigidity of the arteries. Moreover, on account of the changes in the vessels, rupture of the peripheral vessels is a common consequence; and as the vessels of the brain have least support, they are the most frequent seat of rupture. Loss of elasticity and atheroma of the aorta are other consequences of persistent high tension (see § 66).

The subject of *prognosis* will now be more readily understood. People with increased tension may live for a long time, but there is a constant liability to the accidents above mentioned, and they certainly do not live so long as those without high tension. The general

habits and temperament of the patient greatly influence the prognosis both as regards the removability of the causes and the liability to accident. When the heart is hypertrophied the prognosis is not serious so long as compensation does not give way, for the progressive changes can be arrested by lowering the tension and improving elimination. Even in chronic Bright's disease proper treatment may prolong life for a considerable time. In hereditary cases some light may be thrown upon the prognosis by inquiry as to the ages and causes of death of the ancestors. The prognosis is good in general terms where the causes are removable, such as constipation, temporary lithæmia, or plethora, and in cases where the tension is brought down by the administration of salines. On the other hand, it is grave in advanced renal disease, alcoholics, and those who will not, or cannot, adhere to treatment. As regards the untoward signs, a ringing accentuated second sound (indicating dilatation of the aorta) or excessive tortuosity and beading of the vessels (indicating advanced vascular disease) are of unfavourable import. Dilatation of a valvular orifice secondary to vascular strain is more serious than primary valvular disease of the heart, because in the latter, the weakened heart has not the extra work of overcoming the vascular resistance.

Treatment.—The two main indications are to keep the blood free from impurities, and to relieve the strain on the circulation. General hygienic measures occupy a very prominent position. Moderate regular exercise and plenty of fresh air, with a minimum of nitrogenous food, and the avoidance of alcohol, are cardinal principles. Butcher's meat should also be avoided, and sugar taken sparingly (§ 212) ; copious libations of water aid elimination, and great benefit may be derived from the Turkish bath. Purgatives should be regularly administered, especially salines, such as Carlsbad salts, magnesium sulphate and carbonate, or phosphate of sodium, 1 or 2 drachms every morning. A mercurial purge once or twice a week should be given. Mercury is of great value ; the tension may nearly always be reduced by the administration of 1 or 2 grains of calomel, or pil. hydrarg. It may be combined with pulv. ipecac. (½ grain), rhubarb, or colocynth. Heat, either in the form of radiant heat, hot air baths, or hot water, dilates the cutaneous vessels, and thus removes the peripheral resistance sometimes in a very striking manner. Liquor trinitrini, the nitrites and other vaso-dilators temporarily reduce tension (Figs. 19, 20, and 26, p. 114). If the vessels are already the seat of sclerosis, the remedies advised should be employed (§ 67). Iodide of potassium (5 to 15 grains thrice daily) will always reduce high arterial tension. The high potential current has the same effect.

§ **62. Low Arterial Tension.**[1]—In low arterial tension the artery cannot be felt between the beats ; the pulse comes rapidly up to the

[1] " Low-tension pulse " is a term frequently used, but the same objection applies to it as to the term " high-tension pulse "—viz., that a wave cannot be said to have tension or pressure.

finger and rapidly declines, and is very easily obliterated. In *very* low tension a double wave is felt, the second one being small; this is known as the *dicrotic pulse*. A sphygmographic tracing shows an increase of the normal depression (aortic notch) before the dicrotic wave, and the dicrotic wave itself is more marked than in a healthy pulse (Figs. 27 and 28). The dicrotic wave may be as high as the percussion wave, and thus there may appear to be two beats for each cardiac pulsation. When the aortic notch falls below the level of the base line, the pulse is said to be *hyperdicrotic* (Figs. 26 and 30). *Symptoms* of depression, lassitude, prostration, and sometimes dyspepsia and sleeplessness, occur in association with low arterial tension. Purgatives cannot be well borne, and the patient may feel better when the bowels are constipated, because the retained fæces drive the blood out of the splanchnic area of veins, and so raise the general blood-pressure. Capillary pulsation is sometimes met with in low arterial tension,

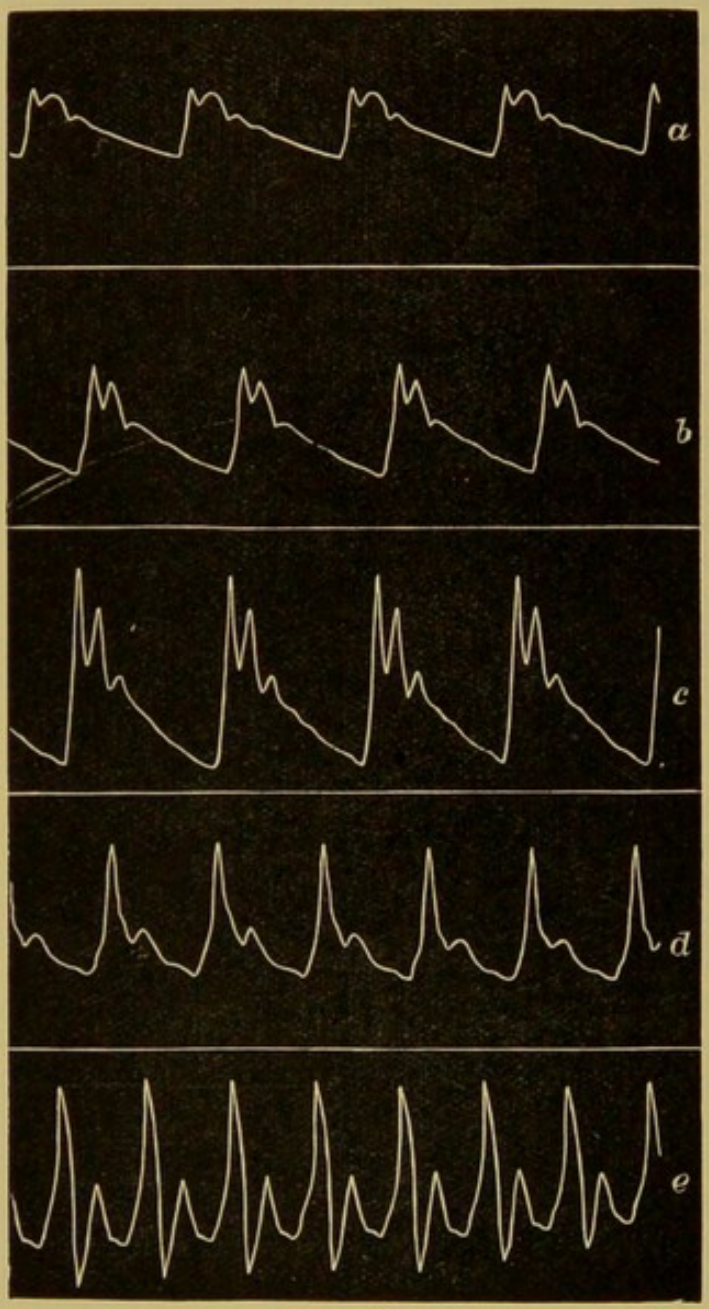

Fig. 26.—HIGH-TENSION PULSE REDUCED to a state of hyperdicrotism (converse of Fig. 30).—Series of tracings showing the efficiency of sodium nitrite in the reduction of high tension. Martin R., aged forty-five, chronic parenchymatous nephritis, under the care of Sir William Gairdner in the Western Infirmary, Glasgow, August, 1895. *a*, Typical high tension. Tracing *b* shows the reduction of tension after 15 grains sodium nitrite in four doses during the twenty-four hours. The remedy was continued, and tracings *c* and *d* on successive days show the gradual reduction of tension effected. They are normal, excepting for the exaggerated aortic wave. In the last tracing *e* a condition of HYPERDICROTISM is shown, the high tension having been entirely replaced by the opposite extreme. (Kindly supplied by Dr. W. S. Cook, who was the house-physician at the time.) Compare this with Fig. 30.

particularly with aortic regurgitation. By drawing a line along the forehead, or lightly pressing down the tip of the nail, the alternate blush and pallor due to the capillary pulsation is well brought out. In extreme states a pulse may even be communicated to the veins on the dorsum of the hand.

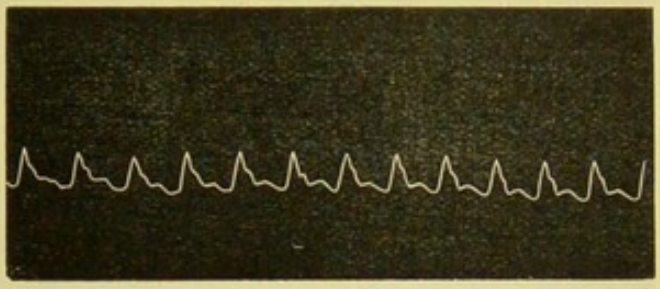

Fig. 27.—Low-Tension Pulse-Tracing, from a case of Intestinal Obstruction.

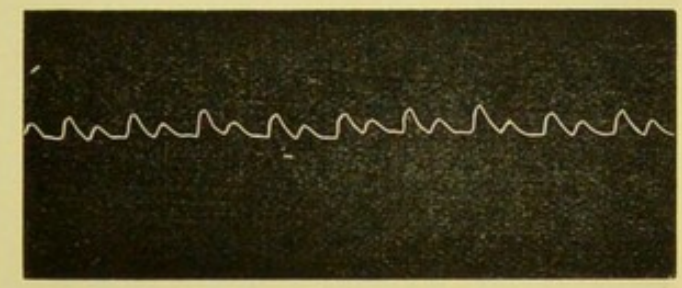

Fig. 28.—Low-Tension Pulse-Tracing, from a case of Enteric Fever, showing marked dicrotism. Patient aged thirty-seven.

Causes.—In *health* a persistent state of low tension is sometimes, though rarely, a hereditary condition. It may be found also after meals, a warm bath or moist warmth.

1. With *cardiac valvular disease*, in all phases of failing compensation, there is low tension. The pulse of *aortic regurgitation* is so characteristic that the lesion can be diagnosed by it. It comes suddenly up to the finger, and as suddenly collapses. This has received the name of "shotty," "slapping," "water-hammer" or "collapsing" pulse, the pulse of unfilled arteries, or *Corrigan's* pulse, after the name of the physician who first described it (Fig. 29). It is best brought out by feeling the radial while the patient's hand is held

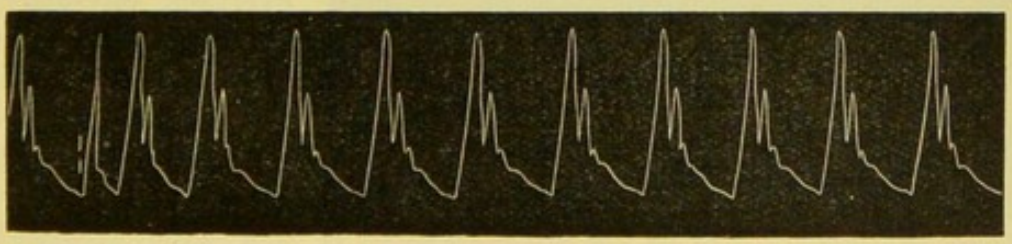

Fig. 29.—Water-Hammer Pulse.—Tracing (taken by Dr. Reginald E. Hanson) from a man, aged thirty-four, with typical aortic regurgitation, accompanied by very great cardiac hypertrophy (bovine heart). Well-marked capillary pulsation and retinal pulsation were present. Typical collapsing or water-hammer pulse. He had had anginoid attacks, which were relieved by sodium nitrite. At the time tracing was taken pulse was 81, respiration 22, and blood-pressure (taken by Hill and Barnard's instrument) 140.

up vertically on a level with the head. In this condition the radial pulse is distinctly postponed—that is to say, the pulse at the wrist occurs later than the apex-beat. Moreover, the artery in aortic regurgitation is large, in contradistinction to aortic stenosis, where the artery is small. The sphygmographic tracing of the "water-hammer" pulse shows a long percussion stroke, scarcely any tidal wave, with a sudden

down stroke, broken only by a small dicrotic wave (Fig. 29). In cases of a double aortic murmur, this pulse is of great diagnostic significance. If the pulse has a distinctive "water-hammer" character, the systolic murmur is due not to aortic stenosis, but simply to roughening of the valves or atheroma of the aortic wall.

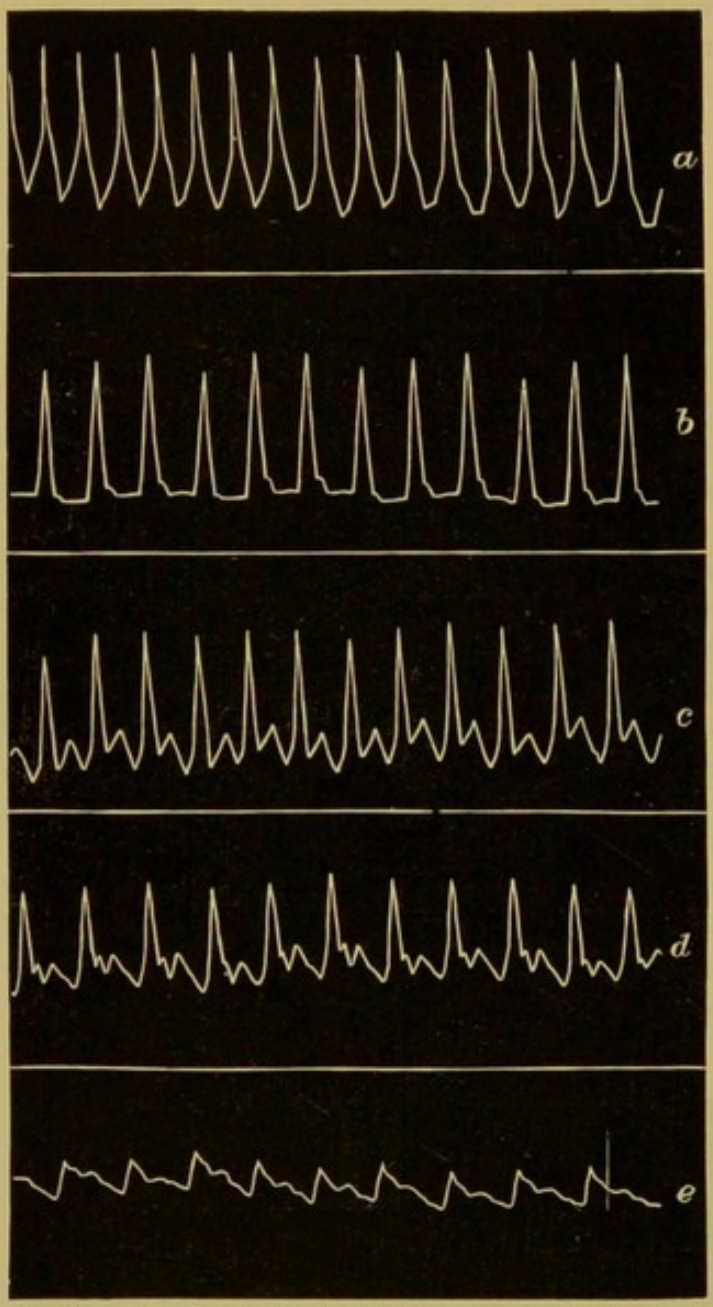

Fig. 30.—HYPERDICROTISM of low tension gradually RAISED TO NORMAL (converse of Fig. 26). The author is indebted to Dr. W. S. Cook for the notes of the following case, which was under Professor Gairdner's care in the Western Infirmary, Glasgow. The patient was attacked with ACUTE DOUBLE PNEUMONIA two days before admission. The dulness had a scattered character, which aroused a suspicion of tuberculosis, but examination of the sputum showed an absence of tubercle bacilli, and a great abundance of Fraenkel's pneumococcus. Great prostration and delirium were marked features in the case. The tracings show very well the hyperdicrotic—*i.e.*, low-tension—pulse at the commencement of the illness, and the gradual assumption of a normal tension in convalescence. The treatment was as follows: On June 5, 6 grains quinine every three hours; on the 6th, a mixture of vin. ipecac., sp. æth. nit., and liq. ammon. acet., with 2 grains quinine, every three hours; on the 7th, 1 drachm whisky every hour; on the 9th, ice-cloths to the abdomen, and paraldehyde at night; on the 11th the ice-cloths were discontinued.

2. In *weak heart*—*e.g.*, fatty heart, and general obesity—the pulse is of low tension, and in all advanced states of cardiac failure (§ 51).

3. Without organic heart disease low tension is met with in neurasthenia and in all *states of exhaustion* and debility, such as are caused by over-exertion, physical or mental, deficient and bad food, or anxiety—all conditions in which vaso-motor paresis might occur.

4. Low tension is also met with in all *asthenic varieties of fever*, especially enteric fever and peritonitis (see Figs. 28, 29, and 30).

Treatment depends upon the cause. In general terms, the food should be nourishing

and easily digestible. Change of climate is often beneficial. Iron, arsenic, quinine, strychnine, and digitalis are all useful; and so is alcohol, which should, however, only be given with meals and in small amounts. It is very useful in the aged. Fig. 30 shows the raising of low arterial tension under suitable treatment.

§ **63. The Pulse in Relation to Prognosis and Treatment of Disease.**—In acute febrile diseases a full bounding pulse is usual, and its absence warns us that we are in the presence of an asthenic and more dangerous type of case. It is in fevers also that the pulse is our chief guide as to whether alcohol should be administered or not. In enteric, for instance, if the pulse is weak and the vital powers flagging, stimulants are called for, but not otherwise. This was Murchison's rule.

In chronic affections the pulse is not so valuable an indication of the patient's general condition. Perhaps the most important fact to bear in mind is that in the aged, and, indeed, all persons past middle life, the pulse should be carefully watched from time to time, because a constant high tension is the main cause of arterial degeneration and many of the most lethal diseases to which persons in the latter half of life are liable. Constant high tension also tends to overstrain and degeneration of the heart.

The administration of digitalis and other cardiac remedies must be regulated by the condition of the pulse. Thus in cardiac valvular disease with slow full pulse digitalis should not be given, but only when the pulse is of low tension, *quick* or *irregular*. If, on the other hand, the pulse becomes irregular during the administration of digitalis, the drug should be at once withdrawn.

The supervention of acute inflammation within the abdomen—needing, perhaps, prompt surgical interference—is indicated by a rapid pulse, anything over one hundred is my own guide.

The student should learn by constant and repeated observation what important lessons can be learned from the pulse.

ARTERIAL DISEASE.

§ **64. Symptomatology.**—Among the symptoms to which arterial disease may give rise are giddiness or "dizziness," feelings of faintness, slight syncopal attacks, headache, paroxysmal dyspnœa, gangrene, or other conditions referable to the extremities (Chapter XVII.). But each of these symptoms, excepting the last named, may be caused by disease of some other physiological system. It is only when several of them are met with together, and an examination of the heart and arteries lends confirmation to the idea, that we are led to conclude that the vascular system is at fault. The importance of arterial disease depends more upon its effects on the heart, and indirectly on the other organs and tissues of the body, than upon the vascular condition *per se*.

§ **65. Physical Signs of Disease of the Arteries.**—The physical signs are very few in number, and consist simply of a visible or palpable thickening, dilatation, or tortuosity of the superficial vessels, such as the temporals, radials, brachials, and sometimes carotids. There are three features to note concerning the accessible arteries : (i.) The size of the artery should be observed as we compress or roll it beneath the fingers—a feature which sometimes requires considerable experience to recognise. (ii.) The thickness of its wall ; and it must not be forgotten that high arterial tension may produce the sensation of a thick wall, and *vice versa*. The thickness of the wall is best ascertained by stopping the pulse with the fingers of one hand, and rolling the empty tube beyond under the fingers of the other hand. (iii.) By passing the fingers up and down the length of the tube, the beadlike thickening due to atheroma may be detected.

The arteries are much more prone to disease than are the veins, which is in keeping with the greater liability of disease to attack the left than the right side of the heart—at least, during extra-uterine life. **The Chronic Diseases of the Arteries** which admit of clinical recognition are as follows :

I. Atheroma. II. Arterial Sclerosis (synonyms : Arterial Fibrosis, Arteriocapillary Fibrosis, etc.). III. Arterial Hypermyotrophy. IV. Functional Disease of the Arteries (see Diseases of the Extremities, Chapter XVII.). V. Aneurysmal Dilatation. VI. Chronic Endarteritis, due to syphilis and other causes, is only recognised by its pathological effects (cerebral softening, gangrene, etc., see Diseases of the Nervous System : anatomy). Acute Endarteritis is generally part of Acute Endocarditis (§ 39). VII. Embolism, or the blocking of an artery by an embolus, is the result usually of cardiac disease, especially infective endocarditis ; or it may be secondary to thrombosis. VIII. Thrombosis, or the coagulation of blood in a living vessel, is usually the result either of local disease involving the vessel, or of some blood change. Both this and Embolism are dealt with elsewhere. See, for example, Localised Dropsy, or Phlebitis (Diseases of the Extremities, Chapter XVII.).

§ **66. Atheroma.**—Atheroma has unfortunately come to be used in a somewhat vague sense, but it is taken here to mean a localised or *patchy* thickening of the tunica intima, occurring for the most part in patients past middle age, unaccompanied, as a rule, by any obvious symptoms during life. It starts as a localised hyperplasia in the deeper (external) layer of the tunica intima ; and the change may go on to fatty, caseous, and sometimes calcareous, degeneration. When it is advanced, the middle, and even the external, coats may be invaded. It is generally more or less widespread, but the disease nearly always commences and predominates in the larger vessels—*i.e.*, in the aorta and its branches. Consequently, if it be detected in the radial or temporal, the inference is that its distribution is extensive and that it involves the vessels of the brain also.[1]

Symptoms are generally altogether wanting.[2] However, when the disease

[1] Some observers say that, with the exception of the coronary arteries and the vessels of the brain, it does not usually affect the smaller arteries, but I have satisfied myself of its existence in the radials and nearly all the arteries of that size in the body in advanced cases of atheroma.

[2] This absence of symptoms has sometimes led me to conjecture whether atheroma might not be, in a sense, a conservative process, an idea which is further strengthened by two other important circumstances—viz. (1) That the change was most constantly found in those situations exposed to the shock of the systole,

involves the aorta (and it nearly always commences in that situation), it impairs the elasticity of that structure, and gives rise to an accentuated second sound of the heart in the aortic area. It also imparts to the pulse that feature called by Broadbent " virtual tension " (Figs. 31 and 19, B.), which is a very constant feature of the senile pulse (p. 110). In the more advanced cases atheroma may sometimes be detected as a nodular or beaded thickening in the radial, temporal, and other superficial arteries, which can be felt by moving the fingers up and down the artery. The patient is, however, unaware of its existence, and may live to old age, unless he be subject to high tension or other cardio-vascular disorder.

Consequences.—Histologically, the change is almost indistinguishable from the syphilitic endarteritis which occurs in younger persons, and it is only by the age of the patient and the history of syphilis that the latter can be differentiated. There is, however, a third important difference—viz., whereas syphilitic endarteritis commonly results in thrombosis, atheroma rarely does so, unless degenerative changes occur. Hæmorrhage is apt to occur in these cases, due not to the atheroma, but to thinning and dilatation of the vessel on the proximal or distal side. Occurring, as it does, only in old people, it is accompanied by, and possibly aids in the production of, debility and other signs of old age; but it is wonderful how extremely common widespread patchy atheroma is even in healthy old people who die of pneumonia or some other intercurrent malady, *e.g.*, the woman aged one hundred, I have mentioned in the Pathological Society's Transactions for 1904. See also footnote, p. *ante*.

No *treatment* will remove the atheromatous condition, but its presence is one of the indications for the avoidance of the causes of high arterial tension.

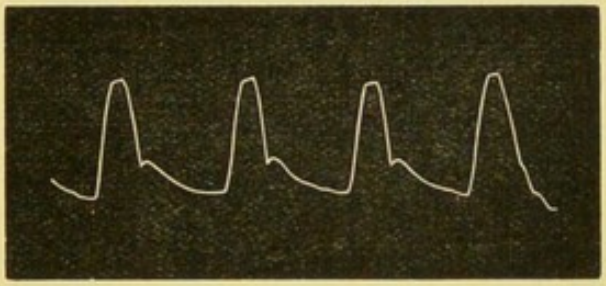

Fig. 31.—Typical Senile Pulse.—W. P., aged eighty-eight. Shows the flat top of *virtual tension*, which is the chief characteristic of the senile pulse.

§ 67. Arterial Sclerosis. — The term " arterial sclerosis " is here used in an anatomical sense, as a generic term to mean any widespread thickening and hardening of the arterial coats which leads to loss of elasticity and contractility of the arteries which is *clinically recognisable*. It is too often forgotten that what may be called the " parenchyma " of the arterial system—that is to say, its functionally active part—is the middle or muscular coat. Upon the proper functioning of this coat depends the whole of the regulator mechanism of the arterial system. My experience at the Paddington Infirmary, which offered a very extensive field for researches into the pathology of the vascular system, went to show that, although very wide changes might exist in the tunica intima or tunica adventitia without symptoms, very slight changes in the media were nearly always attended by some kind of symptom or effect during life. Histologically, therefore, I differentiate intimal, adventitial, and medial sclerosis—the last named being by far the most important.[1] The subject of arterial disease is surrounded with considerable obscurity, partly because the same words are used by different observers in very different senses, and partly on account of the difficulty of making a sufficiently

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as, for instance, on the upper aspect of the arch of the aorta and in the angle of the bifurcation of arteries. (2) It was a constant—and therefore, one might say, a physiological—change in greater or less degree in *all* elderly people dying in the Infirmary, no matter what might be the cause of death. One of the very notable cases was that of a woman, aged one hundred, who died of pneumonia, and who had not at any time presented any cardio-vascular symptoms during life.

[1] " On Arterial Sclerosis " (Transactions of the Pathological Society of London, 1904).

exhaustive examination of the entire arterial system in any given case. A dozen sections of the liver, for instance, will give a fair idea of a morbid change in that organ; but to form an accurate conception of the structural changes in the arterial system in any given case, it may be necessary to carefully examine one or two hundred sections, taken from many different vessels, and to make very accurate *measurements* of these, both naked eye and microscopic.

Symptoms.—The clinical course of the disease may be divided into two stages: (*a*) That in which the ventricular hypertrophy is sufficient to compensate for the increased peripheral resistance; and (*b*) that in which the left ventricle begins to fail—that is, to yield and dilate.

(*a*) In the first stage (1) the patient may be free for many years from any symptoms referable to the vascular system, so long as the increased peripheral resistance is adequately and not excessively compensated for by the ventricular hypertrophy. Sometimes the patient may come to us for loss of vigour or breathlessness; but more often the thickened vessels are discovered, so to speak, by accident, when the patient comes under our notice for some other malady. On examination, however, we may find that the arteries are visibly and palpably thickened at the wrist and on the temples, being cord-like, and sometimes elongated and tortuous. The feel of the vessel much resembles that of high arterial tension (a condition, by the way, which may coexist in the earlier stages), but by compressing the pulse above and so emptying the vessel, and then feeling the artery beyond, the thickening of the wall may be readily revealed. By sliding the fingers up and down, it may be distinguished from the beading of atheroma. (2) As the disorder progresses, the patient loses his former vigour, mental and physical. He is, in a word, "old before his time," for it has been truly said a

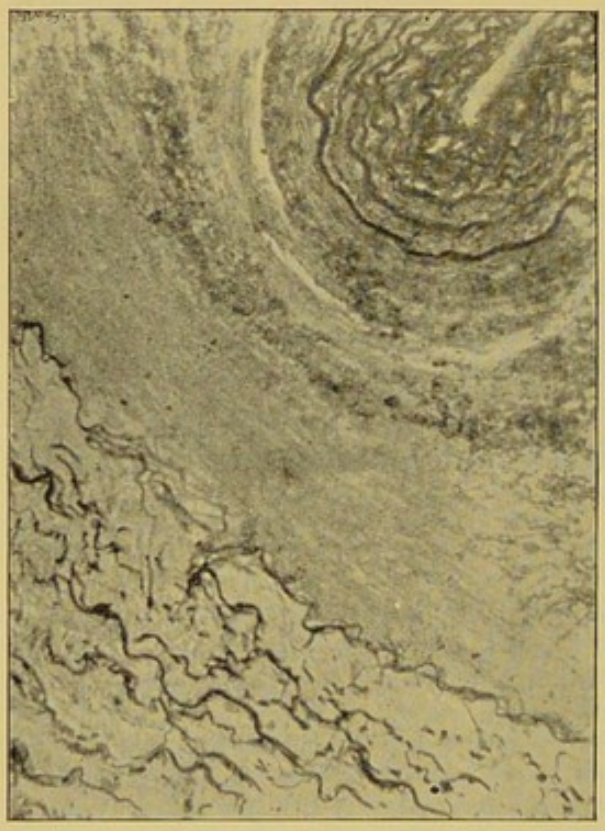

Fig. 32.—Arterial Sclerosis.—Section of a radial artery magnified about 400 diameters, stained with acid orcein to show granular degeneration of muscle fibres in the middle coat, which commences and *predominates in the internal layers of middle coat*. This method of preparation brings out the elastic tissue, as may be seen in tunica adventitia and tunica intima. Similar sections stained sufficiently long in logwood brought out the rod-shaped nuclei of the muscle fibres, showing that the change is not a fibrosis, as Gull and Sutton maintained. The appearances described by them as arterio-capillary fibrosis can always be produced by prolonged hardening coupled with insufficient staining with logwood.

man is " as old as his arteries." This loss of vigour is no doubt due to the fact that all the organs and tissues are deprived of that regulation of nutrition which depends upon the elasticity and healthy contractility and relaxation of the muscular tissue of the arteries. (3) Breathlessness, and a tendency to rapidity of pulse after slight, or even without, exertion are, in my experience, the next most constant features. Sometimes the dyspnœa is paroxysmal, and the case resembles asthma. The heart is irritable, and this is evidenced clinically by breathlessness and a pulse which easily becomes rapid and irregular.

(*b*) When the second stage is reached, symptoms arise which, as a whole, form a distinct and well-marked clinical picture. They are due partly to failure of the arterial functions, but mainly to the failure of cardiac compensation. In addition to the preceding, which become emphasised, the symptoms are as follows : (1) Vertigo may have occurred before the second stage is reached, but the attacks now become more frequent. Indeed, about nine-tenths of the cases of senile vertigo met with in the infirmary could be traced to this malady. It is not, however, a true vertigo, but rather a sensation of " swimming in the head " or " dizziness," and the patient feels either as if he were " going to fall " or " going to faint." So frequently did these attacks occur among the aged and so-called " healthy " old people in the workhouse that they used to pay but little attention to their frequent falls due to this cause. (2) Actual fainting attacks are met with less frequently, but when present they are of more serious import. They vary from a slight interruption of the continuity of thought on the one hand to a prolonged faint or epileptiform seizure on the other. (3) Physical signs pointing to a varying degree of hypertrophy and dilatation of the heart are revealed on examination, together with accentuation of the second sound over the aortic cartilage, if high tension be present. (4) Anginoid attacks, or true angina pectoris, is another symptom. Quite three-fourths of the cases of angina pectoris met with in the infirmary were attended by this condition. (5) In extreme states of the disease, especially when accompanied by atheroma or endarteritis, there may be gangrene of the extremities, cerebral softening, either localised or diffuse, and similar changes in other viscera. Miliary aneurysms may arise in the periphery of the arteries of the brain, and hæmorrhage in this situation is one of the most frequent consequences. There is always in these cases a tendency to the development of low forms of inflammation, especially " senile pneumonia." (6) Renal fibrosis (chronic Bright's disease)—*i.e.*, granular or gouty kidney—is very frequently, but not necessarily, associated with arterial sclerosis. Some hold that chronic Bright's disease is essentially a generalised arterio-renal disease, and that, although arterial thickening may occur without renal disease, the latter is always attended by more or less arterial change.

In the *Etiology* (1) heredity is certainly one of the most important factors, and families are found in which every member shows a tendency to this disease on reaching a certain age. (2) As regards age, the disease is, with rare exceptions, only met with in the latter half of life ;[1] and it is rather more frequent among men. (3) The pathology of the complaint is not yet worked out, but in many cases careful inquiry will reveal one or more of the causes of a constant high tension (*q.v.*). Various toxic blood states possibly act in this way, and there is often a history of alochol, lead, or gout. (4) Overwork and physical strain, by leading to an overfilling of the arteries, are also possible causes, on the principle that increased function causes first increase of structure, and, later, degeneration of the muscular coat.

Diagnosis.—Arterial sclerosis may have to be diagnosed from (1) high arterial tension, by stopping the blood current and examining the artery beyond ; (2) atheroma, which gives to the vessel an unequal or beaded character. (3) Granular kidney, in its slow, insidious onset and vague symptoms, closely resembles arterial sclerosis in its clinical history, and can only be distinguished from it by the presence of urinary changes. The arterial and the renal changes are frequently associated. (4) Other causes of progressive debility (Chapter XVI.) may have to be distinguished from arterial sclerosis.

Prognosis.—In the first stage, though nothing can be done to abolish the thickening of the arterial walls, much may be done to prevent its advance, and if the patient escape pneumonia and other inflammatory conditions to which he is liable, he may live many years. The whole question of prognosis turns very largely on the state of the heart. If the breathlessness is considerable, and the physical signs show marked cardiac dilatation, and the pulse is irregular and rapid, the patient is not likely to live more than a year or two. If, on the other hand, cardiac compensation is good and the patient feels but little distress on movement, then the outlook is not unfavourable.

Treatment.—The indications for treatment are (*a*) to keep down the blood pressure ; (*b*) to aid the heart ; and (*c*) to avoid any extra strain being thrown upon the heart or vessels. (1) Hygienic measures are of great value, and the patient's duration of life will depend upon the kind of existence he can afford to live. He should live a very regular life, free from any strain on mind or body. The diet should be strictly moderate, especially as regards proteids, and should be readily digestible. Unless heart failure is very pronounced, alcohol should be avoided. The calcium salts in the blood are said to be eliminated by sodium bicarbonate (2½ drachms) neutralised with lactic acid, 6 ounces of water are added to this, and it is taken in twenty-four hours. At any

[1] Syphilitic arterial disease is a patchy endarteritis which occurs in younger subjects, usually more or less localised, and therefore does not come within the scope of our opening definition.

rate, this relieves many of the symptoms of cardiac dilatation. (2) The heart needs our special attention, and cardiac tonics and other remedies may be administered on general lines (see Cardiac Failure, § 51). The question of Schott baths and passive exercises is a most important one, and while some advocate them strongly, others say that they are attended with considerable danger by increasing the tendency to hæmorrhage and the other consequences above referred to ; but, in my belief, if the arterial disease is not very advanced, the heart undoubtedly derives considerable benefit by this treatment.

§ 68. Arterial Hypermyotrophy is a term which has been employed by the author to imply a generalised increase in the muscular tissue of the arteries. In a paper read before the British Medical Association at Bournemouth, in 1891,[1] based upon a collection of cases which were observed in the Paddington Infirmary, it was shown that a generalised increase in the muscular tissue of the arteries occurred as a distinct clinical and pathological entity, consequent, in all probability, on states of prolonged or frequently recurring high tension, by the simple physiological law that increased function leads to increased structure. In 1895 Drs. Dickinson and Rolleston [2] showed that a widespread increase of the muscular tissue of the arteries occurs throughout the body in some cases of renal disease. Sir Clifford Allbutt [3] has described, under the name Hyperpiesis (constant high blood-pressure and its attendant symptoms), a condition which, from a clinical standpoint (for none of the cases were confirmed by autopsy), probably corresponds with the condition which the writer, from an anatomo-clinical standpoint, has called "arterial hypermyotrophy."

The change itself consists of a hypertrophy of the middle or muscular coat of the arterial wall. It affects principally the medium and small-sized arteries of the body—those which normally contain more of this tissue than is found in the larger vessels. Patients may exhibit no symptoms, and rarely die in the early stages of the disease or until some granular or other degeneration has taken place in the hypertrophied tunica media. The condition, however, is by no means an infrequent one, to judge from the records of the Paddington Infirmary, and if it were not such a laborious task to examine the arteries of the body, more cases would doubtless be revealed.[4]

Symptoms.—(1) The arteries have a thickened but elastic feel, although they may be of normal size. In a few cases the author has been able to confirm this by means of Oliver's arteriometer. The prolonged first stage is always accompanied sooner or later by cardiac hypertrophy. It may exist unknown to the patient for

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[1] Cases of arterial hypermyotrophy and the resulting degenerations in the muscular tissue were published in the *British Medical Journal*, January 23, 1897, and the Transactions of the Pathological Society of London, 1904.

[2] See the *Lancet*, 1895, vol. ii., p. 137.

[3] The Lane Lectures, *Philadelphia Medical Journal*, April, 1900, pp. 400-500 ; and elsewhere (*e.g.*, "The Hunterian Oration," c. 1885).

[4] It has been said in criticism of these observations that the change consists of a swelling of the individual muscular fibres rather than a numerical increase. This is difficult to refute, because the opportunity does not often occur of examining the arteries before the granular swelling—which is also a consequence of the same cause (high tension)—has also occurred. But, in the first place, I would point to the actual occurrence of true hypertrophy in renal cases, as shown by Rolleston and Dickinson. Secondly, I have occasionally been fortunate enough to secure cases—dying by accident, for example—which undoubtedly exhibited a true hypertrophy without degeneration. Thirdly, a very careful examination of several of my cases shows that there was an actual increase in the unstriped fibres, in addition to their degeneration ; and, fourthly, Sir Clifford Allbutt's *clinical* observations undoubtedly lend confirmation to the existence of such a pathological condition as arterial hypermyotrophy.

many years, and be overlooked by the doctor, or, like arterial sclerosis, discovered accidentally. Sooner or later, however, one or more of these symptoms arise, viz., (2) *postural vertigo* from loss of arterial adaptability to posture; (3) dyspnœa (sometimes of an asthmatic or paroxysmal character); (4) persistent or recurrent headache; and (5) symptoms of high arterial tension. (6) In the second stage of the disease, when granular degeneration and consequent rigidity are present, the symptoms are indistinguishable from those of arterial sclerosis, which, in point of fact, supervenes. It is more often found in persons over forty.

Effects.—The results of the thickening in the *first* stage are (i.) a diminution of the lumen of the vessels by reason of the tonic spasm; (ii.) a more or less permanent increase of arterial tension (blood-pressure); (iii.) *pari passu* with the arterial thickening and high tension there is hypertrophy of the left ventricle. As the result of the insufficient or ill-regulated blood-supply, the tissues are insufficiently nourished, and tend to degenerate, and are more readily prone to inflammation and disease. The patient loses his mental and bodily vigour. In the *second* stage cardiac compensation fails, and the middle coat of the arteries degenerates—the consequences of which are identical with arterial sclerosis above described. Arterial hypermyotrophy is no doubt often associated with granular kidney, probably in about half the cases. But from the cases which the author has collected, it is evident that arterial hypermyotrophy may occur quite independently of renal disease of any kind.

The *Prognosis* of the condition in its early stage is favourable, if the patient can live a careful life, although its existence adds to the gravity of intercurrent diseases.

The *Treatment* in the early stage is that of high arterial tension (§ 61); in the later stages that of arterial sclerosis (§ 67). Symptomatic treatment is always useful. For the breathlessness, nux vomica and digitalis, and aperients, especially 1 or 2 grains of calomel, are at all times useful. For the attacks, especially the vertiginous attacks, nothing gives so much relief as nitroglycerine, and for the severe ones occasional inhalations of amyl nitrite. This is an undoubted fact, and constitutes one of the reasons in support of the theory that these seizures are of entirely circulatory origin. It also supports the idea that it is muscular spasm, and not the degenerated arterial wall, which produces the vertigo. For the fainting attacks, alcohol is indicated in small doses, but I have found that alcohol in anything but very small quantities aggravates the symptoms and consequences of the disorder.

§ 69. Functional Diseases of the Arteries.—Of functional diseases or vasomotor derangements we know but little, although several very important maladies are attributed to this cause—*e.g.*, Raynaud's disease and migraine. Functional derangement of the arteries is also manifested by a large number of symptoms, many of which are vague and evident only to the patient. On this account they are apt to be regarded by medical men as unimportant, and it is true that they are not serious in the sense of being lethal; but to the patient they are often extremely disagreeable, irksome, and often terrifying. Of such we may mention alternate flushing and pallor ("flush-storms"), dead hands, cold hands and feet, chilblains, various other erythematous conditions, blue nose, palpitation, tachycardia (§ 40), paroxysms of copious urination, acroparæsthesia, erythromelalgia, feelings of suffocation, pseudo and true angina pectoris, feelings of tingling, itching, throbbing, and actual swelling of the limbs. A case of the last-named is referred to under the heading Œdema (§ 425).

Aneurysmal Dilatation of the Arteries belongs to surgery, excepting aneurysm of the thoracic aorta (see § 53), the abdominal aorta (§ 188), and the cerebral arteries (Chapter XIX.). **Embolism** and **Thrombosis** are referred to under Diseases of the Extremities (Chapter XVII.) and the Brain (Chapter XIX.).

CHAPTER VI

THE LUNGS AND PLEURÆ

Owing to the extreme vascularity of the lungs, it is not surprising that inflammation of these organs is a frequent complication of acute general or blood diseases. Thus, inflammation of the lungs (pneumonia) is one of the commonest accompaniments of the acute specific fevers, pyæmia, and other microbic disorders. Again, in the generalised blood infection, which arises from a local tuberculous focus, the lungs are, as we should expect, very frequently the seat of tuberculous lesions, and there are three *acute* forms of tuberculosis in which the lungs are more or less involved—namely, a tuberculous form of Acute Pneumonia (§ 87*a*), Acute Miliary (generalised) Tuberculosis (§ 381*a*), and Acute Pulmonary Tuberculosis (§ 83). Chronic Pulmonary Tuberculosis (phthisis) is an instance in which the microbic infection is (mostly) inhaled; and we meet with the same mode of aërial infection in Acute Lobar Pneumonia. But Pulmonary Tuberculosis is now also proved to result by infection from the intestine when tuberculous milk is ingested. In short, whatever infects the blood is apt to infect the lungs.

PART A. SYMPTOMATOLOGY.

The Cardinal Symptoms of diseases of the lungs are **cough, breathlessness, expectoration,** and sometimes **pain in the chest** and **hæmoptysis.** The more general symptoms are pyrexia, emaciation, and debility. The heart, more especially the right side, suffers sooner or later in all serious or prolonged pulmonary diseases by interference with the pulmonary circulation.

§ **70.** Concerning **Cough,** if it is attended by expectoration (as in 1 to 4 below), it points to definite changes either in the lungs or throat. If without expectoration (as in 5 to 8 below), it may point to simple congestion of the throat or larynx, to the presence of pleurisy, to the early stage of some pulmonary disorder, or to some source of reflex irritation. The *Causes of Cough* are as follows:

1. The commonest form of cough is that recurring wheezy cough, attended by expectoration, so typical of bronchitis.

2. Paroxysms of coughing followed by vomiting occur in *whooping-*

cough and advanced *phthisis*. *Bronchiectasis* is attended by paroxysmal cough with fœtid expectoration at intervals. Paroxysmal cough, with or without expectoration, occurs with enlarged bronchial glands and other *mediastinal tumours*.

3. The HAWKING cough of throat affections is very characteristic, and is met with in catarrhal *pharyngitis*. It also occurs in *nervous* and hysterical subjects. It is also associated with digestive disorders (Chapter X.), where there is often a collection of mucus in the pharynx, and with chronic liver disease.

4. The IRRITABLE cough, most marked in the early morning and on going to bed, is especially associated with *early phthisis*. There may or may not be much expectoration.

5. A NIGHT cough may be due to a *long uvula*. When a patient complains that a cough is worse at night or on lying down, the uvula should be carefully examined. A slight degree of congestion will cause considerable elongation of the uvula, so that it will irritate the back of the pharynx when the recumbent posture is assumed. A night cough is also associated sometimes with *threadworms* in children.

6. The long BARKING or nervous cough of *hysteria* is very characteristic. It is unattended with expectoration.

7. The SHORT SUPPRESSED cough associated with *pleurisy* or *pleuro-pneumonia* is so characteristic as to be diagnostic ; in the former it is unattended by expectoration.

8. The GANDER or BRASSY cough associated with aneurysm and other mediastinal tumours is typical, and when once heard is readily recognised.

9. The REFLEX cough, due to irritation in the area of the pnenmogastric, may be caused by (i.) *gastro-intestinal* disorders, such as dyspepsia, constipation, diarrhœa, or worms in children ; (ii.) *pericarditis ;* (iii.) *carious teeth ;* and (iv.) *ear* troubles.

The *Diagnosis* of these varieties of cough is somewhat important in practice, since they arise from, and may be met with, affections other than those of the lungs. When a short dry cough is set up by going into the cold, it may be due to pharyngeal congestion or irritation. In simple throat affections the cough comes on in paroxysms, especially after talking. On the other hand, if such a cough comes on in a warm atmosphere, we should suspect phthisis.

The *Treatment* of cough depends upon the cause, but, in general terms, irritable coughs may be soothed by bromides, minute doses of opium, by a linctus of squills and tolu, or by various medicated lozenges.

§ **71. Breathlessness,** or dyspnœa, is another symptom of lung affections. The causes of breathlessness are dealt with in more detail in the symptomatology of cardiac disorders (§ 20). The types of breathlessness special to respiratory disorders are :

1. Breathlessness attended by SNIFFING and NASAL BUBBLING is caused by *nasal* or *naso-pharyngeal catarrh*. The obstruction in the nose or mouth usual in such a condition may also cause considerable stertor at night-time.

2. STRIDULOUS respiration, in which the stridor attends both inspiration and expiration, is caused by obstruction in, or *pressure* upon, the trachea or larynx. It is accompanied in severe cases by drawing in of the epigastrium and lower costal cartilages during inspiration.

3. Dyspnœa attended by considerable WHEEZING or rhonchi in the chest is very characteristic of *bronchitis*, attended usually by emphysema.

4. The LABOURED respiration which attends other *gross diseases* of the lungs is different from any of the foregoing. Under this heading also comes the expiratory dyspnœa of *emphysema*, which is due to the fixation of the chest in a position of inspiration. Undoubtedly the commonest lung condition giving rise to dyspnœa is emphysema, which is revealed by a barrel-shaped chest and hyper-resonance.

5. PAROXYSMAL dyspnœa is sometimes present in asthmatic attacks, but is more often an indication of *cardiac disorder*, in which circumstances it is often called " cardiac asthma " (§ 20*a*).

§ 72. **Pain in the Chest** is always present with affections of the pleura, but otherwise it is not a constant symptom in pulmonary disorders. The various causes of pain in the chest are enumerated in § 23. The following are the chief types of pain met with in diseases of the lungs :

(i.) The SHARP, cutting, stitch-like pain of *pleurisy*, before the effusion separates the inflamed surfaces, is greatly aggravated by drawing a long breath. This is undoubtedly the commonest of the pulmonary causes of pain in the chest, and this symptom in *pneumonia* indicates involvement of the pleura. It must be remembered, however, that in some *sub-diaphragmatic diseases—e.g.*, of the liver, spleen, or colon—pain is also felt on deep inspiration. One of the most intense forms of pain in the chest is due to *diaphragmatic pleurisy*. It is referred along the lower costal margin, and is accompanied by very shallow respirations, which are chiefly or entirely thoracic. Hiccough is occasionally associated. (ii.) A SORENESS behind the upper part of the sternum attends the onset of *acute bronchitis*. (iii.) SUDDEN severe pain, followed by considerable pulmonary and general distress, occurs with the onset of *pneumothorax*. (iv.) SUDDEN pain, attended by hæmoptysis, marks the occurrence of *embolism* of the lung. (v.) *Cancer* of the lung may or may not be accompanied by pain, according to its proximity to the pleura or other sensitive structures. (vi.) All *mediastinal tumours* give rise sooner or later to pain in the chest.

§ **73. Hæmoptysis** means the spitting of blood (*αἷμα*, blood ; *πτύω*, to spit), but the term is confined to the expectoration of blood from the organs of respiration.

The *fallacies* with regard to this symptom are very important, and it is sometimes as difficult as it is important to decide whether the blood comes from the throat or nose, from the stomach, or from the lungs. The differentiation is given more fully under Hæmatemesis (§ 192), but it may be mentioned here that blood coming from the lungs is thus

characterised : (i.) It is preceded and accompanied by a tickling cough (if the blood be large in quantity it may excite retching on touching the pharynx) ; (ii.) the patient usually goes on *coughing* up a little blood for some time afterwards ; (iii.) the blood has a bright crimson colour, is alkaline, and aërated (if very profuse, it may be dark in colour and un-aërated) ; (iv.) physical signs of disease of the lungs are usually, though not always, present—they may be absent in the early hæmoptysis of phthisis ; (v.) the antecedent history of the patient may point to pulmonary tuberculosis or to cardiac disease, these being undoubtedly the most common causes of hæmoptysis.

Causes.—For practical purposes the causes of hæmoptysis may be divided into two groups :

(*a*) Those which produce slight and sometimes protracted or recurrent bleeding ; and (*b*) those which produce a copious bleeding at one time.

(*a*) *Causes of Slight and Sometimes Protracted Hæmoptysis.*—I. Phthisis is by far the commonest cause. The hæmoptysis of phthisis may occur either in the early or in the advanced stage of the disease, and in *either* case it may be small or very large in amount. The presence of this cause may be recognised (i.) by the previous and family history of the patient ; and (ii.) by evidences of congestion, consolidation or cavitation of the lung (§ 77). Nevertheless, as just mentioned, the most careful examination may fail to reveal any signs, because hæmoptysis is frequently the earliest symptom of invasion by the tubercle bacillus.

II. Cardiac Disease, especially mitral stenosis or mitral regurgitation, is the next most common cause of hæmoptysis. It may arise in such cases either from congestion, or, more rarely, embolism of the lungs. In both cases evidences of cardiac disease are present. *Congestion* of the lungs is recognised by slightly impaired resonance and the presence of râles at the bases of the lungs.

III. Various Pulmonary Diseases other than phthisis may be attended by slight hæmoptysis. Thus, in *acute bronchitis* the sputum may contain streaks of blood from time to time ; and in *pneumonia* the sputum is rust-coloured about the third or fourth day of the illness. The hæmoptysis due to *carcinoma* of the lung may be recognised by the irregularity of the physical signs.

IV. Ulceration of any part of the respiratory passages may give rise to hæmoptysis, small in amount, and apt to be recurrent. A careful examination of the throat and larynx generally reveals this cause.

V. Vicarious Menstruation as a cause of hæmoptysis is disputed by some. It is recognised by the age and sex of the patient, by its occurrence at the time when menstruation is due, the normal menstrual function being absent, and by the absence of signs of disease in the lungs.

VI. Constitutional or idiopathic causes. Finally, there are certain patients in whom slight hæmoptysis occurs from time to time, the history and examination revealing nothing, and the patient living often to a good old age. The hæmoptysis

in such cases is explicable by two hypotheses—the presence of undiscoverable tuberculosis, or a transient congestion due to some constitutional cause. Thus, Sir Andrew Clark found hæmoptysis was of fairly frequent occurrence in subjects of the arthritic diathesis.

§ 73*a*. **Pulmonary Embolism** (with which may be considered pulmonary thrombosis) occurs clinically under two conditions: (*a*) primary, as in pregnancy or other circumstances attended by a hyperinotic blood change, and also after operations attended by considerable loss of blood. (*b*) Complicating some pre-existing disease, such as mitral disease (especially stenosis), or ulcerative endocarditis, or venous thrombosis (especially if septic). The *symptoms* are: (i.) Slight hæmoptysis, continuous for a day or two; (2) sudden moderate pyrexia; (3) sudden dyspnœa; (4) sudden pain in the chest; (5) in some cases a pneumonic patch can be made out beneath the site of pain, in others a pleuritic rub is audible. The condition is not usually serious, and the prognosis is that of the primary condition; but when it occurs three or four weeks after delivery, it is often suddenly fatal, the patient sitting up suddenly, gasping for breath, and dying forthwith.

The *treatment* of the primary group is that of stimulation, by administration of cardiac and general stimulants, such as digitalis, strychnine, and caffeine; that of the secondary form resolves itself into the treatment of the cause.

(*b*) *Causes of Hæmoptysis in which there is a Considerable Quantity of Blood at One Time*—I. Phthisis.—Copious bleeding (which may be continuous, perhaps for hours or for a day or two), without ending fatally, is almost invariably due to pulmonary tuberculosis. The chief features by which it is recognised are given above.

II. Rupture of an Aneurysm into the trachea or bronchus is a by no means rare accident in the history of that malady. It is the one cause of hæmoptysis which is usually followed by immediate death, though in some cases there may be a considerable leakage going on for a day or two before the final issue (§ 53).

III. Ulceration of the larynx, throat, etc., though usually causing small and recurrent hæmorrhages, occasionally leads to a large amount of hæmorrhage.

IV. Purpura, hæmophilia, scurvy, leucocythæmia, and some other Blood Conditions may be attended by bleeding from the lungs. These causes are for the most part rare, but when present are readily recognised.

Differentiation.—In order to arrive at a diagnosis of the cause of hæmoptysis in any given case, we must *first* of all examine the chest (lungs and heart) very thoroughly; *secondly*, use the laryngoscope to investigate the larynx and naso-pharyngeal passages; and, *thirdly*, we must inquire into the patient's history.

The *Prognosis* depends, of course, upon the cause. Hæmoptysis is nearly always a serious symptom, and when profuse is followed by considerable debility. In this way it may hasten the end of an advanced case of phthisis. But the hæmoptysis of early phthisis, though indicating definite involvement of the lung tissue, is not so serious, and with proper precautions the patient may completely recover and live to old age.

Treatment (*refer to Causes*).—(*a*) For *profuse hæmorrhage* immediate

treatment is necessary. The patient must be kept at absolute rest in bed. Ice is usually applied to the chest, but it should not be kept on in one place longer than twenty minutes at a time. The nourishment allowed must be cold. A hypodermic injection of morphine, or full doses of opium with acid. sulph. dil., or turpentine internally, are the most efficacious remedial drugs for early administration. Ac. sulph. dil. (♏ x.) with alum (gr. v.) may then be given every twenty minutes. A large dose of ergotin succeeds in some cases. Whitla recommends turpentine vapour in the room. Other drugs recommended are amyl nitrite, which dilates the arteries and so relieves the veins and capillaries, gallic acid, hazeline, or atropin or digitalis hypodermically. Full doses of ipecacuanha or other nauseants have also been recommended.

(*b*) When hæmoptysis occurs in *small quantity*, calcium chloride (gr. xx. every four hours) renders the blood more coagulable, and is specially useful. The hæmorrhage of congestion due to cardiac disease should not be checked, unless it becomes excessive, as it relieves the pulmonary congestion. When hæmoptysis occurs in elderly arthritics, give a sedative cough mixture, saline purgatives, iodides, and cod-liver-oil.

PART B. PHYSICAL EXAMINATION.

The physical examination of the lungs is carried out by means of Inspection and Mensuration, Percussion, Palpation, and Auscultation.

§ **74. Inspection and Mensuration.**—The inspection of the chest must be carried out in a good light, and the patient must be instructed to stand or sit erect and to breathe deeply. After noting the movements from the front, examine the back, then look from behind over the clavicles in order to make out the slighter distortions or inequalities of the chest. By inspection and mensuration we note (1) the rate and character of the breathing; (2) the shape and size of the chest; (3) the chest capacity. The chief landmarks of the chest are mentioned in § 30, and the regions into which, for descriptive purposes, it is divided are given in Fig. 33.

1. *Rate and Character of the Breathing.*—The rate varies normally from 15 to 20 per minute, or one-fourth the rate of the pulse; and any change in this proportion, or pulse-respiration ratio, should be observed. Notice whether the breathing is rapid, slow, shallow, or irregular. The respiration should be counted without the patient's knowledge, and it is a good plan to feel the radial artery while counting the breathing, as if you were examining the pulse. Both sides should move equally. Flattening or immobility of any part of the chest points to disuse of that part of the lung—*e.g.*, from consolidation. Flattening or protrusion of the interspaces indicates fluid. Drawing in of the interspaces during inspiration is indicative of some interference with the

free entry of air into the lungs (inspiratory dyspnœa), as in laryngeal diphtheria. *Cheyne-Stokes* breathing is a peculiar rhythmical irregularity of breathing (see § 20*b*).

2. *The Shape and Size of the Chest.*—A cross-section of the *healthy* adult chest gives almost the form of an ellipse, the longer diameter being from side to side. In the child it is more circular in shape. The chest should appear symmetrical, although in reality the right side is slightly larger than the left. There should be no marked hollowing anywhere; the clavicle should form only a moderate prominence between the supra- and infra-clavicular regions. The circumference of the chest

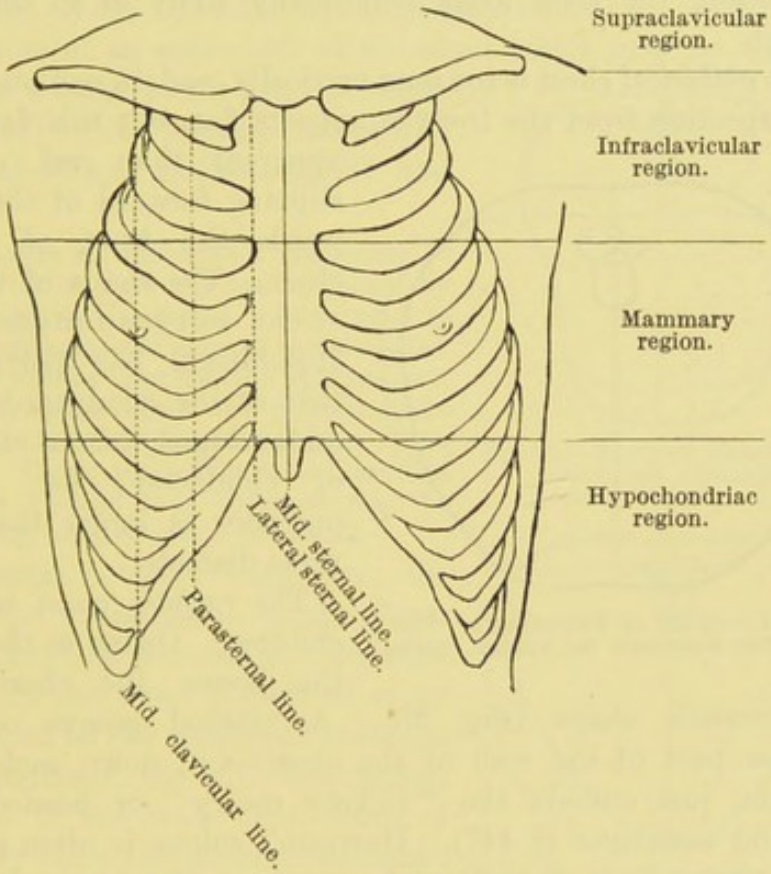

Fig. 33.—Anterior Thoracic Regions.

varies with the height of the individual, but it should average for a man 5 feet 6 inches about 34 to 35 inches. With deep inspiration it should expand about 1½ to 2 inches. The measurement at the level of the nipples in the male is a rough measure of the individual chest capacity (see above). The relative shape and capacity of the two sides is measured by a cyrtometer (see also p. 133). The principal abnormalities in shape are the emphysematous, phthisical, and rachitic chests.

The commonest form of abnormality in the shape of the adult chest is the *emphysematous*, or, as it is called, the barrel-shaped chest. Briefly expressed, this alteration consists of the fixation of the chest in

a position of permanent inspiration, and expiration cannot be completely performed. The sternum becomes curved, the lower part being unduly drawn in ; and a horizontal section shows the chest to be unduly circular (Fig. 34). When the hands are placed flat upon the chest on each side, they readily appreciate the fact that in advanced cases there is elevation, but no lateral expansion of the thorax, during inspiration. The upper ribs are crowded together, whilst the lower ribs are farther apart than normal, and the epigastric angle is very wide. Owing to the permanent elevation of the clavicles and upper part of the chest, and the unusual degree of development of the accessory muscles of inspiration, the neck looks abnormally short in an emphysematous subject.

The *phthisical* chest is too long vertically, and, in section, too rounded. On inspection from the front it appears flat, but this flatness is more apparent than real, owing to the slipping forward of the scapulæ towards the front of the rounded chest. The shape of the phthisical chest corresponds, as Woods Hutchinson has pointed out,[1] to that of the quadrupeds, and to the undeveloped human chest, or chest of childhood ; *i.e.*, the antero-posterior is larger than the transverse diameter.

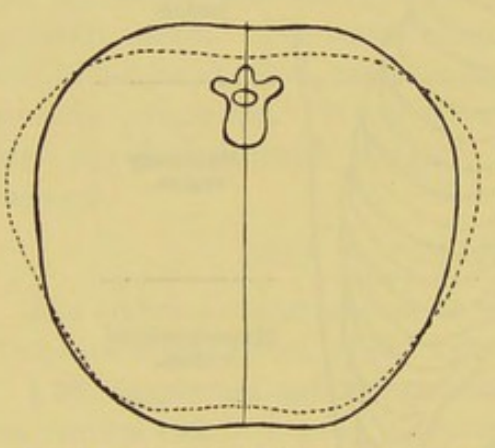

Fig. 34.—Chest of Emphysema. The dotted line represents the normal outline.

The *rachitic* chest is common in children. Owing to the weakness of the bones, the chest acquires a characteristic shape (Fig. 35). A vertical groove occurs at the weakest part of the wall of the chest—*i.e.*, down each side of the sternum, just outside the "rickety rosary" or beaded junction of ribs and cartilages (§ 447). Harrison's sulcus is often present at the same time ; it is a horizontal groove at the level of the xiphoid cartilage, running from the middle line in front obliquely outwards and slightly downwards as far as the mid axilla, along the costal arch.

The *pigeon-breast* is found in those who have had some obstruction to respiration in early youth, such as that due to adenoids. The sternum is prominent, the ribs meeting it at a more or less acute angle. The cross-section of the chest is therefore almost triangular (Fig. 36).

Among the irregular or *asymmetrical abnormalities* in the shape of the chest which the student should look for are *hollowing*, *prominence*, or *contraction*.

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[1] Woods Hutchinson, *Brit Med. Journ.*, 1899, vol. ii., p. 1176.

(α) *Localised Hollowing* or " flattening " of the infraclavicular region may indicate phthisis, or any disease rendering the underlying part of lung useless for respiration.

(β) *Undue Prominence* on one side of the ribs anteriorly may be due to : (i.) Scoliosis—*i.e.*, lateral curvature of the spine, the convexity of the chest being in the opposite direction. (ii.) Intrathoracic tumour, fluid, abscess, or air (pneumothorax) in the chest. (iii.) If the cardiac region be prominent, it may be the result of cardiac disease in early youth, before the ribs were fully developed, and possibly an adherent pericardium. (iv.) An enlarged liver or spleen may also cause a bulging of the lower ribs on the right and left sides respectively.

(γ) *Contraction* of an *entire side* of the chest which may be due to : (i.) collapse of a lung (§ 100) ; (ii.) previous empyema (§ 86) ; (iii.) chronic interstitial pneumonia and fibroid phthisis (§§ 97 and 94*a*).

The *Cyrtometer* is an instrument consisting of two flexible pewter or pure tin bands, joined by a hinge and graduated in inches. It is used to measure the *relative size* and *shape* of the two sides. Place the hinge *exactly* opposite the spinous processes posteriorly, and bend the pewter limbs round to the front, following the contour of the chest precisely. The instrument is then placed on paper, and the outline thus obtained may be marked by running a pencil round the band.

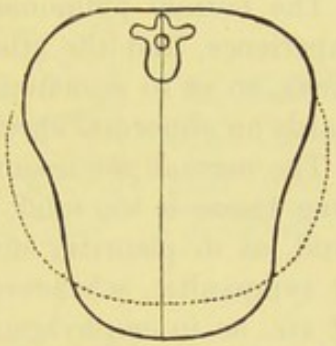

Fig. 35.—RACHITIC CHEST. The dotted line represents the normal outline.

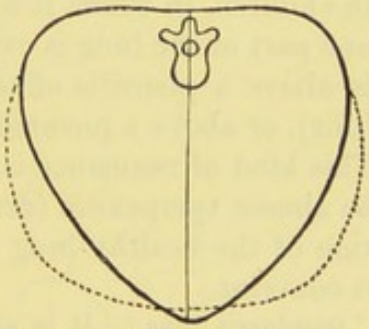

Fig. 36.—PIGEON CHEST. The dotted line represents the normal outline.

§ **75. Percussion** is, after inspection, the next step in the examination of the chest. There are two kinds of percussion, *immediate* and *mediate*. In the latter a piece of ivory or wood is placed on the chest, and is struck by a small hammer, or with the finger. The *immediate* is the more usual form of percussion. To elicit the normal resonance of the lungs percussion should be stronger than when applied to make out the cardiac dulness. Begin at the apex and percuss *alternate sides* at exactly corresponding points in order *to compare the healthy and unhealthy sides*, and thus work gradually downwards. Place the first or second finger *firmly* and *flat* against the chest, in a horizontal position—*i.e.*, parallel to the suspected line of dulness. (Only in suspected mediastinal tumour should it be placed vertically.) Then strike upon it with the tips of all the fingers of the right hand. The blow should come from the wrist, not the elbow ; and the " staccato " movement should be imitated. Some use one (the middle), two, or three of the fingers of the percussing hand, but this makes the stroke too light, unless, as sometimes happens,

it is desirable to demonstrate the delicate shades of pitch, intensity, and quality of the sound.[1]

When examining the *back* of the chest (Fig. 37), the patient should be instructed to cross his arms and bend a little forward so that the scapulæ are drawn out of the way. The normal resonance of the lung note posteriorly extends to the upper border of the eleventh rib on the right side, and the lower border of the eleventh rib on the left side. On deep inspiration the resonance extends an inch lower, and during deep expiration an inch higher. Owing to the thickness of the scapular muscles the note over the scapulæ may be quite dull in muscular people. To examine the *sides* of the chest the patient should be told to put his hands on the top of his head.

The normal pulmonary note can only be learned by practice and experience, and the student should *frequently practise first on normal chests*, so as to accustom himself to the normal resonance; and afterwards on abnormal chests.

The normal percussion note is resonant. It is *dull* or flat when the lung tissue is too solid, as in pneumonia; or when the chest contains fluid, as in pleuritic effusion. The percussion note is *hyper-resonant*, or tympanitic, whenever the lung tissue is unduly open—*i.e.*, too full of air, as in emphysema, when there is a cavity near the surface, or when there is air in the pleura (pneumothorax). Cracked-pot sound is a modification due to a large cavity (see Phthisis, § 94). It is normal in children, in whom it is due to the great elasticity of the ribs. When one part of the lung is over-distended with air, as it is in the part which is above a pleuritic effusion (which compresses the lower part of the lung), or above a pneumonic consolidation, the note is unduly resonant. This kind of resonance is called the "Skodaïc resonance"; and it may be almost tympanitic (drum-like) in character. It is due to the relaxation of the healthy lung tissue, and the increased amount of air which it contains.

Gairdner's Line.[2]—It is useful to remember that a line drawn from the left anterior axillary fold to the umbilicus is normally *resonant throughout its entire length*. Abnormally it may be impinged upon anteriorly by consolidation in the upper part of the left lung, cardiac enlargement, or by enlargement of the liver; and posteriorly by consolidation or fluid at the base of the lung, splenic enlargement, or other abdominal tumours.

Increased Resistance is another quality which can be observed in the process of percussion as above described. This property of resistance can perhaps be better elicited by placing a finger of the right hand over an intercostal space and pressing lightly. It is greatest over fluid, and thus affords an important sign in pleural

[1] The *pitch* or tone of the note is its position on the scale, and the more solid the structure, the higher the note; the more hollow, the lower and more drum-like or tympanitic. The *intensity* of a note depends on the solidity of the wall of a cavity as compared with its size. The *timbre* or quality of a note is a characteristic which depends on the nature and structure of the vibrating body, just as a wire string and a gut string, though producing the same note, possess a different timbre or quality.

[2] It was Sir William Gairdner who emphasised the value of this line in physical diagnosis, but I am not certain if he was the first to do so.

effusion, but is present also in consolidation, though in a less degree. It is a sign which, at first, is difficult to elucidate, but comes to be relied upon, in addition

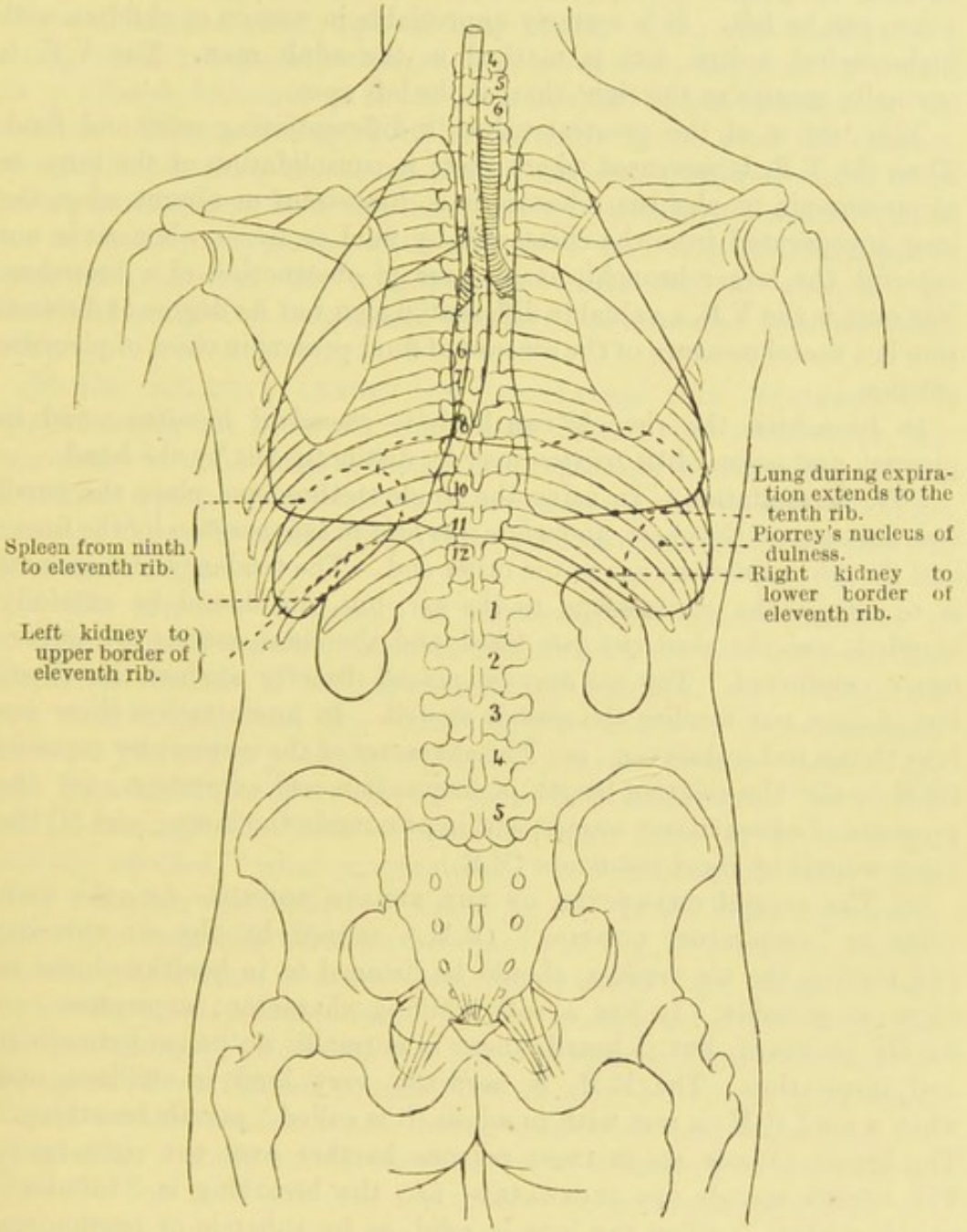

Fig. 37.—The Lungs and Other Viscera from the Back.—The right lung has three lobes, the left lung two only, and the positions of the fissures are shown in the figure. The lines on the figure are only approximate guides. When accuracy is desired, the exact position of the lung fissures is obtained by ausculto-percussion (§ 77*a*). A rough guide to the upper border of the lower lobes is found in the position of the vertebral borders of the scapulæ when the patient crosses his arms in front of him, and places each hand on the opposite shoulder. The great fissure, which separates the middle and lower lobes, on the right side, and the upper and lower lobes on the left side, is indicated on both sides by drawing a line from the second dorsal vertebra to the junction of the sixth costal cartilage with the sternum. The fissure separating the middle and upper lobes on the right side is found by drawing a line from the junction of the fourth costal cartilage with the sternum to meet the line of the great fissure in the mid-axilla.

to percussion, by some, especially by those whose appreciation of differences in note is imperfect.[1]

[1] Robert Maguire, *Practitioner*, April, 1897, p. 371, may also be consulted.

§ **76. Palpation** is the next step in the routine examination of the lungs. By palpation *Vocal Fremitus* (V.F.), or the vibration of the voice, can be felt. It is scarcely appreciable in women or children with high-pitched voices, but is marked in the adult man. The V.F. is normally greater at the right than at the left apex.

This test is of the greatest value in differentiating solid and fluid. Thus the V.F. is *increased* where there is consolidation of the lung, as in pneumonia or phthisis, whereas it is *diminished* or absent when the lung is separated from the chest-wall by fluid or air, or when air is not entering the larger bronchi, as in cases of obstruction of a bronchus. Not only is the V.F. a valuable differential sign, but its degree of diminution is a useful measure of the *amount* of fluid present in cases of pleuritic effusion.

In bronchitis the rhonchi can be felt—*rhonchial fremitus;* and in pleurisy and pericarditis *friction* may be distinctly felt by the hand.

§ **77. Auscultation.**—In using the single stethoscope, place the small end flat against the chest, and *while it is held in this position* by the finger and thumb, adjust the ear to the other end. The tendency of beginners is to adjust the stethoscope to the ear, but this should be carefully avoided, else the *chest end gets tilted*, and the chest sounds are imperfectly conducted. The ear may be placed directly against the chest, but it does not localise the sounds so well. In auscultation there are four things to be observed : (*a*) The character of the respiratory murmur (R.M.) ; (*b*) the relative length of inspiration and expiration ; (*c*) the presence of adventitious sounds within or outside the lungs ; and (*d*) the voice-sounds or vocal resonance (V.R.).

(*a*) The normal CHARACTER OF THE BREATH SOUNDS—*i.e.*, the vesicular or "respiratory murmur" (R.M.), caused by the air entering and leaving the air vesicles, should be listened to in healthy chests as often as possible. It has a soft whiffing character ; expiration can hardly be heard, but if heard, there is normally no pause between it and inspiration. The R.M. is normally very loud in children, and when a loud R.M. is met with *in adults*, it is called "*puerile* breathing." The breath sounds are in most persons harsher over the right apex. The breath sounds are INCREASED—*i.e.*, the breathing is "tubular" or "bronchial"[1] when the lung is solid, as by tubercle or pneumonia or when a new growth lies between the larger bronchial tubes and the surface. In this condition the sound produced in the larger bronchi is conveyed direct from them to the ear owing to the increased conductivity of the solid lung substance. *Bronchial* breathing can be heard *normally* by listening over the upper segment of the sternum, or near

[1] The terms "bronchial" and "tubular" are generally taught as synonymous, but some schools (*e.g.*, the Edinburgh) teach that there are three kinds of bronchial breathing—high-pitched, or tubular ; medium-pitched, or true bronchial breathing ; and low-pitched, or cavernous breathing.

the fourth dorsal vertebra at the back. It has three features—inspiration and expiration are of equal length and character, have an interval between them, and are both rough. *Cavernous* respiration is exaggerated tubular breathing, and is heard when the sound produced in a dilated bronchus or cavity is conveyed in like manner to the surface. Cavernous respiration is normally heard over the trachea. *Amphoric* breathing is a sound like air entering a bell-jar, and is heard over pneumothorax or a very large cavity. The breath sounds (R.M.) are DIMINISHED or absent when a layer of fluid or a thickened pleura intervenes between the lung and the chest wall, or when the air does not enter the lung tissue owing to obstruction in a bronchial tube.

(*b*) The RELATIVE LENGTH OF INSPIRATION AND EXPIRATION is approximately as 10 to 12, but heard through the stethoscope, the inspiratory is three times as long as the expiratory sound, which follows it without a pause. *Expiration is prolonged* in any disease which involves a loss of elasticity of the lung tissue, such as emphysema, or tubercle in an early stage.

(*c*) The presence or absence of ADVENTITIOUS SOUNDS has next to be noted. (i.) *Pleuritic friction* is produced by the two inflamed and roughened surfaces of the pleura rubbing together. (ii.) Within the lung various moist and dry sounds may be *added* to the respiratory murmur. Thus the presence of excessive mucus or other fluid in the large bronchial tubes gives rise to " large or bubbling *râles* " as the air bubbles through the fluid. When the small tubes or air cells are similarly affected, " small mucous râles " or " crepitations " are heard which resemble the rustling of tissue-paper. If the lining membrane of the large bronchial tubes be thickened and dry, or with only a small quantity of moisture present, " sonorous *rhonchi* " are produced, like the snoring of a person asleep. If the smaller tubes are thickened " sibilant rhonchi " are heard (see Fig. 40). Rhonchi are often hard to distinguish from friction sounds, but it may be remembered that, whereas friction sounds heard during inspiration and expiration are separated by a short but distinct interval of silence, rhonchi are not so separated, but fade one into the other. Crepitations sometimes resemble friction sounds, but are distinguished by being audible only during inspiration.

(*d*) The VOICE SOUNDS, or vocal resonance (V.R.). (i.) When the patient speaks, the vocal resonance is INCREASED (*bronchophony*) if the conductivity of the lung substance is rendered greater by consolidation, such as that produced by tubercle or pneumonia. If this be so great that even whispered words are conducted, it is known as *whispering pectoriloquy*. Some authors confine this term to the very exaggerated whispering sounds which are heard over large smooth-walled cavities. (ii.) The vocal resonance is DIMINISHED when a layer of

fluid or air intervenes between the lung and the chest wall (*e.g.*, in pleuritic effusion and pneumothorax), or when there is a thickened pleura. Nevertheless, in a slight pleuritic effusion the higher tones of the voice sounds are sometimes conducted, especially at the angle of the scapula, and resemble the bleating of a goat (hence called *Ægophony*).

Clinically, all the diseases of the lungs may be conveniently divided into those with **dulness on percussion**, those in which the percussion note is **normal**, and those in which it is **hyper-resonant**. Those with **dulness** may be subdivided into two groups—those in which the dulness is due to CONSOLIDATION, and those in which it is due to FLUID. The clinical features by which solidification of the lung is distinguished from fluid in the chest are so important that they are given in a tabular form.

TABLE V.—PHYSICAL SIGNS OF

	Consolidation of Lung.	**Pleural Effusion.**
INSPECTION.	Movement impaired. May be flattening over the part (if infraclavicular region).	Movement impaired. May be bulging (of intercostal spaces).
PALPATION.	V.F. INCREASED.	V.F. DIMINISHED or absent.
PERCUSSION.	Resonance impaired.	Absolutely dull over fluid.
AUSCULTATION.	BREATHING TUBULAR. V.R. INCREASED.	R.M. ABSENT OR WEAK. V.R. DIMINISHED.

(The most important features are in small capitals.)

§ 77*a*. **Ausculto-Percussion,** when employed by experienced observers, enables them to define the boundaries of the heart, or of a mediastinal tumour, with greater accuracy. It is useful to determine the lobe in which disease is situated. In this method the stethoscope (preferably a binaural) is placed over the middle of a lobe, while one coin is tapped on another, first over another lobe, and then over the same lobe as that to which the stethoscope is applied. The listening ear recognises the difference of the impact in the two cases. The coins are then placed over the supposed margins of the lobes, and by the slighter or stronger impact conveyed to the ear the division between the lobes can be readily defined. In pneumothorax the pathognomonic "*bell-sound*" is obtained by this method.

§ 78. **Examination of the Sputum.**—Much may be learned from an examination of the sputum. First, as regards its APPEARANCE. In simple pleurisy, though the cough is distressing, expectoration is absent (*i.e.*, the cough is "dry"). If the disease be confined to a moderate catarrhal process of the bronchial tubes (*e.g.*, bronchitis), the sputum is white, clear, and frothy ("mucous expectoration"). If the process be more severe and suppurative, or if the lung tissue be breaking down, then pus is present, and the sputum is yellowish (muco-purulent). Thin watery sputum is expectorated in large quantity in œdema of the lungs. In phthisis, when the lung is breaking down, the sputum is often voided in thick spherical purulent masses, like coins, hence called *nummular*. In cases of pulmonary abscesses, tuberculous cavities, and an empyema bursting into the lung, large quantities of almost *pure pus* are expectorated from time to time. Extremely fœtid expectoration is voided in gangrene of lungs and in bronchiectasis. The latter is distinguished by having large quantities of *putrid sputum*, brought up by paroxysms of violent cough at *one time*; while in the intervals the cough and expectoration are those of bronchitis. The bronchiectatic sputum, on standing,

separates into three layers—the upper clear and frothy ; the middle granular, with mucus ; the lower purulent, with thick " Traube's plugs " (p. 139). The foul odour is due to valerianic and butyric acids. In *pneumonia* the sputum is very characteristic, being (i.) almost airless and extremely viscid, so that the vessel containing it may be inverted without spilling it, and (ii.) tinged with blood, thus having a " rusty " colour. In severe cases, and in new growth of the lung, the sputum becomes thinner, frothy, and dark red, the " prune-juice " sputum. *Casts* of the bronchial tubes, which can be seen by the naked eye (Fig. 47, p. 163), are expectorated in plastic bronchitis, and occasionally in croupous pneumonia, and shreds of membrane in diphtheria. Hydatid cysts, resembling empty gooseberry-skins, are expectorated in that rare condition hydatid disease of the lungs, or when hydatid of the liver ruptures into them. In town dwellers, and those with dusty occupations, the sputum is dark, or even black, from the presence of carbonaceous and other particles.

Microscopic Examination of the Sputum.—Various *bacteria* (*e.g.*, tubercle, pneumococcus, influenza) are found in the sputum in patients suffering from certain microbic disorders. The method of detecting these is described in Chapter XX.

In all destructive diseases of the lung fragments of pulmonary tissue are present—*i.e.*, epithelial cells and connective tissue. The most characteristic is *elastic tissue*. Elastic fibres are best revealed by taking a small portion of the sputum and boiling it with liquor potassæ, which breaks up and renders clear all the other elements, but leaves the elastic fibres unattacked. These sink to the bottom of the test-tube, and may be withdrawn by a pipette (precautions, see Urinary Deposits) for examination under the microscope. They appear as wavy, highly refractile fibres, of uniform thickness, with square-cut ends, and are typically arranged as if surrounding an air cell (Fig. 38). Elastic tissue is found in the mouth after meals, so the mouth and teeth should, as a precaution, be cleansed before the observation is made ; but circularly arranged elastic fibres are quite distinctive of breaking-down lung tissue. The method of examining for tubercle is given in Bacteriology (Chapter XX.).

Fig. 38.—Elastic Fibres.

The Traube " plugs " of a bronchiectatic sputum (§ 103) are little pellets, which contain pus and epithelial cells, with needle-shaped fatty-acid crystals. Sometimes elastic fibres are also present in small amount. They are believed to be pathognomonic of bronchiectasis.

Fig. 39.—Charcot-Leyden Crystals.

Curschmann's spirals are found in the sputum of asthmatic patients. They form pellets or *perles*, the size of sago grains, which can be uncoiled to form a thread about an inch long. Microscopically, they are seen to consist of fine mucous fibrils wound spirally round a central core of mucus. They are probably allied to small bronchial casts (Finlayson). *Charcot-Leyden* crystals (Fig. 39) are colourless, pointed, octahedral crystals, formerly supposed to be pathognomonic of asthma, but now known to occur in the sputum of bronchitis also. They are composed of phosphate of spermin. Pœhl considers spermin to be a decomposition product of nucleo-albumin which normally circulates in the blood. Many diseases (especially nervous) are associated with an excess of phosphate in the system, which, combined with spermin, may form Charcot-Leyden crystals. They have also been found in the blood of leukæmia. *Hæmatoidin* crystals are brown or yellow needles or plates, found in cases of old hæmorrhage from any cause. *Cholesterin*, *leucin*, and *tyrosin* crystals are found occasionally in cases where the sputum has been

purulent for a long time. Various *parasites* (actinomycosis, echinococcus, *Distoma pulmonale*, etc.) are sometimes found in the sputum. Sarcinæ and *Oïdium albicans* come usually from the alimentary tract.

PART C. DISEASES OF THE LUNGS AND PLEURÆ: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **79. Classification.**—For practical purposes diseases of the lungs and pleuræ, like those of the heart, may be divided into ACUTE and CHRONIC, and each of these may be subdivided into those without dulness, those with dulness, and those with hyper-resonance.

		Acute.		Chronic.
WITHOUT DULNESS.		I. Acute Bronchitis. II. Dry Pleurisy. III. Acute Phthisis. IV. Whooping-cough.		I. Chronic Bronchitis (and Plastic Bronchitis).
WITH DULNESS.	Common.	I. Pleurisy with effusion (and Empyema).		I. Chronic Phthisis[1] (and Fibroid Phthisis). II. Hydrothorax. III. Pulmonary Congestion (or Œdema).
	Rare.	II. Pneumonia— (*a*) Lobar. (*b*) Lobular.		IV. Interstitial Pneumonia. V. Thickened Pleura. VI. Cancer and other neoplasms. VII. Collapse of the lung. VIII. Syphilitic disease.
HYPER-RESONANCE.		I. Pneumothorax.		I. Emphysema.

Paroxysmal.

I. Asthma.

§ **80.** The **Routine Procedure** here resembles in principle that of diseases of the heart. First, *What is the patient's leading symptom?* If suffering from lung disease, his cardinal symptom will be one of those mentioned in section A. Breathlessness and cough are the chief cardinal symptoms.

Secondly, follow this up with a few questions to ascertain the *history of his illness*, and especially whether *the disease be acute or chronic*. Other important points are whether the patient has been exposed to a "chill," and whether there is any "lung disease" in the family. Do not use the word "consumption"; it may frighten your patient unnecessarily.

[1] There is no dulness in quite the early stages of some cases.

Thirdly, proceed to the PHYSICAL EXAMINATION OF THE LUNGS. The routine method is as follows :

1. Ascertain whether there is any increased rate or other modification in the breathing or alteration in the shape of the chest (by *inspection*, and, if necessary, by measurement).
2. Ascertain if there be any dulness or hyper-resonance (by *percussion*).
3. Listen to the breath and voice sounds, directing special attention to any part suspected of disease (by *auscultation*).
4. Test the voice sounds by *palpation*.
5. The sputum should be inspected, and, if necessary, examined microscopically.

The chest should always be stripped, and it is more convenient to examine the patient in a sitting posture, if he be not too ill.

If the illness developed gradually, and is of some standing, and unattended by marked constitutional disturbance, then turn to **Chronic Pulmonary Disorders** (§ 91, p. 160).

If the illness came on recently and suddenly, accompanied by fever, quickened respiration, coated tongue, and with marked malaise, then the case is one of the **Acute Pulmonary Diseases**, below.

There is one disease of the lungs, ASTHMA, which comes on in sudden acute attacks from time to time ; it is **chronic**, with **acute exacerbations** (§ 90, p. 158).

Acute Diseases.—We now proceed to percuss the chest. In all acute diseases special attention should be directed to the lower and back part of the chest just below the scapulæ. Careful percussion of this region will give us important aid in diagnosis.

TABLE VI.—DIAGNOSIS OF ACUTE DISEASES OF THE LUNGS AND PLEURÆ.

	Percussion Note.	Auscultation.
I. **Acute Bronchitis**	Normal	R.M. and V.R. normal ; Loud moist râles and dry rhonchi.
II. **Dry Pleurisy**	Normal	Breath and voice sounds normal ; Pleuritic friction.
III. **Acute Pulmonary Tuberculosis**	Normal, or scattered areas of dulness.	Scattered fine moist râles may be the only auscultatory signs.
IV. **Pleurisy, with effusion**	Dull	R.M., V.R., and V.F. diminished ; Pleuritic friction at early and late stage.
V. **Croupous Pneumonia**	Dull	V.R. and V.F. increased ; Bronchial breathing ; Fine or coarse (redux) crepitations.

The acute diseases without alteration in the percussion note, *i.e.*, **without dulness**, excluding WHOOPING-COUGH, which is an infective disorder, and has no physical signs in the lungs peculiar to it, and ASTHMA, which is of a paroxysmal character—are : I. ACUTE BRONCHITIS ; II. DRY PLEURISY ; and III. one form of ACUTE PULMONARY TUBERCULOSIS.[1]

I. *The patient complains of a cough, with frothy expectoration, and his temperature is slightly elevated ; there is* **no alteration** *in the percussion note, but on auscultating the chest, loud* RHONCHI *are heard. The disease is* ACUTE BRONCHITIS.

§ **81. Acute Bronchitis,** or inflammation of the bronchial tubes, is certainly the most common disease of the lungs in this climate.

Symptoms.—The disease commences gradually in the course of one or two days, with a feeling of tightness of the chest, of soreness behind the sternum, shortness of breath, frequent cough, and slight rise of temperature, 100° to 101°. The inflammatory process lasts from ten days to three weeks, and gradually subsides. The sputum is viscid and scanty during the first few days, and then becomes thinner and more easily coughed up.

Physical Signs.—The percussion note is unaltered unless, as so frequently happens, emphysema be present also, in which case the chest is unduly resonant. On auscultation the vesicular murmur is obscured over the whole chest on both sides by loud rhonchi and moist râles (see Fig. 40). On palpation rhonchial fremitus can frequently be felt.

Causes.—Bronchitis is generally attributed to : (i.) A chill ; that is to say, sudden exposure to cold, with a determination of blood to the interior. (ii.) Sometimes, however, it is caused by spreading from laryngitis. (iii.) It is a frequent complication of many of the specific fevers, especially measles, whooping-cough, and typhoid. It is so frequently present with the first and last as to constitute an aid to the diagnosis of those diseases. (iv.) Certain occupations which expose people to irritating vapours and small particles of dust predispose to acute bronchitis. Thus the cotton-mill hands and chemical manufacturers frequently suffer from bronchitis. It is also common amongst cabmen, mariners, and others who are exposed to all weathers. (v.) It is a common accompaniment of many other pulmonary diseases, though it may be a subordinate feature ; and (vi.) it is commonest in childhood and old age.

The *Diagnosis* is not difficult in most cases, but *acute tuberculosis* is at first very apt to be regarded as acute bronchitis. The diagnosis is aided by the greater elevation, and the intermitting character of the

[1] In the early phase of this malady there is no alteration of the percussion note, but as the disease progresses a patchy dulness appears, if the patient live long enough.

pyrexia in the former, and by the presence of the tubercle bacillus in the sputum. The *capillary bronchitis* of children is really a *broncho-pneumonia* (*q.v.*) ; the constitutional symptoms are much more marked, there may or may not be some dulness, and the differentiation from simple acute bronchitis is not always easy.

The *Prognosis* is favourable in adolescence and adult life, and it

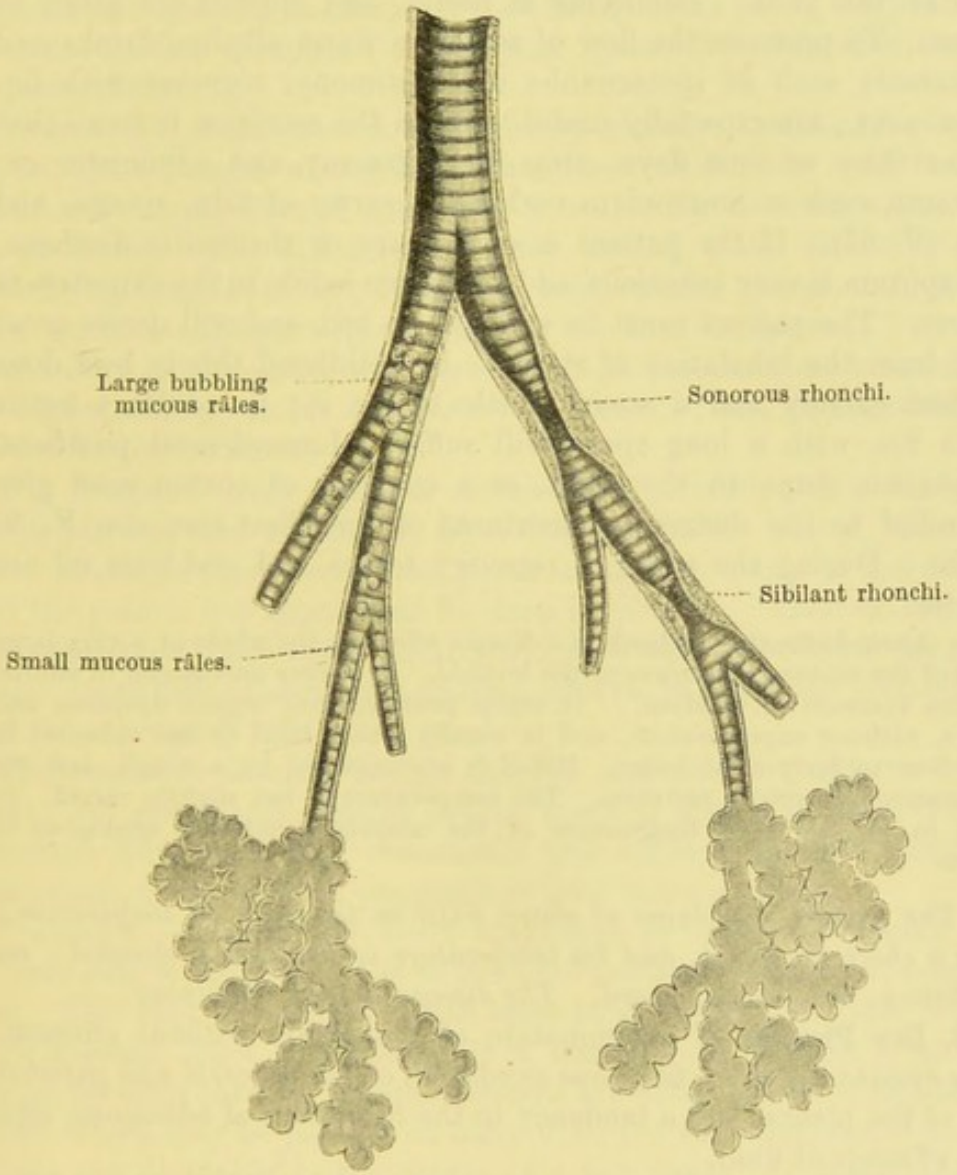

Fig. 40.—Diagram to show the production of **râles** (moist sounds) on left, by mucus in bronchial tubes, and **rhonchi** (dry sounds) on right, by narrowing of the tubes.

always clears up in one to three or four weeks, though it has a special liability to return, and ultimately to become chronic. It is dangerous in infancy and old age, where the resisting powers are feeble. It is one of the commonest causes of death in the latter. If an attack of acute bronchitis does not begin to clear up in two or three weeks, pulmonary tuberculosis should be suspected, especially if the patient be young.

Treatment.—The indications are: (i.) During the first stage, to promote the secretion; (ii.) when the secretion is free, to stimulate the bronchial mucous membrane; (iii.) during convalescence, to improve the general condition so as to enable the patient to throw off his liability to bronchitic attacks. At the onset give an aperient and a diaphoretic mixture, with perhaps a few grains of Dover's powder to soothe the pain. Poulticing is useful, and emetics are given to children. To promote the flow of secretion warm alkaline drinks and expectorants such as ipecacuanha and antimony, together with liq. ammon. acet., are especially useful. When the secretion is free—that is, after three or four days—stop the antimony, and administer expectorants, such as ammonium carbonate, syrup of tolu, senega, and squills (F. 57). If the patient is of a gouty or rheumatic diathesis, or the sputum is very tenacious, add potassium iodide to the expectorant mixtures. The patient must be confined to bed, and will derive great benefit from the inhalation of steam. In childhood this is best done by a bed canopy and a steam kettle beside it; in adults, a kettle on the fire with a long spout will suffice. Linseed-meal poultices, a turpentine stupe to the chest, or a covering of cotton wool give great relief to the distressing tightness of the chest (see also F. 30 and 68). During the stage of recovery tonics and cod-liver oil are called for.

§ 81*a*. **Acute Suffocative Catarrh** is a disease affecting the whole or a very large portion of the mucous membrane of the bronchi. It is very uncommon in adults, but more common in children. It starts acutely, with urgent dyspnœa and cyanosis, without expectoration, and is usually either fatal or has subsided in twenty-four to forty-eight hours. Relief is accompanied by a cough, and the expectoration of copious secretion. The temperature is but slightly raised. It is said to be due to a turgescence of the mucous membrane, analogous to urticaria.

II. *The patient complains of sharp* PAIN *in the chest on inspiration; he has a short dry cough, and his temperature is moderately elevated; on auscultation,* FRICTION *is heard. The disease is* DRY PLEURISY.

§ 82. **Dry Pleurisy** is inflammation of the pleura without effusion. In this disease there is a fibrinous exudation on the visceral and parietal layers of the pleura, and a tendency to the formation of adhesions, and to the effusion of fluid.

Symptoms.—The disease in some cases comes on quite suddenly with a stitch-like pain in the chest. The constitutional disturbance is never very great, and the patient rarely takes to his bed. The temperature may rise to 100° or 101° F., rarely higher. The most obvious symptom in this disease is pain in the chest, affecting one side only in most cases, and characterised by being greatly increased on deep inspiration. The pain is caused by the contact of the inflamed pleural surfaces, and is usually, though not necessarily, located over the diseased part.

Physical Signs.—Percussion reveals nothing. On auscultation, the respiratory murmur may be found to be normal or shortened, as the patient endeavours to restrain the movements of the chest on account of the pain so caused. From the very outset a pleuritic rub is heard over one side, often most marked at the angle of the scapula (compare § 77). Sometimes the inflammation undergoes resolution or adhesion, sometimes it goes on to effusion. As effusion takes place, the pain and pleuritic friction disappear, to reappear again when this subsides (as in pericarditis).

Causes.—(i.) Sometimes it is a primary malady, attributed to chill, especially in persons of a gouty or rheumatic diathesis. (ii.) It may occur as a complication of some acute infective disease, such as measles or scarlatina. (iii.) Inflammation may extend from disease of the underlying lung, such as pneumonia, tuberculosis, cancer, and embolism, or from adjacent organs, such as the liver or spleen. (iv.) Undoubtedly a large number of apparently simple pleurisies are tuberculous in origin (some go so far as to say 82 per cent.) ; and this fact should always be remembered.

The *Diagnosis* from *muscular rheumatism* (pleurodynia) is made by the tenderness and absence of friction sound in the latter. In *intercostal neuralgia* there are tender points along the course of the nerve, and the pain is not aggravated by deep inspiration. Pleuritic friction is distinguished from the rhonchi heard in *bronchitis* by there being in nearly every case of pleurisy a distinct interval between the inspiratory and the expiratory rub.

Prognosis.—It is not a serious malady, and readily yields to treatment ; but sometimes effusion occurs which is apt to become chronic (Pleuritic Effusion, § 85). Sometimes this effusion becomes purulent (Empyema, § 86), and the prognosis at once becomes grave.

Treatment.—Considerable relief is derived by simply strapping the affected side of the chest, so as to limit the costal movements of respiration. This may be combined with some local application ; that which gives greatest relief is undoubtedly a linseed-meal poultice. As the disease becomes chronic, counter-irritants are called for, more especially iodine, which may be painted on daily until the skin becomes sore. If it does not disappear in the course of a few weeks, we must suspect some other cause for the mischief, such as those mentioned under pleurisy with effusion. Diuretics, diaphoretics, iron, and other tonics are useful.

III. *The patient exhibits the signs of subacute bronchitis ; but he has* SEVERE MALAISE *and a* HECTIC TEMPERATURE, *and the sputum contains* TUBERCLE BACILLI. *The disease is* ACUTE PULMONARY TUBERCULOSIS.

§ 83. **Acute Pulmonary Tuberculosis** (acute phthisis, galloping consumption) is a catarrhal process affecting the entire lung tissue, due to the invasion of the

tubercle bacillus. It is often part of a tuberculous process infecting the whole body, and is therefore sometimes described as the pulmonary form of acute general tuberculosis (see Chapter XV., where a chart is given showing the typical course of the temperature in both diseases).

Symptoms.—The malady is of most insidious onset, with progressive weakness and emaciation. Some weeks before any physical signs are evident the thermometer shows the typical intermittent pyrexia so characteristic of tubercle—an evening elevation of 101° to 103° F., and a morning normal temperature. In some cases the inverse type is present, where the temperature is higher in the morning than in the evening. Night-sweats and cough are present, with muco-purulent expectoration. Dyspnœa, and sometimes cyanosis, develop out of proportion to the physical signs; the latter symptom may be extreme, and of itself is a very characteristic feature. Great weakness ensues, and in the third or fourth week the patient may develop the symptoms of the typhoid state.

The *Physical Signs* referable to the lungs are indefinite, or resemble at first those of bronchitis. At first there is no alteration in the percussion note, but by-and-by careful percussion discovers scattered patches of dulness. Auscultation at first may give little help, but in the course of a week or so it reveals rhonchi and fine râles over certain areas, which do not shift from place to place, as in bronchitis. Later on the râles are coarse and bubbling, and areas of tubular breathing may be found.

The *Diagnosis* in the first stage from bronchitis and broncho pneumonia is extremely difficult. We have to rely upon the disproportionate emaciation and cyanosis, the character of the temperature, and the patchy distribution of the physical signs in tuberculosis. In other cases the malady is almost indistinguishable from enteric fever, except for the marked predominance of the pulmonary signs and the absence of abdominal symptoms. In all stages the detection of the tubercle bacillus in the sputum is a valuable aid to diagnosis, though its absence does not exclude acute pulmonary tuberculosis.

Causes.—The disease may occur at any age, but is commonest in young adults, and in those with a family history of consumption. In some instances acute general tuberculosis originates from a primary focus, such as a tuberculous joint, which had been considered cured. Sometimes the disease follows measles or whooping-cough in children.

Prognosis.—The disease is almost uniformly fatal in about two to twelve weeks. *Treatment* is almost entirely symptomatic.

§ **84.** *The patient, a child, has* PAROXYSMS *of coughing which frequently terminate in* VOMITING ; *there is very slight feverishness, but the only signs in the lungs are those of a little bronchial catarrh. The disease is* WHOOPING-COUGH.

Whooping-cough (Pertussis) is an acute infectious disease, and it will be described among the microbic disorders (Chapter XV., § 368).

We now turn to the **Acute Diseases with Dulness on Percussion**—I. PLEURISY WITH EFFUSION (Serous or Purulent); II. PNEUMONIA and III. BRONCHO-PNEUMONIA.

I. *The patient has a* DRY COUGH, *with moderate fever and other constitutional symptoms. On examining the chest, the respiratory murmur, vocal resonance, and vocal fremitus are found to be diminished or absent. The disease is* PLEURISY WITH EFFUSION.

§ **85. Acute Pleurisy with Effusion.**—When describing acute Dry Pleurisy (§ 82) it was pointed out that the disease may undergo resolu-

tion or result in adhesions. It may also go on to effusion—Pleurisy with Effusion.

Symptoms.—There is usually a history of a more or less acute onset with pain in the side (§ 82), but as the disease progresses, and the surfaces of the pleura are separated by fluid, pain becomes less and less marked. The patient suffers from general malaise, and finds it difficult to lie on the sound side, because the weight of the fluid on the other side impedes the action of the lung. A degree of breathlessness may be present, but even with a large amount of fluid this is not invariably a prominent feature.

Physical Signs (see Fig. 41).—Percussion reveals absolute dulness over the fluid. Above the level of the fluid, if the lung be otherwise healthy, there is a hyper-resonant note (Skodaic resonance). When the effusion is large it causes displacement of organs, which may be very considerable (see Fig. 42). The level of the fluid does not usually shift with the position of the patient, as it does when there is non-inflammatory (dropsical) fluid in the chest. On auscultation over the fluid, the breath sounds are absent; the vocal resonance is greatly impaired or lost.[1] At the upper margin of the fluid posteriorly—perhaps just about the angle of the scapula—only the highest pitched tones of the voice are transmitted, and they produce, therefore, a sound like the bleating of a goat (ægophony). On palpation, the vocal fremitus is found to be diminished or absent over the fluid, and there may be bulging of the intercostal spaces. The amount of fluid present may be estimated by (i.) the degree of diminution of the vocal resonance and fremitus, and (ii.) the amount of displacement of organs. The diagnosis of pleurisy in its earlier stages is referred to under Dry

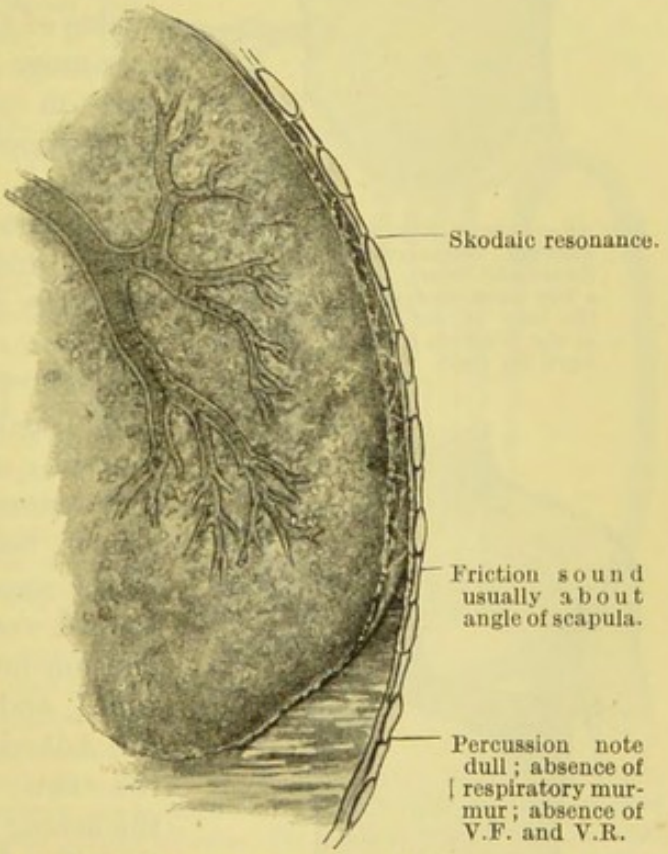

Fig. 41.—Diagram to show physical signs produced in different positions in ACUTE PLEURISY WITH EFFUSION.

[1] Bronchial or tubular breathing occurs wherever there is moderate effusion, because the lung collapses with the progress of the effusion.

Pleurisy. The differentiation of the physical signs of fluid in the chest, as compared with those of consolidation of the lung, is so important that it is given in a tabular form in § 77. It is sometimes difficult to make out the left margin of the cardiac area when there is effusion in the left pleura. Dr. S. H. Habershon has suggested a very valuable aid in such cases. Place a vibrating tuning-fork, such as aural surgeons use, in mid axilla over the seventh rib. Listen with the stethoscope over the centre of the cardiac area, and gradually move it towards the tuning-fork, and in other directions. As the stethoscope crosses the boundary of the heart, there is a distinct difference in the note heard through the stethoscope, and in this way the cardiac boundary may be determined.

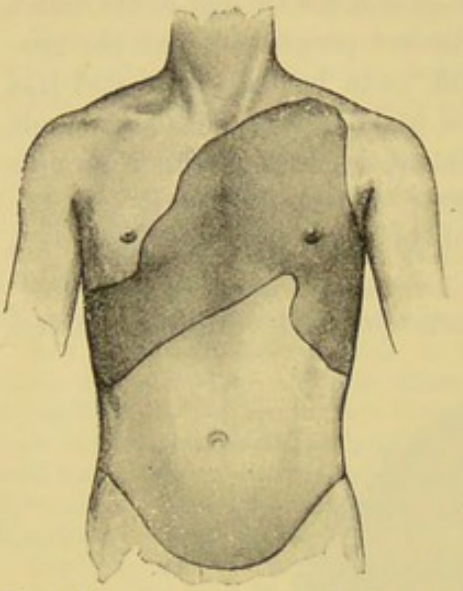

Fig. 42.—Case of PLEURITIC EFFUSION, showing displacement of organs (heart and liver). The patient was a boy aged twelve, admitted under the care of Sir William Gairdner in the Western Infirmary, Glasgow, April 20, 1895.

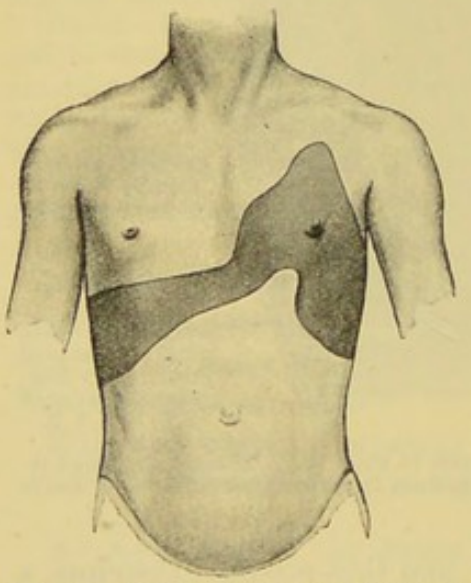

Fig. 43.—Shows altered state of dulness under use of diuretics (April 29). Lin. hydrarg. was applied, and he took internally Pot. cit. gr. x. Pot. iod. gr. V. Liq. am. acet. ℥ xx. Decoct. scopar. ʒii., quartis horis. Patient also had diarrhœa at this time.

Course and Prognosis.—In about a fortnight from the date of onset the fluid usually shows signs of diminution in quantity, the vocal fremitus and resonance return, and the breath sounds become more audible. This is the usual course, but several untoward results may ensue: (i.) The effusion may remain for an indefinite time, and re-collect after tapping. (ii.) Adhesions may take place between the two layers, and considerable thickening of the pleura result. (iii.) The fluid—especially in children after scarlatina—may become purulent (Empyema, see below).

Treatment. — To get rid of the effusion purgatives, diuretics, and diaphoretics (potassium citrate and bitartrate, potassium nitrate, liquor ammoniæ acetatis, etc., F. 55) are often efficacious. Iron and other tonics are useful. If these measures fail after a few weeks' trial, paracentesis should be performed (Figs. 42 and 43). Under certain conditions it is inadvisable to delay paracentesis: (i.) A large effusion (*e.g.*,

with dulness extending upwards as far as the third rib ; (ii.) cardiac embarrassment, as evidenced by cyanosis, palpitation, and a rapid pulse ; (iii.) respiratory embarrassment, shown by urgent dyspnœa and paroxysmal attacks of coughing ; (iv.) subsequent effusion in the other pleura, or œdema of the other lung. It should be remembered, in recurrent effusion, that tubercle may be the cause.

Paracentesis Thoracis.—The instrument used is an adaptation of the familiar trocar and cannula. We are here dealing with a cavity whose contents are under a minus pressure, so it is necessary to have a pump or exhausted bottle communicating with the trocar (see Fig. 44). The site of puncture—usually the seventh interspace in the posterior axillary line—should be thoroughly cleansed with soap and water, then with turpentine, and a carbolic fomentation, 1 per cent., applied for several hours before puncture. The needle of the aspirator should be boiled. The bottle or chamber of the syringe is next exhausted of air. If the point of the instrument be not very sharp, it is desirable to make a nick with a scalpel in the skin, previously pulled downwards over the rib below. Then the instrument is thrust into the intercostal space boldly at the acme of an inspiration. Communication is then established with the bottle or syringe, the flow being regulated by the tap or piston, so that the outflow may not be too rapid. Much coughing by the patient indicates that the point is touching the lung. A quantity varying between 5 and 50 ounces may be withdrawn, but the operation must be stopped if coughing or respiratory distress is caused. To seal the opening, all that is necessary is to take a piece of gauze, pour upon it some collodion, apply it to the skin alongside the cannula, withdraw the latter, at the same time pressing the gauze over the site of puncture, and finally fixing it there with adhesive plaster. If the fluid contain blood, it may denote a slight wound of the lung or carcinoma, or occasionally tubercle. If it be purulent, the surgical measures for empyema are applicable, and it is wise to be prepared for this eventuality. In cases of serous effusion, Sir James Barr [1] recommends that the fluid should be withdrawn by siphonage, and replaced by about half to three-quarters of its bulk of sterilised air. When all the liquid is withdrawn, he injects 4 c.c. of adrenalin (1 in 1,000), diluted with 10 c.c. of sterile normal saline.

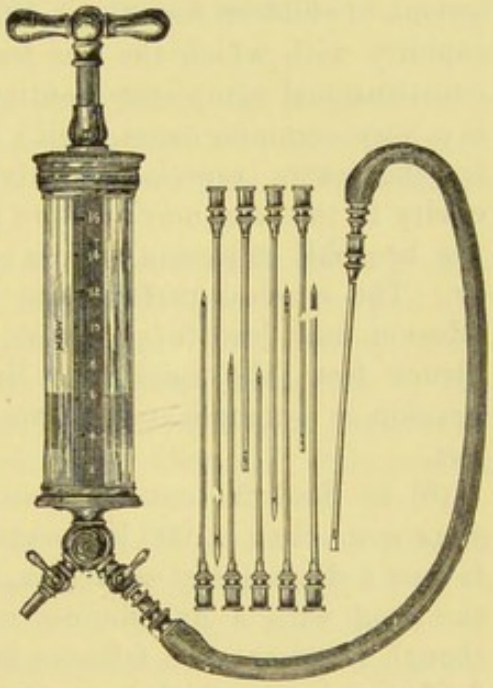

Fig. 44.—Aspirator for pleuritic effusion.

I *a. The physical signs are those of pleurisy with effusion, but it does not clear up in due course, and the patient has* SWEATINGS, SHIVERINGS, *and* IRREGULAR ELEVATIONS *of temperature. The disease is probably* EMPYEMA.

§ **86. Empyema** is a collection of purulent or sero-purulent fluid within the pleura. It most often follows a serous effusion, but it may be purulent from the beginning.

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[1] See Bradshaw Lecture, *Brit. Med. Journ.*, November 9, 1907.

The *Symptoms* and *Physical Signs* are similar to those of serous effusion (*q.v.*, *supra*), with certain others in addition—viz. : (1) It may be found that the fluid *does not clear up* as a serous effusion should do, and thus the presence of pus may be suspected. (2) Whenever pus forms, either in the pleura or elsewhere, it is marked by the occurrence of sweatings, shiverings, and an intermittent pyrexia. (3) Œdema of the integument, the pointing of an abscess in an intercostal space, over the clavicle, or even in the groin, or copious discharge of pus by the mouth, are in rare instances the first distinct evidence of a localised empyema. (4) The history generally throws considerable light on the case by revealing one of the *causes* of empyema—namely :

(i.) It is far more frequent in childhood than in adults. The empyema of children sometimes differs from that of adults in the extreme rapidity with which the pus forms, and the frequent absence of the constitutional symptoms mentioned above.[1] (ii.) Tuberculous disease is a very common cause. (iii.) It frequently follows one of the acute specific fevers, especially scarlatina, measles, and smallpox. (iv.) A cavity in the lung may rupture into the pleura. If it is shut off from the bronchi, empyema ensues ; if not, pyopneumothorax is caused. (v.) The careless performance of paracentesis in a serous pleuritic effusion may lead to empyema. (vi.) Abscesses may burst into the pleura from the spine, the liver, etc. (vii.) When signs of fluid develop as a complication of pneumonia, it practically always indicates pus.

(5) In doubtful cases a leucocyte count should always be made, since more than 20,000 leucocytes per cubic millimetre would strongly favour a diagnosis of empyema. (6) The aspiration of a few drops of the fluid with a hypodermic needle will often settle the diagnosis, though there are two fallacies in this method : first, in rare cases the fluid may be too thick to come through the needle ; or, again, the pus may be encysted between the lobes of the lung. In either case, the use of the longer cannula of an aspirator will generally obviate the difficulty.

Prognosis.—Empyema is always serious, and may run a somewhat prolonged course of some months. Cases of pure pneumococcal empyema are much more favourable than those due to streptococci or staphylococci, either alone or with the tubercle bacillus. Its course can be considerably modified by prompt and adequate surgical treatment. Early operation, adequate drainage, and strict aseptic precautions, both at the operation and at the subsequent dressings, are the points in treatment which most favourably influence prognosis. If left to itself, the results vary : sometimes there is compression and destruction of the lung ; sometimes there is a falling-in of the side of the chest ; sometimes, as above mentioned, the pus burrows in various directions ; or it may become partially absorbed, and result in a caseous mass.

[1] Many of the remarks relative to Pyopericarditis (§ 38*a*) apply here.

Treatment.—When we are sure that the fluid is purulent the empyema should be opened and drained without delay. Every aseptic precaution should be taken, and from 1 inch to 1½ inches of rib should be removed. When the patient is anæsthetised, a needle should first be inserted to locate the pus, and this should determine the site of the operation, a point being selected in as dependent a position as possible. Drainage should be effected by means of a large tube, which can usually be shortened to 1 inch or so after about forty-eight hours. At subsequent dressings the same strict asepsis should be maintained, because secondary infection makes the prognosis much worse. In the case of very large empyemata, causing great embarrassment, it is sometimes advisable to remove some of the pus by aspiration as a preliminary measure, but this should be followed by operation after about twelve hours.

II. *The patient has been* TAKEN ILL SUDDENLY ; *the temperature is high, the dyspnœa considerable, and the expectoration soon becomes rusty ; there are* SIGNS OF CONSOLIDATION *at the base of one lung. The disease is* ACUTE LOBAR PNEUMONIA.

§ **87. Pneumonia**—*i.e.*, inflammation of the pulmonary tissue proper, or parenchymatous inflammation—occurs in two forms. The *first* and more acute is, from its area of distribution, termed " Lobar Pneumonia," or, from the nature of the inflammation, " Croupous Pneumonia." The *second* is termed " Lobular Pneumonia," because it affects the lobules of the lungs (also called Broncho-pneumonia, Catarrhal Pneumonia ; see below).

Acute Lobar Pneumonia commences suddenly, with well-marked constitutional symptoms, such as headache, backache, rigor, and, in children, vomiting. The temperature during the rigor rises to 103° or 104° F., and it remains at this point for about a week (Fig. 45). The aspect of a pneumonia patient is very characteristic (§ 7)—the face is flushed, and herpes often appears on one side of the mouth. There is pain in the affected side, short cough, shallow, rapid breathing, and on the third or fourth day tenacious rusty-coloured sputum. The pulse-respiration ratio is 2 to 1, instead of the normal 4 to 1. The urine is scanty, high-coloured, with diminution of the chlorides. The patient shows more and more distress, and in a short time there may be delirium, with signs pointing to failure of the heart. About the *seventh* or *eighth* day the fever, in favourable cases, terminates by crisis, falling to normal in the course of a few hours. This is accompanied by marked general improvement ; the pulse-respiration ratio returns to normal, and a critical sweating or diarrhœa may occur. Crisis often occurs on the odd days—*i.e.*, fifth, seventh, ninth, or eleventh of the disease. Pseudo-crises occasionally occur, but these are distinguished from true crises by the fact that the pulse and respiration do not return to normal. In rare cases the temperature falls by lysis. The whole

illness lasts about two to three weeks. If it lasts longer, *tuberculosis should be suspected* (§ 87*a*).

The *Physical Signs* are limited to one lobe or one lung, usually the right lower lobe. It is only in rare cases that both lungs are affected. Percussion may, for the first day or two, reveal no dulness, but, as a rule, there is elicited early in the disease slight impairment of the percussion note, which soon becomes dull. On auscultation, the breath sounds are weak, and fine rustling crepitations are heard, which have been compared to the rustling of hair or tissue-paper against the ears. As the inflammatory exudation increases, the lung tissue becomes solid, and over the dull area we get all the *signs of consolidation* (p. 138). When the fever abates, coarse moist râles (redux crepitations) are heard, and the percussion resonance and normal breath sounds gradually return.

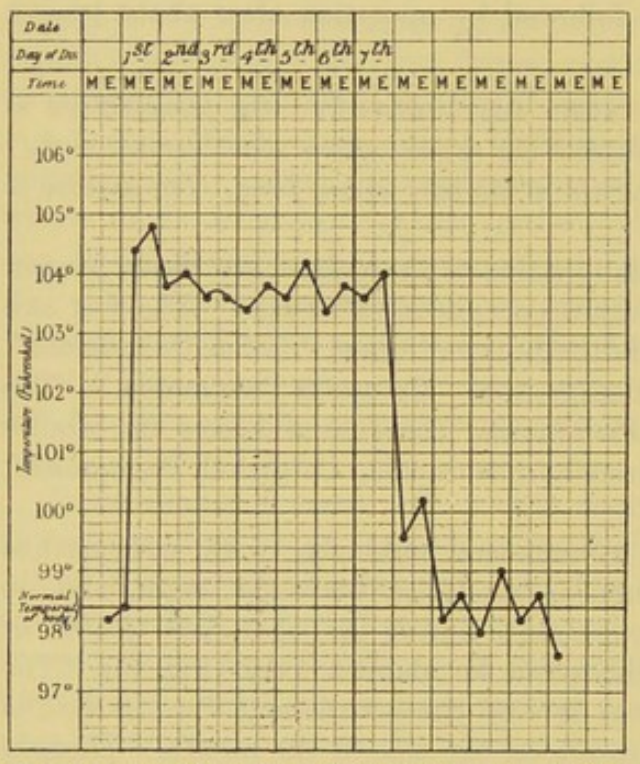

Fig. 45.—Acute Lobar Pneumonia, showing typical crisis on the seventh day. George H., aged thirty-five, was taken ill very suddenly when in the infirmary with shivering and acute pain in the side.

Etiology.—Pneumonia occurs at all ages and in both sexes, but is commonest in adult males. It is a microbic disease, the specific cause being a diplococcus, the pneumococcus of Fraenkel. Debilitating influences, such as exposure, are said to predispose to the disease; but it is surprising how often strong, apparently healthy men are attacked, and these not infrequently succumb. A blow on the chest may determine an attack. Like other local inflammatory diseases, it may arise as a complication of a constitutional malady; the acute specific fevers in particular rendering a person vulnerable to the pneumococcus. When pneumonia runs an atypical course we should always bear in mind the possibility of the lung affection being only a complication of a constitutional disease such as typhoid fever.

Diagnosis.—Acute *pleurisy with effusion* is diagnosed from pneumonia by means of the data given in the table of diagnosis between consolidation of the lungs and fluid in the pleura (p. 138). *Broncho-pneumonia* runs a different course, and the signs are scattered over both lungs (see

table below). The sudden onset of acute pneumonia resembles that of *scarlet fever*, *erysipelas*, and *smallpox*, but the absence of rusty sputum and altered pulse-respiration ratio distinguishes them. There is a pneumonic form of *acute pulmonary tuberculosis* which has to be borne in mind (§ 87*a*) ; also various *aberrant forms of pneumonia* (§ 87*b*).

Table VII.—Differentiation between

	Lobar or Croupous Pneumonia.	Lobular or Broncho-pneumonia.
Onset	Sudden, with rigors ..	Gradual, and preceded by bronchitis.
Course of Temperature ..	Continuous	Remittent.
Defervescence ..	By crisis seventh day ..	By lysis in three to four weeks.
Percussion ..	Dulness in one lung, usually the base.	Scattered patches of dulness in both lungs.
Auscultation ..	(i.) Fine crepitations .. (ii.) Consolidation signs in a day or two.	(i.) Fine crepitations and consolidation signs over dull areas, though obscured by rhonchi and bronchitic râles.
Sputum ..	Rusty	Frothy and muco-purulent.
Respiration ..	Pulse-respiration ratio 2 : 1.	No marked difference of pulse-respiration ratio.

Prognosis.—The case mortality varies from 20 to 40 per cent. in hospital cases. The usual mode of termination is by heart failure. Much depends on the position and extent of the lesion, which is graver when both lungs are involved or when the disease attacks the apex. The reason for this is that apical pneumonia usually occurs in a lung already damaged by tubercle. It must, however, be remembered that small areas of consolidation may be associated with very great toxæmia. A lethal termination may be anticipated with marked cyanosis, a typhoid condition, scattered râles over both bases (indicating œdema), with lowered temperature. The absence of the usual increase in the leucocytes is also an unfavourable sign. It is graver at the extremes of life, in alcoholics and in debilitated persons ; but robust men in the prime of life often succumb, although the prognosis is generally stated to be good in healthy adults. As regards complications, meningitis is generally fatal, and endocarditis extremely grave. But of all conditions influencing the prognosis of lobar pneumonia chronic alcoholism is, in my belief, the worst.

Treatment.—There is at present no specific remedy for pneumonia, so that treatment is mainly expectant. Our chief endeavour should be to maintain the patient's strength, and to achieve this, rest in bed, good nursing, and visits by the doctor at least twice a day are essential. Patients treated near an open window have less dyspnœa and cyanosis, and sleep better than those treated in a vitiated atmosphere. They should be kept thoroughly warm by blankets and hot bottles. The

diet must be fluid, 2 to 3 pints of milk, into which may be stirred three or four eggs, being given in the twenty-four hours. *Sleep* is of such paramount importance that no patient should be allowed to spend a restless night. The cause for the restlessness should be sought for and treated. Frequently *pain* is the disturbing factor. This may be relieved by the local application of ice,[1] fomentation, or poultices, or a leech. Another cause of sleeplessness is *engorgement of the right heart.* In every case of pneumonia careful watch should be kept over the right heart. If the patient is blue and restless, the cardiac dulness increased considerably to the right, the liver enlarged, and the veins of the neck full, we should immediately relieve the right heart, either by venesection (about 5 ounces) or by applying six leeches to the skin over the liver. This extreme condition may be averted by the timely use of two or three leeches. *High pyrexia* may be the reason for sleeplessness, and may be reduced by tepid sponging, a measure which next to the relief of pain and engorgement of the right heart is the most satisfactory means of procuring sleep. If there is no obvious cause for the sleeplessness, hypnotics, such as trional, paraldehyde, or chloralamide may be given, but never chloral or sulphonal. Opium may be used in the early stages of the disease, and is often of the greatest value, its sedative effect more than counterbalancing its action as a cardiac depressant.

General and Cardiac Stimulants.—Strychnine should be injected hypodermically, beginning with 3 minims of liquor strychninæ every eight hours on the fourth day, gradually increasing, the dose according to circumstances, until the crisis is over. Digitalis is of less use, but may be given if there are signs of cardiac muscular weakness. Ammonium carbonate affords an additional and rapidly-acting stimulant. Concerning alcohol, there is much difference of opinion. It is particularly indicated in alcoholic patients, for whom it should be used freely, and in conditions of collapse near the crisis, when it may tide the patient over so that he is out of danger before the subsequent depressing effect of the drug becomes manifest. Atropine is helpful, but tends to increase the delirium. Oxygen inhalations should be tried for the respiratory distress. Vaccine treatment is being tried, but at present the results are too few to justify any conclusions as to its value.

§ 87*a*. **A Pneumonic Form of Acute Pulmonary Tuberculosis,** or pneumonic phthisis, is sometimes met with. The symptoms resemble those of pneumonia, and may start suddenly with a rapid rise of temperature and pain in the side. The temperature may continue high for a week or so. The physical signs also

[1] Dr. Lees claims that the ice-bag has a direct inhibitory action on the pneumonic process in the area of lung over which it is applied. Two ice-bags should be placed, one in front and one behind, directly on the skin. The legs must at the same time be kept warm by hot water bottles ("The Treatment of Some Acute Visceral Inflammations, and other Papers," 1904).

resemble those of pneumonia. It differs from this disease, however, in the presence of tubercle bacilli in the sputum, and the temperature, instead of falling abruptly by crisis about the seventh day, gradually becomes intermittent, and the *course of the disease* becomes indefinitely prolonged for weeks. This is followed by physical signs of breaking down, purulent expectoration, night sweats, and *generally death* in five to twelve weeks from exhaustion, hæmoptysis, or complications, such as pneumothorax (§ 94).

§ 87*b*. **Aberrant Acute Pneumonias** (Deuteropathic Pneumonia).—We have seen that in pleurisy, acute lobar pneumonia, and in other inflammatory diseases of the lungs, the course of the malady is fairly definite, and the physical signs in the lungs are characteristic. But it is important to remember that these same conditions may occur secondary to, and as part of, some general disorder. Under these circumstances some of the symptoms or physical signs may be wanting or irregular, and it may not be possible to arrive at a diagnosis, except by passing in review the whole history of the case, and by making a thorough and systematic examination of all the other organs. Instances of this eccentric group of pneumonias are met with secondary to enteric fever, cancer of the lung, acute glanders, anthrax, syphilis of the lung, and actinomycosis.

The practical outcome of these considerations is that when a case of pneumonia, or other apparently local inflammatory condition, is *irregular* in its physical signs or its clinical history, we probably have to do with a condition which is secondary to one of the conditions just mentioned, or to some general disease, such as enteric fever, scarlatina, pyæmia, or other general infective disorder.

III. *The illness has come on somewhat* GRADUALLY ; *there is cough, with frothy expectoration ; the physical signs of* CONSOLIDATION *are* SCATTERED *and accompanied by signs of bronchitis. The disease is probably* BRONCHO-PNEUMONIA.

§ **88. Acute Lobular Pneumonia**, or Broncho-pneumonia (catarrhal pneumonia), is also an acute parenchymatous inflammation of the lungs, but it runs a very different course to that of acute lobar pneumonia. The inflammatory process occurs in small patches, scattered unequally throughout both lungs, and it is accompanied by bronchitis : hence its name.

The *Constitutional Symptoms* come on more gradually in this disease. The temperature is remittent, about 100° F. in the mornings and 101° to 103° F. in the evenings, accompanied by cough, dyspnœa, and frothy sputum. The pulse is rapid, but the pulse-respiration ratio is not altered to anything like the extent of that in lobar pneumonia, and the face is generally pale instead of flushed. The fever is maintained by the fresh implication of neighbouring lobules for about three to six weeks or longer.

Physical Signs.—When the patches of consolidation are small, there may be no dulness on percussion, but only tubular breathing ; but when they are of moderate size, signs of consolidation (p. 138) can be made out. The chief auscultatory signs in children consist of *intensely loud,* " consonating," râles and rhonchi. There is a very serious form, in which small areas of consolidation are scattered throughout both lungs. The only signs are deficient air entry and numerous fine râles (capillary bronchitis).

Etiology.—Broncho-pneumonia occurs at all ages, but is *especially frequent in young children.* The cases fall into two groups, primary and secondary. Primary broncho-pneumonia, due to the pneumococcus, arises in much the same way as lobar pneumonia. Secondary forms arise: (i.) Complicating acute infections, such as measles, whooping-cough, diphtheria, smallpox, typhoid and scarlet fever; (ii.) complicating chronic debilitating conditions, such as chronic Bright's disease, chronic cardiac disease, or bed-lying, as from fracture of the femur in old people; (iii.) *aspiration* or *deglutition pneumonia*, such as occurs after operations on the tongue, mouth, or nose, in quinsy, cancer of the œsophagus communicating with the air-passages, bronchiectasis, and following hæmoptysis or the passage of food down an insensitive trachea, as in post-diphtheritic paralysis. A common but more chronic variety is of tuberculous origin.

Diagnosis.—*Chronic phthisis* is limited to the apex at first, and runs a characteristically chronic course. The pulmonary signs of *measles*, *whooping-cough*, and *bronchitis* resemble broncho-pneumonia in its early stages, and it may not be easy to diagnose these several diseases until the rash of the one or the whoop of the other appears. The constitutional symptoms in acute bronchitis are much less severe. The diagnosis from *acute miliary tuberculosis* may be very difficult, as sputum is usually not obtainable, but if it can be examined, the tubercle bacillus will be found. The diagnosis from *lobar pneumonia* is given in tabular form above (p. 153).

Prognosis.—The case mortality in children under five varies from 30 to 50 per cent. (Osler); the younger the child the more fatal is the disease. The strength of the patient and the duration of the disease are leading factors in the prognosis. If he is debilitated, especially if the environment is unfavourable, he soon becomes a prey to the tubercle bacillus, and the case rapidly runs on to phthisis (*q.v.*). Similarly, the longer the case lasts the more likely is it to have a fatal termination, and in adults this is very often the case. Broncho-pneumonia is nearly always secondary, and the third leading factor in the prognosis is the nature of the antecedent disease. When a child weakened by a *prolonged* fever is attacked, the prognosis is very grave, but after whooping-cough and measles it is much more favourable. Nevertheless, children often recover in apparently hopeless cases. The aspiration and deglutition pneumonias are usually fatal.

Treatment resembles that of lobar pneumonia, but stimulants are indicated from the outset of the disease; two drops of brandy for every month of an infant's age may be given every second hour. Children should be placed in a steam-tent, and small frequent doses of tr. belladonnæ administered. For adults the pain and incessant cough may require opium, best given as Dover's powder, and poultices applied to the back give considerable temporary relief. The chest afterwards may be covered with a cotton-wool jacket. If the symptoms become more distressing and the cough and dyspnœa increase, stimulating expectorants should be ordered; and if the cough continue difficult an emetic may be given. For the reduction of the hyperpyrexia, cold sponging may be adopted, especially if cerebral symptoms are present.

We now turn to the **acute disease with hyper-resonance on percussion** —viz., Pneumothorax. We must bear in mind that an acute disease may supervene upon a chronic condition accompanied by hyper-resonance—*e.g.*, when acute bronchitis supervenes on emphysema (see Table IX., p. 180).

The patient is in MARKED DISTRESS, *which has come on* SUDDENLY ; *on examining the chest the* BELL SOUND *is elicited. The disease is* PNEUMOTHORAX.

§ 89. **Pneumothorax** is a term applied to air in the pleural cavity, the air having gained admission by perforation of the pleura, either from within or from without. The air is almost invariably accompanied by pus, and the condition is then known as pyopneumothorax; if accompanied by serous effusion, as hydropneumothorax (Fig. 46).

The *Symptoms* of the onset of the condition differ according to the condition of the lung—*i.e.*, whether it is fairly healthy or is widely diseased. (*a*) When pneumothorax occurs in the less affected of the two lungs—the other side being extensively diseased—the symptoms are very urgent, and consist of severe pain in the side, attended by great dyspnœa, shallow, quick breathing, cyanosis, and some degree of collapse, with sweating, lividity, and a weak pulse. (*b*) In other cases, where pneumothorax comes on in a lung which is already much diseased, the onset may be hardly noticed.

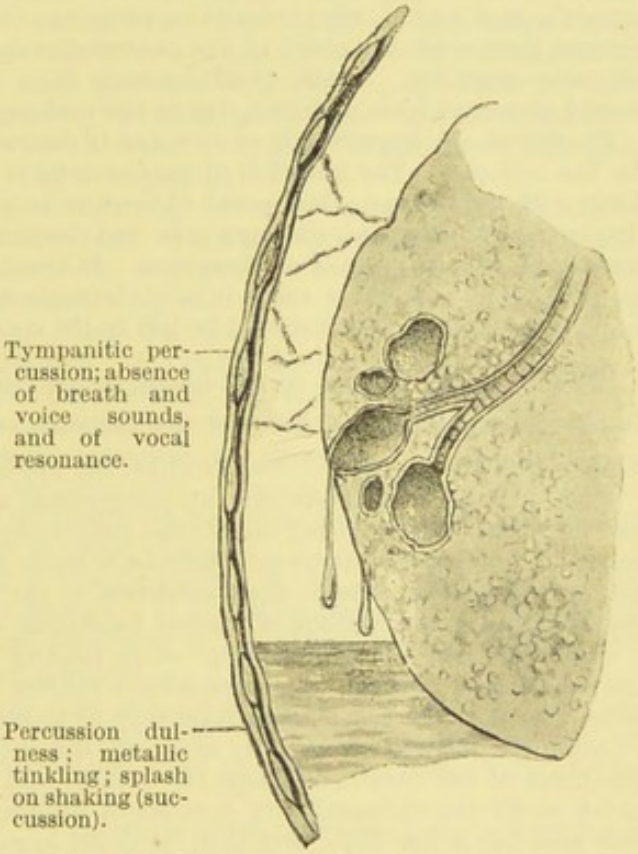

Fig. 46.—Diagram of HYDROPNEUMOTHORAX.

The *Physical Signs* consist of: (i.) A bulging on the affected side ; (ii.) diminished vocal fremitus ; (iii.) hyper-resonance on percussion (unless there is very great distension, when the note may be dull) ; (iv.) on auscultation the respiratory murmur may either be inaudible or amphoric ; the vocal resonance is usually diminished, but pectoriloquy and bronchophony are sometimes present. The *bell sound* may be elicited on tapping the chest with two coins in one position, and listening with a stethoscope in another. When fluid is also present, and this is usual, metallic tinkling is heard. The *Succussion Splash*, when it can be elicited without damage to the patient, is the most characteristic sign of hydropneumothorax—a fact which was well known to Hippocrates.[1] It may be obtained by placing one's ear against the chest, and shaking the patient's body to and fro.

Etiology.—(i.) Undoubtedly the commonest cause is advanced phthisis, when a cavity bursts into the pleura. (ii.) The converse process may take place in em-

[1] "De Morbis," lib. ii., cap. xvi.

pyema, when the pus bursts into the lung. (iii.) A fractured rib may lead to perforation of the pleura. (iv.) Less common causes are gangrene of the lung, abscess connected with the spine, or an ulcer of the stomach or œsophagus, bursting into the pleural cavity. (v.) It rarely happens in healthy people, although cases have been recorded.[1]

Prognosis.—The occurrence of pneumothorax is always very grave. It is difficult to estimate its case mortality, because death may be sometimes produced by the condition of the lung apart from the accident, but about half die within the first week, and some in a few hours, from shock or suffocation, when the lung on which the patient has been mainly dependent gives way. Only about 10 per cent. of all cases taken together ultimately recover. The *immediate* risk depends upon the urgency of the dyspnœa and cyanosis, the state of the other lung, the patient's general health, and the cause of the accident. As regards the *cause*, the pneumothorax that results from late phthisis or gangrene of the lung is very fatal; but that which occasionally complicates whooping-cough, pneumonia, early phthisis, and injury, often results in recovery. Certain it is that the longer the patient lives after the onset of the pneumothorax, the better is the prognosis for ultimate recovery. Death usually occurs from shock and suffocation, as mentioned above, or from asthenia, due to the prolonged discharge and lung disease.

Treatment.—A hypodermic of morphia is desirable for the pain, and stimulants for the collapse. The question of paracentesis is an important one. It has been done with advantage when great distension is present, as indicated by marked displacement of organs, extreme pain and discomfort. When pus is present, the treatment should be that of empyema. If clear fluid is present, aspiration may be performed. In other cases it is inadvisable to aspirate or operate, especially in phthisical cases, which should be left to the *vis medicatrix naturæ*.

There is one disease of the lungs which belongs neither to the acute nor to the chronic category, but is PAROXYSMAL, *occurring in attacks of sudden onset, usually* WITHOUT ELEVATION OF TEMPERATURE—ASTHMA.

§ **90. Asthma** is characterised by paroxysmal attacks of very severe dyspnœa, the inspiratory effort being short, the expiratory prolonged. It is accompanied by much lividity and distress. Asthma is liable to complicate chronic bronchitis, but it is important here to draw attention to the frequent error which is made by regarding all exacerbations of chronic bronchitis as asthma.

Symptoms and Clinical History.—The leading characteristic of this disease is its paroxysmal nature. A person who is subject to asthma may be perfectly well at one minute, and half an hour later may be seized with the most violent dyspnœa. It often commences in the early morning, the patient awakening with a feeling of tightness of the chest; he gasps for breath, and clings to surrounding objects in order to fix the extraordinary muscles of respiration. Each attack lasts from a few hours to a few days, and then, without apparent reason, the patient rapidly recovers his normal and healthy condition.

There are many curious and unexplained features in connection with this malady, one of the most interesting being the tendency to skin eruptions (especially urticaria and the various forms of erythema), and another the fact that these eruptions may alternate with the attacks of dyspnœa. Various other neuroses, and even attacks of insanity, may alternate in the same way. The paroxysms of asthma are occasionally preceded or ushered in by violent attacks of sneezing, by itching, or by the passing of large quantities of limpid urine. Sometimes an attack is terminated in this way.[2]

Physical Signs.—On inspection the chest is seen to be maintained in a position of inspiration, undergoing but little expansion with the short inspiratory efforts. The percussion note may be unaltered, but, after many attacks, emphysema

[1] Transactions of the Medical Society, 1897, vol. xx., p. 120.

[2] These facts point possibly to an infection of the general vaso-motor system similar to that in the pulmonary system which produces the spasmodic dyspnœa.

supervenes, with consequent hyper-resonance. On auscultation the short inspiratory effort is feeble and scarcely audible; expiration prolonged. Loud rhonchi replace the normal vesicular murmur, and often coarse râles, owing to the accompanying bronchitis.

Etiology.—Some regard the asthmatic attack as a series of spasmodic attempts on the part of the diaphragm, intercostal muscles, and extraordinary muscles of inspiration, to overcome some obstruction to the entry of air. But the central fact, which alone explains all the symptoms, is a narrowing of the bronchial tubes. This is probably due to spasm of the involuntary bronchial muscles, which is attended by hyperæmia of the submucosa and swelling of the mucous membrane. Some hold that the latter is the primary condition, and that the disease is therefore an angioneurosis.

Among the *predisposing* causes we find: (i.) A neurotic family history. Careful inquiry may reveal asthma or other neuroses, especially those so-called functional diseases of the nervous system connected with the involuntary muscular system, such as attacks of flushing and shivering, faints, and the like. (ii.) Asthma may occur at any *age*, but true asthma nearly always makes its first appearance soon after the age of puberty. (iii.) Any previous lung disease, especially chronic bronchitis, may predispose to asthma. Malaria, gout, and other constitutional conditions, are often associated with it. (iv.) Conditions of the nasal passages, such as ulceration, hypertrophic rhinitis or polypi.

Among the *exciting* causes of an attack may be mentioned: (i.) Certain atmospheric conditions which are ill-understood, and often appear to be most contradictory. Thus I know one patient who is free from asthma in London, but develops an attack immediately she seeks a high altitude. Another always develops an attack when she enters London. Some find the sea relieves them, others that a seaside place determines their attacks. (ii.) Reflex causes, such as derangement of the alimentary canal, and dietetic indiscretions (*e.g.*, the eating of cheese or fish), will often determine an attack; and so also will (iii.) dust and irritating particles.

Diagnosis.—The diagnosis usually presents no difficulty. The paroxysmal occurrence of the disease is quite characteristic. *Chronic Bright's disease* is manifested sometimes in its very earliest stages by paroxysms of dyspnœa resembling asthma. These are especially apt to occur at night.

Cardiac Asthma is a term which is loosely applied to the paroxysmal attacks of dyspnœa which accompany heart disease. It is differentiated from true asthma (i.) by its frequent recurrence, independently of the causes of true asthma, and (ii.) by the evidences of the cardiac mischief.

Prognosis.—The disease of itself does not shorten life, but tends to produce emphysema, bronchitis, and their attendant evils. Children may grow out of the disease; adults never lose it completely. The severity and frequency of the attacks are our only guides to prognosis.

Treatment—(*a*) *Before the Attack.*—Various remedies have been tried. Mentioned in the order in which I have found them most useful there are: tr. lobelia, ether, belladonna, hyoscyamus, opium in small doses, and pyridin, a remedy introduced and strongly advocated by Germain See. The diet during the attack should be the lightest possible; milk alone is best. Various inhalations are sometimes useful, either for the prevention or relief of an attack—*e.g.*, the vapour from a teaspoonful of turpentine and chloroform in equal parts, or the fumes of paper prepared with a strong solution of nitrate of potash, or the inhalation of amyl nitrite. If a mixture containing equal parts of the leaves of stramonium, lobelia, black tea, and potassium nitrate, be burnt in a tin plate, and the fumes be inhaled, much relief is usually afforded. Various other preparations, in the form of cigarettes of stramonium, potassium nitrate, and belladonna, are used. Experimentally on animals, I have found pilocarpin relaxes the bronchial muscles, and it would be worth trying, therefore, in asthma.

(*b*) *Between the Attacks.*—The effect of locality on the disease can only be ascertained by experience, and, as above mentioned, it is impossible to foretell what effect a particular climate will have. As a rule, though with many exceptions,

town air and fogs are detrimental. To prevent an attack, special attention should be directed to the diet. Light nourishing food should be advised, and the avoidance of solids after two o'clock in the day. Iodide of potassium, administered for a long period of time, certainly tends to ward off attacks in some patients, and potassium bromide at bedtime may act similarly. Arsenic also is very useful. The nose should be examined for polypi, etc., and these, as possible causes of irritation, must be removed. Some obtain great relief by using an atomiser to spray the nostrils. The chief virtue appears to lie in the fineness of the spray, but minute quantities of local anæsthetics, such as cocaine, are occasionally added.

CHRONIC DISEASES OF THE LUNGS AND PLEURÆ.

§ **91. Classification.**—Chronic disorders of the lungs and pleuræ may follow an acute attack of the conditions described in the previous sections, as when chronic bronchitis and emphysema succeed attacks of acute bronchitis. But many of the chronic diseases of the lungs, such as pulmonary tuberculosis, start insidiously, and attention may not be directed to the lungs for a considerable time.

The chronic diseases, like the acute, may be classified for clinical purposes, according to the results of percussion. It is convenient in actual practice, although unscientific, from the point of view of classification, to make a subsidiary group in which the sputum is of a highly offensive character.

(*a*) **Chronic Disease** in which the **Percussion Note is unaltered**

I. Chronic bronchitis .. .. § 93

(*b*) **Chronic Diseases** attended by **Dulness on Percussion,**

The *commoner* disorders presenting dulness in regular and *defined areas either at base or apex* are—

I. Chronic phthisis .. .. § 94
II. Hydrothorax .. .. § 95
III. Pulmonary congestion (or œdema of the lungs) .. § 96

The *rarer* diseases, having *irregular* and *scattered* areas of dulness are—

IV. Interstitial pneumonia .. .. § 97
V. Thickened pleura .. .. § 98
VI. Cancer and other neoplasms .. .. § 99
VII. Collapse of the lung tissue .. .. § 100
VIII. Syphilitic disease of the lung .. .. .. § 101
(IX. Mediastinal tumours) .. .. § 54

(*c*) **Chronic Diseases** attended by **Hyper-resonance**

I. Emphysema .. .. § 102
II. Pneumothorax[1] and various other conditions in which the hyper-resonance is not the leading or constant feature .. .. § 89

(*d*) **Diseases** recognised by the **Offensive Character of the Sputa** :

I. Bronchiectasis .. .. § 103
II. Gangrene of the lung .. .. § 104
III. Abscess of the lung .. .. § 105

[1] Pneumothorax sometimes comes on acutely, but it is more often part of a chronic disease.

§ **92. Method of Procedure.**—The routine examination is conducted as in acute disorders (§ 80)—viz., after ascertaining the leading symptom, and the history of the illness, we proceed to Inspection, Percussion, Auscultation, and Palpation. In percussion, remember to keep the hand flat and firmly pressed against the chest, while it is struck by one or more fingers of the other hand, used as a hammer, and with a staccato stroke. Remember also that the note is normally dull over the mammæ in most women, over the scapulæ in muscular men, and that it is slightly lower-pitched at the right than the left apex. The chest must, of course, be stripped.

Group A.—The patient's symptoms point to **chronic disease of the lungs**, and on examining the chest there is **no alteration in the percussion note.**

I. *The patient has a chronic cough ; there is no elevation of temperature, and on auscultation* rhonchi *and* râles *are heard over the chest. The disease is* Chronic Bronchitis.

93. Chronic Bronchitis is a chronic inflammation of the bronchial tubes. It may be chronic from the beginning, or it may supervene on repeated attacks of the acute disorder.

Symptoms.—A patient with chronic bronchitis and—its usual sequel—dilated right heart presents a typical appearance. Stout in build, with short, thick neck, of florid complexion, short of breath, wheezy respiration, and pulsating jugular veins, he presents an aspect which can be recognised at once. The clinical history extends over many years, with alternate diminution and aggravation of the symptoms. The cough is usually present during the winter, and improves as the weather gets warmer. The constant coughing and straining to bring up the secretion results sooner or later in generalised emphysema. In later stages the cough continues all the year round, and finally an attack of capillary bronchitis, œdema of the lung, or some intercurrent malady, throws a little extra strain upon the overburdened right heart, and death ensues. There are, as a rule, no febrile or constitutional symptoms.

The *Physical Signs* vary with the amount of secretion present, the amount of the complicating emphysema (§ 102), and bronchiectasis (§ 103). In cases of long duration the chest is barrel-shaped (emphysematous, § 74). Rhonchial fremitus may be felt on palpation. On percussion there is never any dulness, and the note is hyper-resonant in proportion to the emphysema present. On auscultation sibilant and sonorous rhonchi and bubbling râles can be heard ; and crepitations at the base, due to œdema, may be present.

There are four recognised varieties of this disease : (i.) *Bronchitis* with *winter cough*, attended by slight or abundant expectoration, mucous or muco-purulent, sometimes fibrinous, sometimes containing

streaks of blood. (ii.) *Dry Bronchitis* is attended by a frequent cough and soreness of the chest, but little or no secretion; it is of a very obstinate character, and occurs mostly in elderly people of a gouty diathesis. (iii.) *Bronchorrhœa* is recognised by the expectoration, which is of a thin, clear, or thick and ropy nature, very abundant and devoid of air. (iv.) *Fœtid Bronchitis* may occur in the later stages, and marks the onset of bronchiectasis. The sputum is very fœtid from time to time (see Bronchiectasis, § 103).

The *Diagnosis* of chronic bronchitis is not usually difficult. It may be readily diagnosed from *chronic phthisis* by the appearance of the patient, by the absence of hectic fever and emaciation, and by the absence of the tubercle bacillus from the sputum.

Etiology.—Chronic bronchitis may occur at any age, but is more common in elderly people. Sometimes, as before stated, it follows repeated attacks of acute bronchitis, but it may be chronic from the beginning. It often affects plethoric subjects, especially those of a gouty habit, and it is one of the recognised complications of Bright's disease. It is a frequent sequel to cardiac valvular disease, more especially disease of the mitral orifice. It may complicate other diseases of the lungs, especially phthisis, and may be a sequel of the acute specific fevers, especially measles and enteric fever.

Prognosis.—Patients with chronic bronchitis seldom entirely recover, though they may live for a great many years; and if the heart is fairly healthy and care be taken to avoid exposure, life is not very materially shortened. The co-existence of gout, Bright's disease, and cardio-vascular degeneration make the prognosis somewhat less favourable. The condition of the lungs is not so much a guide to prognosis as the condition of the heart. This, indeed, is the point around which the prognosis centres, and the untoward symptoms which render the prognosis grave are thus referable to the heart—viz., considerable dilatation of the right heart with evidences of cardiac failure, such as dropsy, rapid, irregular pulse, great breathlessness, and cyanosis (see § 50).

Treatment.—The extreme frequency of the disorder renders the treatment a matter of considerable importance. In severe cases the patient must be confined to one room at a uniform temperature of 62° F. day and night. When the mucous membrane is dry and irritable, a steam kettle gives great relief; it must be kept constantly going, not used intermittently. In slight cases, however, the patient can go about, but chill and exposure should be avoided. The important question of when a patient may go out must depend largely on the weather—cold and moisture, especially when in combination, are especially injurious.

The indications as to treatment are: (i.) To stimulate the relaxed mucous membrane with such remedies as am. carb., senega, squills,

etc. (ii.) When the cough is dry, soothing remedies, such as bromides, codeia, and tr. camph. co. should be given, or remedies directed to promote the secretion, such as ipecac., ammon. chlor., potass. iod., and alkalies (the last two especially in rheumatic or gouty cases), may be employed. (iii.) When the sputum is very abundant we should endeavour to diminish secretion by such remedies as the balsams (Tolu and Peru), tar preparations (creosote, guaiacol, petroleum), turpentine, camphor, senega, etc., given either internally or in the form of inhalations. For the latter, tar, creosote, and terebene may be used. Counter-irritants to the chest—*e.g.*, turpentine, camphor, or eucalyptus, are very popular with some. (iv.) When there is much spasm of the tubes, lobelia, iodide, and other asthmatic remedies are to be tried. (v.) Cardiac tonics and stimulants are called for sooner or later where dyspnœa and other cardiac symptoms are present. (vi). In cases with a gouty taint Ems water each morning, and small doses of iodide may be added to the other treatment, and the emunctories may be aided by sipping hot water morning and evening, and at intervals during the day.

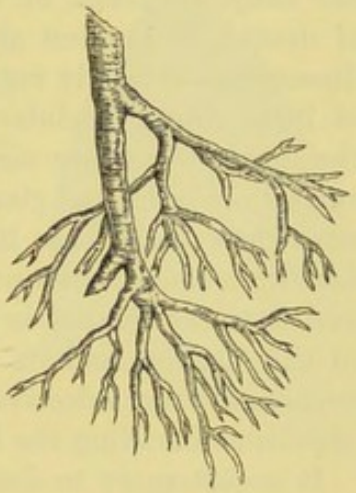

Fig. 47.—Bronchial Cast.

§ 93*a*. **Plastic Bronchitis** is inflammation of the bronchi, with the formation of fibro-plastic casts, which are expectorated.

Symptoms.—The symptoms consist of (i.) violent attacks of coughing, with expiratory dyspnœa, followed by (ii.) the expectoration of a fibrinous cast of a bronchus (*vide* Fig. 47). (iii.) The patient generally suffers from chronic bronchitis, and a little hæmoptysis may follow the expulsion of a cast. (iv.) Sometimes there are no constitutional symptoms, but slight pyrexia, and in some cases even rigors may be present. Such symptoms supervening in a case of chronic bronchitis lead us to suspect the condition.

Physical Signs may be absent. If present, they are those of an obstructed bronchus—an absent or diminished respiratory murmur, accompanied possibly by impaired percussion note. Whistling rhonchi or " flapping " sounds may be heard.

Causes.—The disease is twice as common in men as in women. It may occur at any age in subjects of chronic bronchitis.

Prognosis.—The condition is more serious than bronchitis. Two varieties have been described : (1) An acute form, lasting for some weeks ; and (2) a chronic form, occurring at intervals, for years, in the course of chronic bronchitis. Each attack may last for some weeks, and the casts be coughed up daily. The condition occasionally leads up to a fatal issue from dyspnœa, as when a large cast cannot be brought up.

The *Treatment* differs but little from that of bronchitis. The removal of the membrane may be promoted by the inhalation of lime-water, atomised by means of a spray, which is used with a view to dissolve the mucin in the cast. Various oils (*e.g.*, creosote oil, 1 in 40) have been injected as solvents, but the results have not been very promising.

Group B. — We now turn to those chronic diseases of the lungs which are accompanied by **dulness on percussion.** (*a*) The more *common*

diseases, in which the dulness occurs in regular and fairly DEFINED AREAS at base or apex, are: I. CHRONIC PULMONARY TUBERCULOSIS; II. HYDROTHORAX; and III. PULMONARY CONGESTION OR ŒDEMA.

I. *The patient complains of gradual emaciation and perhaps cough; on examination of the chest* SIGNS OF CONSOLIDATION *may be found, most marked at the* APEX *of the lung; there is* INTERMITTENT PYREXIA, *and the sputum may contain the tubercle bacillus. The disease is* CHRONIC PULMONARY TUBERCULOSIS (*Phthisis*).

§ **94. Chronic Pulmonary Tuberculosis** (Phthisis) may be defined as a wasting disorder due to tuberculosis of the lungs. The word phthisis is objectionable because it only indicates one of the symptoms—viz., the wasting (*φθίνω*, to waste). In view of the fact that this disease is the chief cause of death in Great Britain, the importance of the subject cannot be overestimated. The average annual number of deaths in London alone, for the decade 1889-98, was 8,453. The disease was formerly regarded as due to a kind of neoplasm, consisting of little round, nodular growths, "tubercles," scattered throughout the lungs, which are made up of a large number of small round cells, epithelioid cells, and giant cells. Owing to the discoveries of Koch we now know that these little nodules are only the inflammatory manifestations consequent on the irritation of a bacillus (the tubercle bacillus), and that the disease is primarily due to the ravages in the economy of this bacillus and its toxic products, and secondarily, to the supervention of other infective processes, and especially those due to pyogenic organisms invading the lungs.

It is customary to describe the anatomy in three stages. It is now generally believed that tuberculosis of the lungs begins as a tuberculous endo-bronchitis, due to the settling of the microbe in one of the smaller bronchial ramifications. It has been shown by Birch-Hirschfield, who took metallic castings of the bronchial tubes, that the reason the microbe settles at the apex is because in this situation there is, as it were, a "dead end," in which air is not so readily changed as in other situations. As a consequence, any dusty particles containing the bacillus which are inhaled and reach this situation, settle down, and there set up an irritation, resulting in a small localised ulceration of the mucous membrane. This corresponds to the generally accepted teaching that pulmonary tuberculosis is chiefly caused by the inhalation of tubercle bacilli. Dr. Whitla, however, believes that in most cases of phthisis the tubercle bacilli come primarily from the intestine.[1] The *congestion* which takes place around the primary foci constitutes (*a*) the first stage. (*b*) In the second stage there is considerable cell proliferation filling up the air cells and resulting in the formation of nodules consisting of granulomatous material in the neighbourhood of the primary mis-

[1] *Vide Lancet*, 1908, vol. ii., p. 186.

chief. This is the stage of *consolidation*. (*c*) The third stage is one of *breaking down*. Owing to the indolent character and low vitality of the new cell formation, it caseates and softens, becomes the seat of pyogenic organisms, and destruction of the air cells and formation of smaller or larger cavities results. Thus we have three stages : (*a*) Congestion ; (*b*) consolidation ; and (*c*) breaking down, with the formation of cavities (Fig. 48).

Symptoms.—The disease is essentially a chronic one, and its onset is very insidious. It is always more amenable to treatment in the early stage, and since the introduction of modern methods of treatment an early recognition of the disease has come to be of paramount importance.

(*a*) *Prodromal Stage.*—Phthisis has six modes of onset, which, in order of frequency, are as follows : (i.) Progressive weakness, attended perhaps by cough ; (ii.) hæmoptysis[1] ; (iii.) dyspepsia ; (iv.) laryngeal tuberculosis; (v.) dry pleurisy; (vi.) acute pneumonia (§ 87*a*), bronchitis, or broncho-pneumonia. Among the earlier *general symptoms* which should make us suspect the invasion of tubercle are unexplained debility, attended by languor and anæmia on the one hand ; or loss of weight, with unexplained dyspepsia, or slight elevations of temperature in the evening on the other. The temperature is an indication of the very greatest importance, for *no* ACTIVE *tuberculous process can take place in any part of the body without the occurrence of some pyrexia*, however slight. The type of this pyrexia is equally distinctive, for it is of an *intermittent character*, being normal in the morning and raised in the afternoon or at night; in rare instances this is reversed. If we have any suspicion of tubercle, the temperature should be taken every two hours, so that we may not miss any slight access of temperature during the day. Allbutt has pointed out that a premenstrual elevation of temperature sometimes occurs. In the early stage the patient may not be aware of the feverishness, though generally he feels a chilliness in the evening, and as the disease progresses, night sweats form one of its most characteristic features.

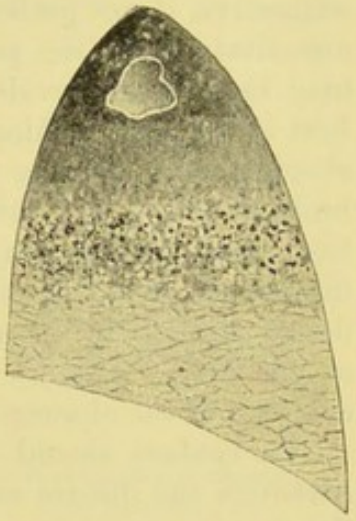

Fig. 48.—THE THREE STAGES OF PULMONARY TUBERCULOSIS, SOMETIMES PRESENT TOGETHER IN THE SAME LUNG.

[1] Early hæmoptysis of a very profuse kind may occur before any physical signs are discoverable.

The later symptoms of the disease are largely due to the action of organisms other than the tubercle bacillus. The clinical manifestations of these "mixed infections" are not always obvious, but many hold that whenever the temperature rises above 100·5° or 101° F. it is due to a superadded infection of this kind. It adds considerably to the gravity of a case, and its prevention, by fresh air, cleanliness, and a hygienic mode of life is important.

The *Physical Signs* accompanying the prodromal stage are necessarily somewhat vague and difficult to detect. The patient's chest should be thoroughly stripped, and he should be taken to a room where perfect quiet prevails; and if with the above symptoms we find weak or harsh breathing at one apex—especially if this is accompanied by an occasional single sibilant râle—we may be fairly certain that the disease is developing. Persistent inspiratory "sticky" clicks at one apex are very suggestive, if not pathognomonic of early phthisis. It is important to auscultate while the patient coughs, for râles not previously audible may thus become evident. The signs just named can often be heard best at the apex, behind, by placing the patient's hand on his opposite shoulder and listening to that part of the lung, just external to the bronchi, which will thus be *uncovered by the scapula*. Fine crepitations may be heard in that situation weeks before any signs can be discovered at the apex in front. Sometimes, later on, we are led to detect phthisis by an undue loudness of the *heart* sounds at the apex of one lung. Absence of dulness, like the absence of bacilli, is not evidence of the absence of tubercle.

The *sputum* should be repeatedly examined for tubercle bacilli. Although the disease can be diagnosed in their absence, by means of the above physical signs, it is always useful to employ this confirmatory test. The absence of bacilli, even after a series of examinations, does not indicate the absence of phthisis; but their presence establishes the disease beyond dispute. Another confirmatory test is the use of Koch's "old" tuberculin (see p. 172; and § 387), which, although not much approved of in England, is extensively used in Germany. It is followed by a definite reaction in phthisical subjects, even in the early stage. The opsonic index may be used as a means of diagnosis. One which is abnormally low, or which is abnormally high, or shows wide variations, indicates the presence of active tuberculosis somewhere in the body. The normal range is between 0·8 and 1·2. Calmette's and Von Pirquet's reactions are present in tuberculous subjects (*vide* Chapter XVI., § 405).

(*b*) The *stage of consolidation* and (*c*) the *stage of softening* and cavitation may be dealt with together. The symptoms, physical signs, and the corresponding lung changes are given in the form of a table for the purposes of convenience. The physical signs usually begin at the apex, and are generally best heard at the back, sometimes at the apex of the

lower lobe. From this position they extend downwards, and thus it is possible in the same patient to recognise in advanced cases the signs of the third stage, or cavitation, at the apex; below these, signs of consolidation; and below these, signs of congestion (as in Fig. 48). Such a condition indicates considerable activity. Many accessory signs may be mentioned: enlarged heart area due to retracted lung, hæmic heart murmurs due to anæmia, clubbed fingers in chronic cases of long duration, etc. Extensive tuberculous disease may sometimes exist with but little constitutional disturbance, and on the other hand, considerable disturbance of health may be present, without any abnormal physical signs—depending, partly, on the distance of the lesion from the surface of the lung.

TABLE VIII.—THREE STAGES OF PHTHISIS.

Anatomy. (See Fig. 48, p. 165.)	*Physical Signs.*	*Symptoms.*
(*a*) CONGESTION of lung tissue, consequent on invasion by tubercle bacilli.	At apex of the lung— (i.) Feeble R.M., with occasional fine crepitation heard at end of inspiration; or (ii.) Unduly harsh breathing, with a prolonged expiration.	(i.) Increasing languor on exertion; (ii.) Slight morning cough; (iii.) Slight rise of temperature; (iv.) In some cases hæmoptysis.
(*b*) CONSOLIDATION—due to the hyperplasia, cell infiltration, and the fusing together of the tuberculous foci.	Over diseased part, usually at apex, are: (i.) Impaired movement; (ii.) Flattening; (iii.) Increased vocal fremitus; (iv.) Dull percussion note; (v.) Bronchial or tubular breathing; (vi.) Bronchophony (increased V.R.).	(i.) Weakness and emaciation increase; (ii.) Temperature markedly higher in the evening; (iii.) Night sweats; (iv.) Anæmia.
(*c*) BREAKING DOWN and EXCAVATION.	Signs as in (*b*), *plus*— 1. Moist clicking râles; and later on— 2. Signs of presence of cavity: (i.) Cavernous breathing. (ii.) Post-tussic suction; (iii.) Pectoriloquy; (iv.) Râles with metallic tinkle.	All the above symptoms aggravated: (i.) Cough distressing, with quantities of nummular expectoration; hæmoptysis may be profuse; (ii.) Temperature high, and with wide range; (iii.) Sometimes diarrhœa, etc.

The presence or absence of A CAVITY is in the majority of cases impossible to diagnose with certainty. The percussion note is usually dull, but varies with circumstances. Thus the note is resonant when (i.) the cavity is large, or lies very superficially; and (ii.) there is not a great amount of consolidated lung tissue between the cavity and the chest wall. When the cavity is large and superficial, and the communicating bronchus remains patent, a characteristic note, almost tympanitic, is obtained on percussion whilst the patient keeps his mouth open. This is known as the "cracked-pot" sound (*bruit de pot fêlé*). Many attribute most importance to the sign known as "post-tussic suction." To elicit this sign, the stethoscope is applied over the suspected cavity, the patient is told to cough, and immediately after the cough a characteristic swishing sound is heard, due to the sucking of air into the cavity. This may be accompanied by copious râles.

The *Diagnosis* of the disease is not difficult except in the early stages, and in the absence of bacilli in the sputum. (i.) Various other causes of hæmoptysis may have to be differentiated (see § 73); (ii.) various other causes of anæmia may have to be eliminated (Chapter XVI.); (iii.) when the condition begins with dyspepsia it is very liable to be overlooked unless the physician is aware of this mode of commencement; (iv.) other causes of cough may be mistaken for phthisis (see § 70); (v.) various laryngeal affections may have to be eliminated (§ 119); (vi.) simple pleurisy is often impossible to distinguish from tuberculous pleurisy. According to many authors, 82 per cent. of apparently simple pleurisies with effusion are in reality tuberculous. (vii.) When it supervenes on bronchitis or broncho-pneumonia, our only clue consists in a delayed convalescence, together with the persistence of râles; and the fine clicking râles of phthisis are quite distinctive to the experienced ear. In the later stages of the disease the differentiation from the other cases of percussion dulness is not difficult (table, p. 160).

Etiology.—In phthisis, as in other microbic disorders, there are, on the one hand, predisposing causes which relate to the patient (*i.e.*, the soil on which the bacillus grows) and his powers of resistance; and on the other hand, exciting causes which relate to the microbe itself. If the "soil" is not suitable—*i.e.*, if the person is not predisposed by heredity or other cause, the bacillus will rarely grow. For these reasons its infectivity has been overlooked all these years. (1) Heredity is a potent cause, the individual being born with a predisposition to the disease. This factor, however, does not, as we shall see, occupy the prominent position which it was formerly believed to occupy. In a large proportion of cases no evidence of heredity is obtainable. (2) Both sexes are pretty equally affected, but the favourite age at which the disease usually supervenes is between twenty and thirty. The patient may be attacked at any time of life, although it is very rare under two years. (3) Any condition of malnutrition may produce a predisposition to the bacillus invasion, whether it arise from deficient food, from hyper-lactation, from exhausting diseases such as diabetes, or the acute specific fevers, after which an attack of phthisis is by no means infre-

quent. It is a curious circumstance that pregnant women are not prone to the disorder, and a phthisical subject becoming pregnant will often improve until after her confinement, when an exacerbation of the disease will occur, which has usually a fatal result. (4) Unhealthy surroundings play a most important part in the production of phthisis, and indoor occupations such as those of lace-makers and city clerks are specially unfavourable. (i.) A damp soil undoubtedly favours the production of the disease—even the dampness from faulty construction of a dwelling will do so. (ii.) A moist, hot atmosphere, such as exists in certain factories, favours the spread of the disease. (iii.) A dust-laden atmosphere, such as that of stonemasons, knife-grinders, tin and copper miners, fustian-cutters, is a potent cause of phthisis.

It is, however, an undoubted fact that mankind is naturally resistant to the tubercle bacillus. Birch-Hirschfield undertook a laborious investigation of 4,000 post-mortems, and he found that in 40 per cent. of these persons, dying from all manner of diseases, the lungs showed evidences of tubercle which had become quiescent and undergone spontaneous recovery. In view of these facts, and that most of the predisposing causes above mentioned are preventable, there is no reason why phthisis should not one day become as rare in England as leprosy is to-day.

Prognosis.—1. Usual *course* and *duration*. Phthisis is essentially a chronic but progressive disorder, and until recently nearly all cases applying for treatment terminated fatally. The death-rate from phthisis in 1838 was 38 per 1,000 ; in 1892, 14 per 1,000 living (Pollock, Discussion, Roy. Med. Chir. Soc., December 1899). Rapid cases may terminate in death in the course of three to six months. When the disease is indolent, and the patient resistant to the microbe, it may drag on for years. There are four chief modes of death, which in order of frequency are—(1) asthenia, (2) hæmoptysis, (3) asphyxia from pneumothorax, (4) the occurrence of other complications.

2. The prognosis in reference to *Causation* depends on : (i.) Heredity. Unquestionably it takes a more favourable course and the process tends to be less active in cases where there is no family history of tuberculosis. (ii.) The age of the patient influences the course considerably, for it is much more rapid in the young than in people over thirty. (iii.) The hygienic surroundings of a patient, as we shall see under treatment, make considerable difference to the course of the disease. Where the patient is well-to-do and can be removed from those conditions which have promoted the disease, he has a good chance nowadays of recovery ; but among the poor, who are forced to continue among their squalid surroundings and at their work, a fatal issue almost necessarily results. (iv.) Previous alcoholic excess diminishes the chance of recovery.

3. *Untoward Symptoms.*—(i.) Undoubtedly the most important feature is the temperature. Not only is an active tuberculosis evidenced by pyrexia, but the degree of fever, and still more the extent of the variations, are a fairly precise measure of the activity of the tuberculous process. (ii.) The condition of the lung is of course important. The presence of râles, as denoting softening and advancing disease, is unfavourable; and their disappearance favourable, but the extent of lung involved is as important a factor in prognosis. Thus, a man in the third stage, with an apex cavity in one lung, and little disease elsewhere, has a better chance of recovery than one with slight tuberculous foci scattered through the lung. If both lungs show disease in the third stage recovery is rare, though health has been restored in some cases after prolonged treatment. (iii.) The general symptoms also aid us in recognising the rate of progress. When the weight is increasing, the temperature declining, and food is taken well, the chances of recovery are good. (iv.) Early hæmoptysis does not affect the prognosis in any way, but occurring later in any quantity is apt to weaken the patient considerably.

4. *Complications.*—The presence of complications is undoubtedly bad. The commonest complications are: (1) Pleurisy, which is very frequent, but is often of a conservative nature, for adhesions may sometimes prevent pneumothorax; (2) tubercle may occur in other parts—the peritoneum, meninges, and especially in the intestine, giving rise to ulceration and an exhausting diarrhœa[1]; (3) the larynx may be affected either previously or subsequently, and undoubtedly it adversely influences the prognosis; (4) lardaceous disease of the liver, spleen, and other organs frequently ensues, and this again constitutes an unfavourable element, as it cannot be removed; (5) pneumothorax and pyopneumothorax may ensue from the bursting of a cavity into the pleura—fatal asphyxia *may* result (§ 89); (6) thrombosis of various veins is a less common complication; (7) peripheral neuritis is now a recognised occurrence, sometimes very early in the disease; (8) vomiting (see p. 171).

It is a good rule never to commit yourself to an opinion on any case of phthisis without first noting the effects of treatment.

Treatment of Phthisis.—The subject of treatment will be dealt with under four headings: (*a*) remedial; (*b*) symptomatic treatment; (*c*) treatment by tuberculin; and (*d*) the open-air treatment. The indications of all treatment are to reduce the inflammation, to destroy the virus, to build up the strength, and to palliate the symptoms.

(*a*) The *Remedial Treatment* formerly in vogue was mainly directed to building up the strength by means of cod-liver oil, maltine, hypophosphites, and other tonics. These are still useful except when there

[1] Diarrhœa may also occur as part of the hectic fever without any ulceration of the bowels.

is considerable fever present, or there are evidences of rapid breaking down. Guaiacol, 20 grains (gradually increased), creosote, thymol, eucalyptus, and other antiseptics may be given at any time ; and a few years ago these were also administered by some as an injection into the lung. Perhaps the best of this kind of treatment is the use of a spray four times a day, lasting fifteen minutes, of formalin.[1] Inhalation of antiseptics may be administered by Coghill's respirator.

Counter-irritants were largely used to reduce the inflammation, the favourites being iodine or croton oil applied over the apex of the lung. These measures were supplemented in wealthier patients by sea-voyages, high, dry mountain air, and residence abroad during the winter, combined with a liberal dietary and general hygienic mode of life.[2] These various methods are still useful, but at the present time we have two powerful means of combating this lethal disease : viz., Koch's tuberculin, and the open-air treatment ; see (*c*) and (*d*) below.

(*b*) *Symptomatic Treatment.*—It will be seen that in the third stage there is not much hope of recovery, but even in the worst cases we can ameliorate the symptoms, and so ease the passage to the grave. (1) For the cough, tinct. camph. co. and other expectorants are not of much use. The best cough mixture is one containing liquor morphinæ, or, better still, codeia in small doses with dilute sulphuric acid. Warm alkaline drinks promote expectoration. (2) Night sweats, which are often very profuse and exhausting, may be combated by atropine, zinc oxide, picrotoxin, and strychnine, especially the first named. Night sweats are said to be seldom troublesome if there be free exposure to fresh air. (3) The diarrhœa is also very exhausting, and must be combated with catechu, opium, intestinal disinfectants, and mineral acids. (4) Pleuritic pains may be eased by stupes, or painting with tincture of iodine. (5) The concurrent dyspepsia must be combated in the usual way, but the vomiting is often a very troublesome symptom, and there are three kinds of vomiting which admit of three different methods of treatment. (*a*) If preceded by nausea, it points to disorder of the stomach, and should be treated by bismuth, etc., on the usual lines. (*b*) If the vomiting be preceded and caused by coughing, it is a good plan to give hot drinks just before a meal, in order to encourage the expectoration and get the paroxysms of coughing over before the meal

[1] Dr. Lardner Green recommends the following formula (Formalin = 40 per cent. of Formic Aldehyde) : Formalin, ℥i. ; Glycerine, ℥iv. ; Aq. Dest., ℥v. Use as spray four times a day, fifteen minutes at each inhalation (*Lancet*, August 19, 1899, p. 521).

[2] Simple respiratory exercises, such as the following, designed to expand the chest, form a useful adjunct : (i.) With the back against the wall, fully extend the arms to the level of the shoulders slowly for eight times. (ii.) Continue the same movements until the arms meet above the head. (iii.) Start with the hands above and in front of the head, and bring them slowly down until the backs of the hands meet behind the body, at the level of the buttocks, the arms being rigid all the while. These should be done twice daily, gradually increased to five to six times daily.

is commenced. (*c*) If neither of these causes can be traced, the vomiting is probably due to irritation of the vagus, and may sometimes be relieved by opium. Sometimes vomiting is controlled by the will. (6) The treatment of hæmoptysis, pneumothorax, and laryngeal ulceration are dealt with elsewhere. (7) Now that the microbic origin of tubercle is established, the question of how far it is a contagious disorder is keenly debated, but whether all cases take origin directly, or indirectly, from previous cases or not, true it is that if there were no bacilli there would be no consumption. Our preventive measures must therefore be directed (1) to the destruction of the microbe, and (2) to render those liable to the disease by heredity, resistant to the disease by a perfect state of health. The measures for the destruction of the bacillus are given in textbooks on hygiene, but, briefly, the means consist of: (1) The destruction of the sputum by collecting it in a disinfecting medium; and (2) the thorough disinfection of rooms occupied by phthisical persons. The larger preventive measures of stamping out the disease from a community are of the highest importance. How much improvement hygiene can affect in this respect is shown by the marked fall in the death-rate from phthisis.

(*c*) *Treatment by Tuberculin* depends upon the principle of immunisation (Chapter XV.). Koch followed his discovery of the tubercle bacillus by soon afterwards issuing to the world the toxin produced by the bacillus. This he called tuberculin, and it is now called the *old tuberculin*. It was administered hypodermically and produced considerable "reaction"—*i.e.*, constitutional disturbance—in the patient. It was largely used, especially in cases of lupus, but the results were not satisfactory. Since then a *new tuberculin* has been issued by Koch, which is not free from danger, for as sold it contains active bacilli, which may produce general blood infection. The opsonic method has again put this line of treatment on its trial in the treatment of phthisis, as for other tuberculous disease, but a *bacillary emulsion* is now used, and the dose is considerably smaller than that formerly administered (see Immunisation, §§ 386 and 387).

(*d*) The "*open-air*," hygienic, or sanatorium treatment of phthisis, as it is now called, is not altogether a new method, for fresh air has always been advocated as advantageous to these patients. But the systematic open-air treatment as first established at Nordrach, and more recently in Britain, is comparatively modern, but there are now numerous sanatoria devoted entirely to this plan of treatment. Much discussion has taken place as to whether it cannot be carried out without a sanatorium.[1] Among the well-to-do, perhaps, a sanatorium is not indispensable, but in the middle and lower classes the necessary discipline cannot be otherwise carried out. That residence in a sanatorium is not absolutely necessary is evidenced by cases which have been under

[1] Discussion, Roy. Med. Chir. Soc., December, 1899.

my care, even in an advanced stage of phthisis, who were unable to go away. One of them spent all the daytime in Kensington Gardens, in all weathers, and when indoors the windows were always open. This patient recovered in six months.[1]

Briefly, the *advantages* gained by this method of treatment consist of: (i.) Increased medical supervision from day to day and hour to hour by the medical officer of the sanatorium; (ii.) the continuous exposure of the patient to fresh, pure air, night and day, the windows never being shut, and sometimes wholly removed; (iii.) systematic exercise in suitable cases; (iv.) the ingestion of a large amount of suitable food; (v.) a suitable amount of rest during the fever stage, and a freedom from excitement; (vi.) the avoidance of mixed infections by hygienic mode of life. Cleanliness and fresh air tend to obviate pyogenic processes and infections. All possibility of the introduction of influenza and other infective disorders should be avoided by the *proper regulation of visitors* to patients. I believe that some day these latter will be subjected to the most rigorous scrutiny and inquiry before being allowed to come in contact with the consumptive patients in a sanatorium. The mixed or superadded infections do more harm than the tubercle bacillus. This is probably the reason why tuberculous patients do so badly in the wards of a general hospital.

The possible *disadvantages* urged are: (i.) The fear of hyper-medication that may go on in sanatoria; and (ii.) certain unsuitable cases (see below) may be deleteriously affected.

In carrying out this treatment, seven rules should be observed:

(1) Much depends on the suitability of the case, and the *earlier the stage* the better. There are three conditions in which the sanatorium, or open-air treatment, is undesirable: (i.) When the process is too active, as evidenced by a high and wide range of temperature[2]; (ii.) when the lungs are too far destroyed; and (iii.) when the case is attended by active bronchial catarrh.

(2) The food must be abundant, and the cuisine appetising and attractive.[3] But here an important caution comes in, else the patient puts on fat without influencing the disease. The food must be in proportion to the exercise, and the patient's weight should never much exceed his previously normal weight. The proteid foods should be increased relatively to the farinaceous, otherwise the patient becomes plethoric and breathless.

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[1] See also a case reported in the *Lancet*, January 20, 1900.

[2] Some do not regard this as a contra-indication to open-air treatment; but in such cases the length of the journey has to be considered, and the undesirability of mixing such patients with others.

[3] Thirteen pints of milk, or its equivalent, is in most institutions adopted as a fair standard of diet; that is, 9 ounces of proteid, 7¾ ounces of fat, 10½ ounces of carbohydrate: total, 27¼ ounces water-free food. With "Parkes' Hygiene" percentage composition tables, varying diets can easily be made up containing the above proportion of food elements.

(3) Evidences of benefit should be carefully looked for. They are three in number : (i.) A lowering of the temperature and a lessening of its range ; (ii.) an increase in the appetite ; (iii.) increase of weight *combined* with the two previous features.

(4) In deciding the important question of rest or exercise, the great value of accurate temperature records is again seen. When the highest daily record exceeds 102° F., the patient should be confined to bed. If the highest record exceeds 100·5° F., he should only be allowed to move about 100 yards at a time. If it does not exceed 100·5° F., then daily outdoor exercise should be enforced, and especially slow uphill walking ; for this strengthens the heart, expands the lungs, and improves the tone of the body generally.

(5) Amusement is necessary, but it requires to be carefully regulated. The patient should not talk too much, and any excitement or heated discussion is bad. *The whole day, and, if possible, the night also, should be spent out of doors, no matter what the weather may be,* and outdoor amusement cultivated. A very useful contrivance is the *liegehalle*, which is practically a small revolving summer-house, the front of which is open, and can be turned away from the wind.

(6) The duration of the treatment must be sufficient, and should be continued for some time after all symptoms have disappeared. According to Clifford Allbutt (" Discussion on Tuberculosis," *British Medical Journal*, October 28, 1899), if the case is only in the first stage, cure may be accomplished in six months ; if the second stage is reached (consolidation), cure can only be accomplished after a disciplinary treatment of eighteen months ; and in the third stage cure is rare, but is possible if the treatment be carried over two or three years.

(7) The hygiene and the locality of the building are important matters, but the reader must refer to special works for this.

§ 94*a*. **Fibroid Phthisis** is one of the least common of the varieties of pulmonary tuberculosis. It may be defined as a tuberculo-fibroid disease of the lungs, occurring for the most part in elderly subjects, running a protracted course, and terminating in contraction of the lung. This disease is very apt to be confused with chronic interstitial pneumonia or cirrhosis of the lung (§ 97).[1]

Symptoms.—The disease is essentially one of insidious onset and long duration. The patient complains of a chronic cough for many years. Later on this may become paroxysmal, and especially troublesome in the morning. Progressive shortness of breath, clubbed fingers, slowly increasing weakness and emaciation, with little or no fever, constitute the other symptoms.

The *Physical Signs* begin and are almost always most marked at the apex. *Both lungs* are usually affected (which contrasts with interstitial pneumonia), but the signs of disease are afterwards more advanced on one side. There is impairment of the chest movement, and later on contraction of one side of the chest. The area of præcordial dulness is increased when the left lung is involved ; and the heart and other viscera may be displaced. The signs of consolidation, with gradual softening, may also be present. Hæmoptysis sometimes occurs, and the tubercle bacillus may be discovered on careful and repeated examination of the sputum.

[1] Reference to chronic interstitial pneumonia (§ 97) will show to what condition the term " fibroid phthisis " should be confined.

The *Diagnosis* from other forms of *phthisis* is made by the extremely protracted course of this disease and the age of the patient. *Chronic interstitial pneumonia* resembles it very closely, both in its physical signs and symptoms, and the diagnosis of interstitial pneumonia can only be inferred (i.) from the absence of the tubercle bacillus after oft-repeated examinations, and (ii.) from the more usual localisation in one lung.

Etiology.—Fibroid phthisis is more frequently met with at and after middle life. It may follow chronic bronchitis, broncho-pneumonia, or repeated attacks of pleurisy. In true Fibroid Phthisis the tubercle bacillus is primarily deposited in a healthy lung under the same circumstances as in chronic pulmonary tuberculosis, and then causes an indolent fibroid reaction. On the other hand, chronic interstitial pneumonia may become the seat of tuberculous invasion, and in that case the causes of chronic interstitial pneumonia are the causes of fibroid phthisis (see Interstitial Pneumonia, § 97).

Prognosis.—Its course is very indefinite and protracted. Sometimes acute tuberculosis supervenes. The chief complications are bronchiectasis, compensatory emphysema of the lungs, lardaceous disease of other organs, and cardiac failure. In general terms the prognosis depends upon the same conditions as those of pulmonary tuberculosis, and the *Treatment* is the same.

II. *The patient complains of breathlessness; on examining the chest,* **dulness** *is found at one or both* **bases,** *and* SIGNS OF FLUID *are detected there. The disease is* HYDROTHORAX.

§ **95. Hydrothorax** is a chronic collection of serous fluid in the pleural cavity, differing from the effusion of pleurisy in being non-inflammatory. The surfaces of the pleuræ are smooth, the fluid is free from flakes, and generally alters its level with the position of the patient.

Symptoms.—The general symptoms may be but little marked if the fluid is small in quantity. The onset is usually gradual. Dyspnœa is generally present, especially on exercise, but its degree depends upon the amount of fluid. As hydrothorax is always a secondary condition, the symptoms may be masked by the presence of dropsy elsewhere; and it is remarkable how often hydrothorax is overlooked on this account. In rare cases the fluid collects with great rapidity, as in a fatal case of double hydrothorax supervening on scarlatina, mentioned by Osler.[1]

The *Physical Signs* are those of fluid in the chest (*vide* § 77). The level of the fluid in hydrothorax, unless excessive in quantity, moves when the patient alters his position, thus differing from the inflammatory fluid of acute pleurisy. This is an important diagnostic feature which can always be elicited, except when the fluid is confined by adhesions.

Diagnosis.—The disease has to be diagnosed from other disorders giving rise to dulness on percussion (p. 160). As regards *pleurisy*, in addition to the mobility of the fluid, hydrothorax is distinguished by the absence of pyrexia at the onset, by the absence of pain, and by the fact that the fluid occurs usually on both sides. The use of the aspirating needle settles the diagnosis from *consolidation* (see also Table V., § 77).

Etiology.—(i.) Hydrothorax may form part of the *general* dropsy of Bright's disease, in which circumstances both pleuræ are involved. Here the hydrothorax is of no very great importance *per se*, but the onset of dyspnœa in Bright's disease should always direct our attention to the pleuræ. (ii.) Similarly, it may form part of *cardiac* dropsy, in which circumstances one pleura is often solely or chiefly affected. (iii.) New growths in the pleura are generally attended by hydrothorax. This is especially so in the case of carcinoma, which should always be suspected in the aged. In this case the fluid is blood-stained, and may be found to contain cancer cells. In tubercle there is rarely much fluid in the pleura, adhesions being

[1] Osler, "Prin. and Pract. of Med.," second edition, p. 608.

more common. (iv.) Aneurysm or other intrathoracic tumours pressing on the veins of the thorax may give rise to hydrothorax on one or both sides.

Prognosis.—The disease is essentially chronic, and runs a prolonged and indefinite course, the duration depending very much upon the cause. In general terms the prognosis of the condition is favourable, unless due to new growths. The patient should be carefully watched for the occurrence of shivering, sweating, or intermitting pyrexia, as indicative of empyema. The sudden onset of signs of fluid in the chest, accompanied by shock or collapse, in a case which has previously presented the symptoms of aneurysm, points to the occurrence of hæmorrhage into the pleural cavity.

Treatment.—The treatment is comparatively simple. The administration of brisk hydragogue purgatives will generally reduce the amount of fluid; if this fails, or if the fluid return, or in any case where dyspnœa is extreme, paracentesis (p. 149) should be resorted to. The operation of tapping may be repeated indefinitely. Diuretics or cardiac stimulants are useful. For the rest, the treatment must be directed to the primary condition (see also § 85).

III. *The patient complains of breathlessness; on examining the chest,* **dulness** *is found at one or both* **bases,** *and on auscultation,* FINE CREPITATIONS *are heard. The disease is* PULMONARY CONGESTION OR ŒDEMA.

§ **96. Œdema of the Lung** (Pulmonary Congestion).—Œdema of the lung is a serous exudation into and around the air vesicles. It is synonymous with the term "hypostatic congestion," or, as it is sometimes called, "hypostatic pneumonia." It determines the end of many serious disorders.

Symptoms.—(i.) It is never a primary condition, and therefore our attention is first directed to the symptoms of its cause. The advent of hypostatic congestion is always insidious, and it is only by careful watching that it can be detected. (ii.) A considerable amount of dyspnœa is present, which may amount to orthopnœa. (iii.) There is an abundant frothy mucous expectoration, not infrequently tinged with blood.

The *Physical Signs* are somewhat indefinite, but they are found, as is implied by the term "hypostatic," chiefly at the bases of both lungs. The percussion note is somewhat impaired, and the air entry at the bases is diminished, and is attended by abundant moist crepitations.

Diagnosis.—The condition is diagnosed from true pneumonia by the gradual onset, the indefinite signs, and the absence, for the most part, of pyrexia and other constitutional symptoms. Any rise of temperature that may be present is due to the primary or causal condition.

Etiology.—(i.) The disease is most frequently met with in elderly people. (ii.) Pulmonary œdema complicates various blood disorders and fevers, especially typhus and typhoid fevers. The latter, indeed, is so frequently complicated in this way that hypostatic congestion is an aid to the diagnosis in the second and third weeks of the disease. In Bright's disease and anæmia, œdema of the lungs occurs as part of a generalised dropsy. (iii.) Cardiac and other diseases, leading to mechanical dropsy, produce œdema of the lungs. (iv.) Tumours pressing on the veins within the mediastinum may result in pulmonary œdema.

Prognosis.—The prognosis is always grave, because pulmonary œdema indicates either considerable impediment to the circulation in the lungs, or a serious toxic condition of the blood. It frequently terminates life in circulatory disorders, and in specific fevers of the asthenic type. In pneumonia it heralds a fatal issue. Œdema of the lungs is one of the most constant lesions found in typhoid fever. The extent of the œdema is indicated very fairly by the degree of dyspnœa.

Treatment.—The indications are to relieve the cause, if possible, and to stimulate the heart. Ammonium carbonate and other stimulating expectorants aid the heart and promote expectoration. The liberal administration of alcohol and other diffusible stimulants is called for. In the aged, among whom even slight disorders are apt to be attended by pulmonary œdema, it is well *to keep the patient*

propped up in a semi-recumbent posture. For the same reason it is advisable, in cases of fracture and other surgical maladies in the aged, to get them up as soon as possible, even at the risk of doing harm to their surgical ailment, so as to obviate the occurrence of hypostatic congestion.

GROUP B.—*We now turn to the* **rarer** *chronic diseases attended by* **dulness** *on percussion, in which the dulness occurs in irregular and* **scattered areas** : IV. INTERSTITIAL PNEUMONIA ; V. THICKENED PLEURA ; VI. CANCER AND OTHER TUMOURS ; VII. COLLAPSE ; VIII. SYPHILITIC DISEASE ; and IX. MEDIASTINAL TUMOURS.

§ **97. Chronic Interstitial Pneumonia**—apart from that form due to the malign effects of certain trades—is a rare disease. It may be defined as a chronic interstitial fibrosis of the lung, localised or diffuse, according to the variety, running a protracted course, and resulting in contraction of the pulmonary tissue.

An increase of the fibrous tissue of the lung may take place under the following conditions, all being chronic processes :

(i.) An indolent tuberculous process may assume a fibroid character. Fibrosis is one of the ordinary terminations of a tuberculous focus ; but when the progress is very slow and protracted, with excessive formation of fibrous tissue, it constitutes true *fibroid phthisis.*

(ii.) The constant inhalation of dust in certain trades (*e.g.*, fustian cutters, jute workers, wool-sorters, stone, knife, and other grinders and polishers, iron and coal miners, etc.) gives rise to a *chronic broncho-pneumonia*, followed by a peribronchial fibrosis, which later on involves considerable areas of lung tissue.

(iii.) Repeated attacks of *pleurisy* may be attended by a subpleural fibrosis (thickened pleura), and dense bands of fibrous tissue may extend into the lung (Sir Andrew Clark).

(iv.) *Acute broncho-pneumonia*, becoming chronic, may, although very rarely, result in an interstitial fibrosis. This form very often terminates by becoming tuberculous.

(v.) An *acute lobar pneumonia*, similarly, may assume a chronic course, and may result in an interstitial fibrosis (Addison). This form has not the same tendency to become tuberculous.

(vi.) *Syphilitic disease* of the lung is rare, except as a congenital manifestation in infancy, in which circumstances the change consists of a fibroid induration of the lung (Kingston Fowler).

All these may become the seat of tuberculous disease, but only the first, which is a *tuberculo-fibroid* process, should be called " fibroid phthisis." The other varieties constitute cirrhosis of the lung, and if they are invaded by the tubercle bacillus, they form a *fibro-tuberculous* process, which in its later stages may be indistinguishable from fibroid phthisis.

The general *Symptoms* consist of progressive weakness and dyspnœa. There is no fever unless there is ulceration of the bronchi or septicæmia—a common occurrence in late stages of the disease.

The *Physical Signs* may be found either at the base or the apex, though usually the former. Except in the variety due to the inhalation of irritating particles, only *one lung* is involved, thus differing from fibroid phthisis, in which both lungs are usually affected. There is deficient mobility of the diseased side, which later on undergoes contraction, so that there may be considerable difference in the measurement of the two sides of the chest. There is dulness on percussion. On auscultation, bubbling râles may be heard, but sometimes the only symptom is weak bronchial breathing or a weak respiratory murmur. The expectoration sometimes contains blood, but never the tubercle bacillus.

The *Diagnosis* of interstitial pneumonia from fibroid phthisis is sometimes very difficult, as may be seen in the description of the various processes just named. It is also liable to be mistaken for empyema.

The *Etiology* of the condition is given above. It is met with chiefly in male subjects under the age of fifty—especially between fifteen and thirty. Alcoholism predisposes. The commonest form of chronic interstitial pneumonia is that met with in persons engaged in trades attended by the inhalation of irritating particles. Sometimes it is a sequel to other pulmonary disorders.

Prognosis.—The prognosis is serious, because nothing will remove the fibrous tissue. As regards the duration of life, the prognosis is good if the patient is not losing weight and the disease is not too extensive. The *complications* are bronchiectasis, a very frequent sequel, dilated right heart, and emphysema occurring in other parts of the lung. When ulceration of the bronchi has taken place, lardaceous disease and septicæmia may ensue.

Treatment.—Counter-irritation and respiratory exercises are given on the lines advised in Chronic Phthisis (§ 94). F. 61 is useful.

§ **98. Thickened Pleura** is a condition which sometimes succeeds dry pleurisy, especially recurrent dry pleurisy. It is important to be able to recognise it, lest it should be mistaken for some more serious condition, though it is somewhat difficult to do so. It is more often localised to one part, and that most commonly at the apex associated with chronic phthisis.

The *Symptoms* of thickened pleura are: (i.) A localised enfeeblement of the respiratory murmur; (ii.) dulness on percussion; and (iii.) a slight diminution in the vocal resonance and fremitus.

The *Diagnosis* is arrived at (i.) by the history of the case—*e.g.*, there has been an attack of pleurisy or pneumonia in the past—and (ii.) by the absence of signs of active disease when the patient is kept for some time under observation. The condition is often discovered only by chance, when the patient seeks advice for other ailments.

Treatment is of no avail; and, if only moderate in degree, the disease is not of much consequence. Counter-irritation may be applied.

§ **99. Malignant Disease of the Lung.**—Cancer of the lung is rarely a primary condition, but is most frequently secondary to cancer of the breast, liver, or spleen. It occurs in two forms—(*a*) hard, nodular, isolated masses, when it is probably scirrhus or epithelioma; (*b*) a soft, irregular infiltration, probably encephaloid cancer. The evidences of the former are usually more distinct than those of the latter.

Symptoms.—The lung trouble may be preceded by signs of malignant disease elsewhere. The first evidence of involvement of the lung is breathlessness. This is followed by cough and by expectoration, which may from time to time be tinged with blood. Pain is often present, and indicates generally that the pleura is invaded, in which case there is usually a certain amount of pleuritic (blood-stained) effusion.

The *Physical Signs* are often very indefinite. Clinically, there are two forms—(*a*) The *nodular* form is usually attended by serous effusion (see Hydrothorax, § 95). Effusion into the pleura coming on slowly, or returning persistently, in an elderly person, is of itself suspicious, and the diagnosis is confirmed when, on aspiration, the fluid is found to be blood-stained. Sometimes in the midst of what appears to be a hydrothorax we detect the signs of consolidation. This probably indicates that the neoplasm has come to the surface in one locality.

(*b*) With the *infiltrated* form we find signs of consolidation, accompanied later on by the moist sounds due to the breaking down of the growth. Here again nearly every variety of physical sign may be met with in different parts of the lung, and if the main bronchus be obstructed, there is entire absence of the breath sounds.

Diagnosis.—The condition has to be diagnosed from different forms of pneumonia, from pleurisy with effusion, and from hydrothorax. The age of the patient, the chronic course of the affection, the absence (usually) of pyrexia, and the presence of cachexia, should enable us to come to a conclusion.

Prognosis.—The question is one of duration, and this can only be gauged by

daily observation of the case, and by noting the rate at which the growth appears to be spreading. Death usually occurs in about six months.

The *Treatment* resolves itself into the relief of pain and the amelioration of other symptoms which may be present.

§ 100. Atelectasis, or **Collapse of the Lung,** is a condition in which the lung tissue is in an unexpanded state. The term "atelectasis" is usually applied to lung tissue which has never properly expanded, and is therefore a congenital condition, due to imperfect development. The term "collapse of the lung" is applied to lung tissue which has previously expanded, but in which the air vesicles have subsequently collapsed.

Atelectasis is a *congenital* condition, of which symptoms occur in the new-born child, and consist of cyanosis, with shallow, rapid respiration. The lower part of the chest is drawn in by each respiration. On auscultation, the respiratory murmur is found to be very faint.

The *Symptoms* of **collapse of the lung** follow and complicate those of the disease which has led to the condition; for instance, the patient may not recover so rapidly as he ought, or the breathing is more embarrassed than can be accounted for by the concurrent disease in the chest. The physical signs vary considerably with the degree of collapse. Thus:

(*a*) In *complete* collapse of a part of the lung, as, for instance, in collapse due to compression or complete obstruction of a bronchus high up, there is impairment of the percussion note, with a diminution or absence of the breath sounds, and of the vocal resonance and fremitus.

(*b*) Where the collapse is only *partial* in degree—*e.g.*, where the bronchi remain patent, as occurs sometimes when the lung is compressed by pleuritic or pericardial effusion—there are signs resembling those of consolidation (p. 138), except that the percussion dulness is not so marked, and the breath sounds, though bronchial in character, are somewhat feeble.

(*c*) Where the collapse is *slight* and limited the chief sign is an enfeebled respiratory murmur. In addition, during deep inspiration are heard fine rustling crepitations, due to the expansion of the collapsed vesicles.

The *Diagnosis* is made usually by the existence of a causal condition. When this is detected, attention may then be directed to the physical signs of the lungs. It will be observed that the signs of partial collapse resemble the signs of consolidation, and those due to slight collapse resemble early pneumonia.

Etiology.—The causes are of two kinds: (*a*) Causes which produce *obstruction*, such as (i.) a tumour at the root of the lung (*e.g.*, aneurysm); (ii.) obstruction in the throat (? adenoids); (iii.) stricture of a bronchus (*e.g.*, gumma); (iv.) secretion obstructing the bronchi, though this is only sufficient to cause obstruction in children suffering, for example, from measles, whooping-cough, or broncho-pneumonia; (v.) foreign bodies obstructing the larynx or bronchus.

(*b*) *Compression* of the lung may be produced by pleural or pericardial effusion, an enlarged heart, or tumours of the mediastinum. The condition is often the result of spinal curvature.

In *adults* collapse is most often met with as the result of pleural effusion or tumours in the chest; in *children*, of bronchitis or broncho-pneumonia.

Prognosis.—The course of the disease depends very much upon the cause. Recovery usually soon takes place after compression by effusions, obstruction or stricture of the bronchi, and throat affections. It is unfavourable when associated with mediastinal tumours or foreign bodies in the bronchus.

The *Treatment* is unsatisfactory. It should be directed to the removal of the cause, and especially to the promotion of recovery of any concurrent pulmonary disorder. That form which yields best to treatment is met with in children with bronchitis and broncho-pneumonia. In adults it might be well to try the efficacy of respiratory exercises.[1]

[1] "Respiratory Exercises in Treatment of Disease, notably of the Heart, Lungs, Nervous and Digestive Systems," by Dr. Harry Campbell. London, 1899.

§ **101. Syphilis of the Lung.**—In children this disease may take one of two forms: (*a*) The pneumonic condition of lung, which is found in infants, usually still-born, is universally regarded as an interstitial pneumonia of syphilitic origin.

(*b*) Gummata are occasionally met with in the lungs of infants who are the subjects of hereditary syphilis; still more rarely they are met with in adults.

Dyspnœa is usually the only symptom. The signs are those of consolidation, and collapse.

In adults syphilis of the lungs may take other forms—*e.g.*, broncho-pneumonia, bronchiectasis, etc.—and may lead to extensive infiltration and breaking down, or to fibrosis.

Group C.—Chronic Diseases attended by **Hyper-resonance** on percussion: I. In quite nine out of ten cases of hyper-resonance it exists on both sides, and is due to Emphysema. There are five other conditions which give rise to it—namely: II. Pneumothorax (§ 89); III. Skodaic Resonance (§ 75); IV. A very large cavity in the lung (Phthisis, § 94); V. A Tumour between the chest wall, and a large bronchus (§ 99); VI. Dissemination of Solid Material through the lung in certain exceptional circumstances (*e.g.*, pneumonia, sarcoma, etc.). The diagnosis of these various conditions is given in the form of a table. All except Emphysema are described elsewhere.

Table IX.—Causes of Hyper-resonance.

Cause.	Hyper-resonance.	Auscultation.	Other Diagnostic Features.
I. **Emphysema.**	Bilateral and universal.	R.M. distinct and expn. much prolonged; signs of bronchitis, if present.	Barrel-shaped chest, cardiac dulness obscured, and organs displaced.
II. **Pneumothorax,** mostly Hydro-pneumothorax. An acute condition.	Hyper-resonance always unilateral, though it may extend beyond middle line.	Absence of R.M. and V.F. over affected area; sometimes amphoric breathing. Bell sound.	Organs displaced; history of emphysema or tuberculous cavity.
III. **Skodaic Resonance**—*i.e.*, the high-pitched note above a large *pleuritic effusion*, when the lung is otherwise healthy.	Unilateral; level may shift with position of patient.	Loud R.M.; V.F. felt over affected area.	History of pleurisy: signs of fluid lower part of chest.
IV. A very **large cavity,** or extensive bronchiectasis (rare).	Unilateral, and of limited extent (may be cracked-pot sound).	Amphoric breathing, whispering pectoriloquy.	Expectoration of pus and long history of phthisis or bronchitis.
V. A **Tumour** (or pneumonic consolidation) between the chest wall and a large bronchus (rare).	Unilateral, and of limited extent; dulness elsewhere.	Tubular breathing and bronchophony.	Symptoms of intra-thoracic tumour.
VI. **Infiltration of solid** and even fluid material throughout the lung, *e.g.*, early stage of pneumonia, miliary tubercle, etc. (rare).	Hyper-resonance not marked (may be bilateral).	Signs of consolidation in some parts.	Hyper-resonance generally transient.

I. *The patient has complained of breathlessness for some years. There is* **hyper-resonance** *on* **both** *sides of the chest. The disease is* EMPHYSEMA.

§ **102. Emphysema** is a chronic non-febrile disease of the lungs in which the air vesicles become hyper-distended, the walls separating each vesicle become atrophied, inelastic, and ruptured, and as a result the aerating surface is greatly diminished, and the lungs deficient in their elastic recoil.

Symptoms.—(1) The onset of the disease is imperceptible, and generally supervenes gradually after repeated attacks of bronchitis, the patient becoming more and more breathless after each attack. (2) This breathlessness is practically the only symptom, and it differs from all other kinds of breathlessness in this, that the chest remains *permanently in the inspiratory position*—in other words, owing to the inelastic state of the lungs and the shape of the chest, the patient finds it more difficult to expire than to inspire. A certain degree of cyanosis is generally present. (3) Symptoms of bronchitis are *nearly always present.*

The *Physical Signs*, expressed *briefly*, are a barrel-shaped chest, hyper-resonance, and prolonged expiration. The shape of the chest is special to emphysema (see Fig. 34). The chest assumes permanently the shape of a healthy chest in a position of deep inspiration. The antero-posterior diameter is considerably increased (see § 74). The hyper-resonance is always bilateral, and it obscures the dulness of the neighbouring organs—namely, the heart, the liver, and the spleen. These organs are also displaced downwards. The apex-beat may not be palpable, but epigastric pulsation is usually felt. On auscultation, the respiratory murmur is modified ; the inspiratory sound, which is full, is followed by a pause, and then by a prolonged expiratory sound. There are no adventitious sounds proper to emphysema, but, as just mentioned, bronchitis (*q.v.*) nearly always accompanies it. The heart sounds, especially at the base, may not be heard, or only with difficulty. Well-established emphysema interferes considerably with the pulmonary circulation, on account of the ruptured alveoli, and consequently the right side of the heart in course of time becomes dilated.

Variety.—In old people there is sometimes hyper-resonance with weak breath sounds, but no enlarged barrel chest ; this is called *Atrophic* Emphysema, and is due to the giving way of degenerate air vesicles.

The *Diagnosis* is extremely easy, because the bilateral hyper-resonance, the prolonged expiration, and the barrel-shaped chest are quite characteristic (*vide* Table of Diagnosis).

Etiology.—(i.) Emphysema occurs usually in elderly subjects. Both sexes are affected, but it is much commoner in males owing to the prevalence of bronchitis and asthma in them. (ii.) Heredity is said to play no part in the disease, but undoubtedly a hereditary tendency can frequently be traced. (iii.) Certain occupations render people prone to

emphysema—*i.e.*, those which throw strain upon the lungs, such as glass-blowers, wind-instrument blowers, etc. (iv.) The disease is frequently associated with senile degeneration, chronic Bright's disease, and cardio-vascular changes. (v.) Bronchitis is the most frequent of the exciting causes, owing to the prolonged coughing and straining to get up phlegm, and owing also to the blocking of certain tubes with thickened secretion, which prevents the access of air to some alveoli, and unduly distends others. (vi.) Asthma is also a potent exciting cause, owing to the constant strain on the elastic tissue of the lungs.

Prognosis.—Patients may live with emphysema to a good old age, and provided it is only moderate in degree it does not necessarily shorten life, though it predisposes to, and adds to, the seriousness of other pulmonary disorders. The gravity of any particular case is best measured by the extent of cardiac involvement (*q.v.*).

Treatment.—The indications are: (i.) To relieve the accompanying bronchitis (see § 93); (ii.) to improve the cardiac condition; and (iii.) to restore as far as may be the elasticity of the lungs. The diet is of considerable importance in advanced emphysema, for any distension of the stomach greatly adds to the respiratory distress. It is a good rule never to let patients take a solid meal later than two o'clock in the day; otherwise their nights become considerably disturbed by the breathlessness. Cardiac tonics, especially strychnine, and, in my experience, tinctura cacti grandiflori, are useful. Quinine and cod-liver oil often do good, although I cannot explain how the latter acts.

To restore the elasticity of the lungs is important, but difficult to accomplish satisfactorily; of late years a special form of respiratory exercise has been put forward as fulfilling this condition, and to relieve the difficulty of expiration. With this end in view, Gerrhardt[1] has recommended the employment of mechanical expiration by compression of the thorax methodically, every day for five or ten minutes, by another person, who places his two hands flat upon either side of the patient's chest. A similar result has been attained by Rossbach's Althemstühl (breathing-chair).

Group D.—There are three chronic pulmonary conditions in which the percussion note varies considerably in different cases, but the **offensive character of the sputum** reveals their presence—viz.: I. Bronchiectasis; II. Gangrene; and III. Abscess of the Lung. In Abscess the sputum is not so invariably offensive as in the others.

§ 103. Bronchiectasis.—Bronchiectasis is a cylindrical or saccular dilatation of the bronchial tubes. The condition is met with most frequently as a complication of chronic bronchitis or chronic pneumonia.

Symptoms.—The patient complains chiefly of persistent cough. At intervals of several days violent increase of coughing occurs; it is started, perhaps, by some change of posture, and is followed by the expectoration of a large quantity of

[1] Strumpel's "Pathologie und Therapie."

extremely fœtid sputum. In the intervals the sputum is scanty, but the breath is offensive. The fœtid sputum contains pellets or "Traube plugs," and sets characteristically in three layers (see § 78).

The *Physical Signs*, if present, are mostly those of a cavity, attended by general signs of chronic bronchitis in both lungs; occasionally only one is affected. The patient is often cyanosed, and has clubbed fingers.

Diagnosis.—The extremely fœtid odour of the sputum—occurring as it does *at intervals* of perhaps several days or weeks, in which the sputum is not fœtid—distinguishes bronchiectasis from all other diseases. In *gangrene* of the lung the sputum may be fœtid, although in a less degree, and it lacks the intermittent character. The position of the bronchiectasis is generally marked by dulness in the lower lobe of one lung, which may be made to disappear by making the patient lie face downwards for some time, with his head low, till he coughs up a large amount of sputum. The causes of the two affections also aid the diagnosis. Abscess of the lung is attended by a very profuse purulent expectoration, but it is not so fœtid.

Etiology.—(i.) In patients past middle life by far the most usual cause is prolonged chronic bronchitis, and in children whooping-cough. The dilated bronchial tube results from the continual strain of coughing on the weakened walls. (ii.) Various forms of chronic pneumonia and chronic phthisis are believed to be attended by bronchiectasis, but in such cases it is probably a cavity in the lung tissue, and not true bronchiectasis, that we meet with. (iii.) A foreign body plugging a bronchus is an occasional cause. (iv.) In very rare cases it is a congenital defect.

Prognosis.—The condition is a very serious one, and for the most part incurable. The patient may live from one to ten years. The prognosis is much worse in bilateral cases, or in cases associated with extensive disease of the lungs or pleura.

The *Complications* which may occur are fatal hæmorrhage, gangrene of the lung, lobular pneumonia, and pyæmia.

Treatment.—The indications are to relieve the disgusting fœtor and to cure the primary disease. The first is accomplished by liberal antiseptic inhalations of turpentine, coal-tar, or creosote.[1] The most valuable form of treatment is the creosote vapour bath. Patients are placed in an air-tight room, in which creosote is volatilised, the time of exposure being gradually increased from five to twenty minutes. Terebene and creosote may be given in capsules (4 minims) three times a day. Some inject menthol or guaiacol into the trachea in the proportion of 5 and 2 grains in 1 drachm olive oil twice a day. When the cavity is low down, surgical measures for its drainage have been adopted.

§ 104. Gangrene of the Lung.—Owing to the extreme vascularity of the pulmonary tissues, gangrene of the lung is a rare condition, but it occasionally occurs over a limited area. It is usually a secondary condition, but it sometimes occurs in a lung previously healthy.

Symptoms.—(1) The onset may be acute, and marked by prostration and an irregular, intermittent pyrexia of a pyæmic type, with a very rapid pulse. (2) If, as is usual, the gangrenous part opens into the bronchi, a profuse fœtid expectoration soon follows. The sputum contains fragments of lung tissue, and generally blood also. The fœtor is continuous, and not intermittent, as in bronchiectasis. (3) The breath is extremely fœtid. (4) Pain in the side is usual, though it depends upon the involvement of the pleura. There is persistent cough, which aggravates the pain. (5) The *Physical Signs* are those of consolidation, sometimes those of a cavity.

Diagnosis.—The only condition which is liable to be mistaken for it, by reason of its fœtid expectoration, is bronchiectasis, which is distinguished by having (i.) "Traube plugs" in the sputum, and (ii.) a gradual onset and longer course.

[1] *Brit. Med. Journ.*, June 22, 1895.

Etiology.—It is predisposed to by intemperance, old age, diabetes, and marasmus. Exciting causes are: (i.) Particles of food entering the lung, as in the insane, or patients with laryngeal paralysis, or persons in a drunken coma. In children a foreign body swallowed may produce it, though rarely. (ii.) Septic matter passing from the throat or mouth. (iii.) Severe sthenic types of pneumonia are occasionally so complicated. (iv.) It may complicate bronchiectasis. (v.) Embolism of the pulmonary artery. (vi.) Aneurysm pressing on the root of the lung. (vii.) The extension of an abscess near the lung.

Prognosis.—The disease is almost invariably fatal, either immediately from collapse, sometimes from fatal hæmorrhage, or, later, from prostration. A few cases have recovered where the patch was of small extent. In cases which have been recorded as lasting for months or years it is very doubtful whether the lesion was true gangrene; bronchiectasis is more probable. Occasionally the condition leads to pyopneumothorax.

Treatment.—The main object is to keep up the strength of the patient by means of abundant nutritive stimulants, bark, iron and quinine. For the rest, the treatment is the same as in bronchiectasis.

§ 105. Abscess of the Lung is a serious and, happily, rare condition, but as it is nearly always secondary to some grave or fatal disorder, it does not add very materially to the gravity of the situation.

It is usually manifested by the expectoration of a large quantity of purulent pus, which may or may not be fœtid, and is never so fœtid as in bronchiectasis or gangrene. The constitutional disturbance to which it gives rise is usually masked by that of the primary malady. The *Physical Signs* are those of localised consolidation, but these also are generally masked by those of the primary lesion.

It may occur in the course of (i.) advanced pulmonary tuberculosis; (ii.) pneumonia; (iii.) pyæmia; (iv.) cancer or other tumours of the lung, such as a gumma breaking down—a somewhat rare condition; (v.) it occasionally follows the introduction of septic foreign bodies or wounds in the throat.

Prognosis.—The prognosis is very grave, but depends upon the causes. Occurring in the course of pyæmia, it indicates the progress towards a fatal termination. It is less grave in pneumonia occurring in otherwise healthy persons.

Treatment.—Medical treatment is not of much use. Surgical interference is not good in malignant and pyæmic conditions, but in other conditions, if fairly superficial, the abscess may be drained.

CHAPTER VII

THE UPPER RESPIRATORY PASSAGES AND THE THYROID GLAND

THE throat may be the seat of the same morbid processes as affect other mucous structures, such as catarrh, ulceration, or new growths. It is, moreover, in this position that several very important general or constitutional maladies, such as diphtheria, scarlatina, and syphilis, have important local manifestations. These facts have long been known, but it has come to be recognised only quite recently that the throat, and especially the tonsils—organs whose functions are still imperfectly known—may constitute the portal of entry of certain microbic conditions. It has also been suggested that the virus of influenza, rheumatism, malignant endocarditis, and other septic conditions, thus gain admission into the general systemic circulation.

This chapter will deal with the symptoms referable to the **pharynx** (§ 106), the **larynx** (§ 119), the **nasal cavities** (§ 129), and the **thyroid gland** (§ 135).

THE THROAT.

§ **106. Symptomatology.**—"The throat" may be said to consist of the fauces, tonsils, palate, pharynx, and larynx, and we are here concerned with the investigation of these structures. The symptoms indicating disease of these parts are principally two—namely, SORE THROAT and HOARSENESS. The examination of the mouth and tongue is described under Disorders of Digestive Tract (Chapter VIII.).

(*a*) SORE THROAT is indicative mainly of disease of the *pharynx*, tonsils, and structures around. If the patient complains of "sore throat," turn to § 108, p. 187.

(*b*) HOARSENESS AND OTHER ALTERATIONS OF THE VOICE are indicative of some affection of the *larynx* (§ 119, p. 195). If NASAL INTONATION or NASAL DISCHARGE be present, turn to § 129, p. 207.

There are also several minor symptoms which arise in conjunction with these, such as a dryness accompanied by tickling sensations, or an excessive secretion, which leads to "hawking" and "coughing." Thus it happens that we may be consulted for what the patient believes

to be pulmonary disease, when in reality the lungs are perfectly healthy. Dyspnœa and dysphagia may also be produced by local conditions of the throat and larynx. "Globus," or paroxysmal sensations as of a ball in, or constriction of, the throat is a symptom of hysteria.

§ **107. Clinical Investigation.**—The anatomy and relations of the throat are indicated in Fig. 49; the various parts may be investigated by (*a*) direct, and (*b*) indirect (*i.e.*, laryngoscopic) examination.

(*a*) For the DIRECT EXAMINATION of the fauces and neighbouring structures all that is necessary is a good light and a spatula or spoon

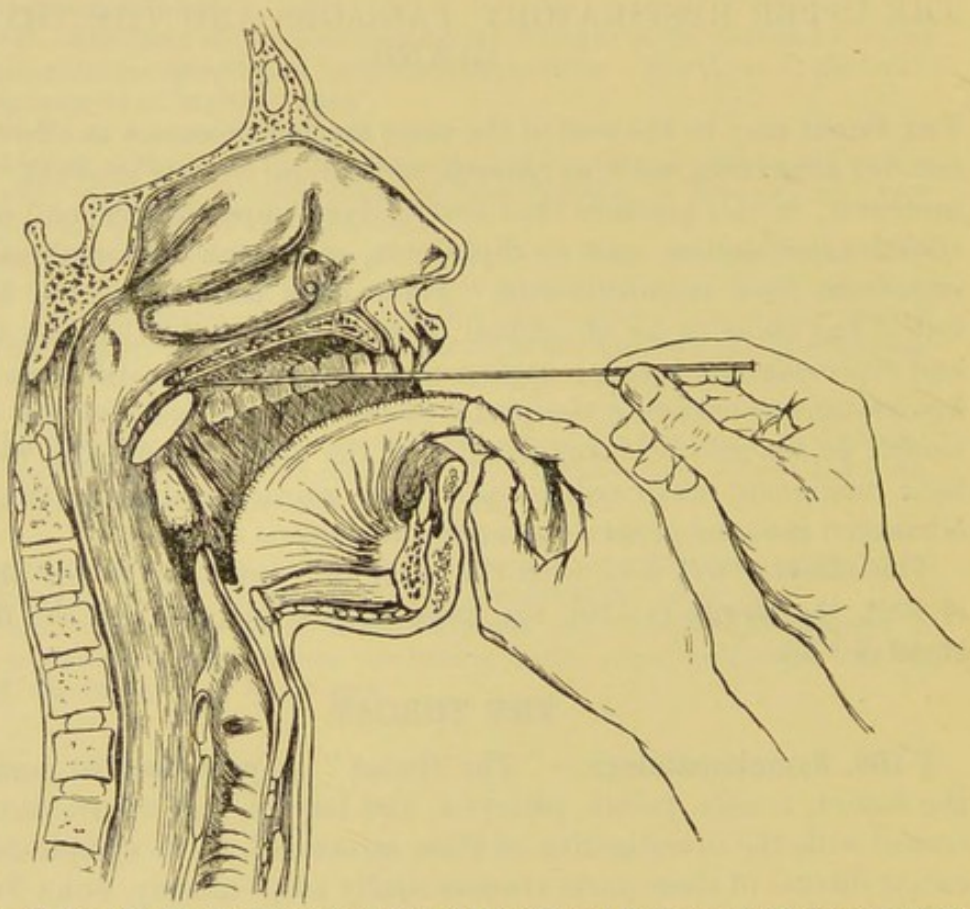

Fig. 49.—NASAL AND BUCCAL CAVITIES, showing the method of LARYNGOSCOPIC EXAMINATION. —The three turbinate bones are seen, the anterior end of the inferior turbinate bone having been removed to show the inner opening of the lachrymal duct. The opening of the Eustachian tube is just behind the posterior end of the inferior turbinate bone. The exact position of laryngoscopic mirror in examination of larynx is shown—namely, over the root of the uvula.

to depress the tongue. If direct daylight is not available—as for instance, when the patient is in bed—a laryngoscopic mirror can be used (*vide infra*), or a wax vesta, with or without a bright spoon behind it to act as a reflector. The patient should be instructed *not to strain, and to "breathe quietly in and out."* The posterior wall may be seen by directing the patient to say "Ha—ah," in which procedure the soft palate is raised. Note should be made of the colour of the mucous membrane, the presence of exudation or ulceration, of granulations or

adenoid vegetations in the pharynx, of any mucous patches such as occur in syphilis, or any bulging of the pharyngeal walls. The size and length of the uvula should always be observed, for a long uvula may be the sole cause of chronic cough and numerous otherwise unexplained symptoms. When a patient complains of cough coming on, or getting worse, at night or when he lies down, elongated uvula should be suspected. It does not follow that such a uvula may appear too long at the time of inspection. Temporary congestion from various causes—*e.g.*, much talking—produces undue elongation and nocturnal cough. Painting with tannin may reduce it, but the proper treatment is amputation, and it is wonderful what immediate relief is obtained.

(*b*) The INDIRECT or LARYNGOSCOPIC EXAMINATION of the throat will be given below, prior to modifications of the *voice* (p. 195).

§ **108. Classification, Diagnosis, Prognosis, and Treatment.—Sore Throat** is a symptom common to nearly all diseases of the throat. Mentioned in order of frequency, the diseases which give rise to sore throat are as follows (*laryngeal affections being excluded for* the present ; see § 119) :

Commoner Causes.	*Rarer Causes.*
I. Catarrhal pharyngitis, including two acute and three chronic varieties.	VI. Retro-pharyngeal abscess or tumour.
II. Tonsillitis.	VII. Phlegmonous sore throat.
III. Scarlet fever.	VIII. Cancer, and other new growths.
IV. Diphtheria.	IX. Tuberculosis.
V. Syphilis.	X. Other acute specific fevers.

§ **109. I. Acute Catarrhal Pharyngitis** is an inflammation of the mucous membrane of pharynx and soft palate, and to a certain extent of the tonsils also. It may be so mild as to cause only slight discomfort in swallowing, dryness of the throat, tickling and hawking, and in such mild cases there is only a moderate congestion of the parts. But in severer cases there are constitutional symptoms of some severity, and locally there may be œdema and ulceration. The temperature in such cases varies from 100° to 104° F. The disease rarely lasts more than a few days, ending generally in resolution, although sometimes it passes into a chronic condition.

Hospital Sore Throat is a severe variety of the preceding, attended by considerable ulceration upon the fauces, tonsils, and even pharynx. There is the greatest difficulty in swallowing, speaking, and sometimes in breathing. The submaxillary and cervical glands are enlarged, and there is often considerable pyrexia and constitutional disturbance, the prostration being out of all proportion to the local inflammation.

(*a*) **Chronic Catarrhal Pharyngitis** presents the same symptoms as the acute variety, in a milder degree, and extending over a longer period of time. It is often known as Relaxed or Relapsing Sore Throat,

on account of the chronic congestion of the parts and the consequent predisposition to the repeated occurrence of subacute attacks. It forms one variety of clergyman's or school-teacher's sore throat.

(*b*) **Granular (Follicular) Pharyngitis** is a *chronic* condition, the local symptoms of which resemble the foregoing, with the addition of visible granulations on the pharyngeal walls due to the enlargement of the follicles[1]; hence it is sometimes called follicular pharyngitis. This is a common condition, and a person who is the subject of it, although apparently in good health, is liable to suffer from repeated attacks of sore throat whenever the weather is damp or his health a little below par. There is excessive mucous secretion, which collects in the throat, especially in the morning, which leads to chronic cough and hawking. When the disease has lasted some time the throat becomes dry from atrophy of the follicles (Pharyngitis sicca).

(*c*) **Granular (Adenoid) Pharyngitis** is another form of *chronic* pharyngitis, due to the presence of adenoid hyperplasia and vegetations in the pharynx and naso-pharynx. They are sometimes confined to the naso-pharynx, and by an ordinary inspection of the fauces little mischief, excepting congestion, can be discovered. The lymphoid granulations may involve a large part of the naso-pharynx, occurring as a large grooved cushion or pedunculated growth, which, on examination, can be seen and felt behind the soft palate. This condition is said always to start in childhood. The child *breathes with the mouth open*, and thus acquires a characteristic vacancy of expression. Curiously enough, the intellect is sometimes below the average. The voice has a dull or nasal twang, and there are snoring and disturbed sleep. The nares are narrowed, and the palate may be high from the negative pressure in the nose, the diminished air tension in the nose not counterbalancing the normal air tension on the buccal aspect of the hard palate. The condition is a pregnant cause of middle-ear catarrh and subsequent deafness. Adenoids in the naso-pharynx are usually accompanied by chronic enlargement of the tonsils. The disease often runs in families.

The *Causes of pharyngeal catarrh* vary somewhat in the different forms, although the several causes are largely interchangeable. (1) There is no doubt that in certain persons exposure to cold and damp is immediately followed by an attack of pharyngitis, but how far this acts as an exciting cause, or whether, as in the possible case also of tonsillitis, the condition is set up by a microbe which thrives under certain atmospheric conditions, is not yet determined. (2) Unhygienic surroundings, such as bad drains, the atmosphere of a hospital, and the like, may certainly give rise to a very severe ulcerating pharyngitis (*e.g.*,

[1] The word "follicle" is applied, not only to the lacunar glands or crypts in the tonsil, but also to the localised collections of adenoid tissue found in the posterior wall of the pharynx. These latter, when enlarged, form the "granulations" of the granular pharynx.

hospital sore throat) ; and here, again, the cause may be a microbe. The same condition may arise in private houses in which the drainage is out of order. (3) Bad health in the individual affords undoubtedly a predisposition to the disease, and especially to granular pharyngitis, so much so that the throat in some persons constitutes a veritable barometer of the state of their health. (4) The gouty and rheumatic diatheses are said by many to offer a predisposition to pharyngitis (compare No. 6 below). (5) Various local conditions, such as nasal obstruction or insufficiency, leading to mouth-breathing ; and thus chronic rhinitis and *adenoid vegetations* are potent causes of recurring "sore throats." Excessive use or misuse of the voice (clergyman's and school-teacher's sore throat), excessive smoking, the constant use of alcohol, spiced or hot foods, or working in a dust-laden atmosphere, often play an important part. The bristle of a tooth-brush or a fish-bone impacted in the pharynx is a not infrequent though unsuspected cause. (6) I have often met with chronic pharyngitis in people who live too well. The excessive secretion and the perpetual hawking which results have in several instances directed the attention of the patient and of his medical adviser to the throat, larynx, or lungs ; but the cure of these cases cannot be accomplished until dietetic and other measures are directed to the relief of the portal congestion. (7) Pharyngitis, especially in its chronic forms, is often associated with anæmia, and iron is one of the most valuable remedies we have.

Prognosis.—Pharyngitis is rarely fatal, but it is one of the most frequent and troublesome of the minor ailments which we are called upon to treat. The milder varieties of the acute pharyngitis last only a few days, but the severer forms, such as hospital sore throat, may last many weeks, and be followed by considerable debility. All the chronic forms have a great *tendency to relapse.*

Treatment.—The indications are to relieve the local inflammation, to improve the general condition, and to prevent relapse. For the *acute forms*, most of the remedies mentioned under Tonsillitis are available. In all subacute and chronic forms, smoking, alcohol, and other causes of local irritation must be avoided. Excessive secretion may be removed by a gargle of bicarbonate of soda. For the "relaxed throat" a gargle consisting of a wine-glassful of water, to which a pinch of salt has been added, is useful; so, also, are gargles of alum, potassium chlorate, and ammonium chloride (F. 15 to 19). Probably carbolic acid, painted on as glycerine (B.P.), or employed as a spray, gargle, or lozenge, is the best application. A good spray is that of menthol (10 grains to the ounce of paroleine). Later, astringent paints should be used—*e.g.*, nitrate of silver (20 grains to the ounce) or equal parts of tincture of iodine and the glycerine of alum.

The most efficient treatment for the granular forms of pharyngitis, where gargles are of little use, is painting with silver nitrate (40 or 80

grains to the ounce), or with liquor ferri perchloridi, or iodine in glycerine. Trichloracetic acid is recommended highly. In cases of dry pharyngitis the ammonium chloride inhaler or lozenges are very useful. All these measures, however, may give only temporary relief to the *adenoid* variety, and then scraping away the vegetations is necessary. For a permanent and radical cure the vegetations must be thoroughly removed under general anæsthesia. Nasal obstruction, if present, must also be relieved.

The general health in certain forms, especially the granular varieties, is often of more importance than the local condition, and many a relapsing and granular pharyngitis can be cured by Blaud's pills. Any rheumatic or gouty diathesis should receive attention, and dyspepsia or constipation, especially if associated with portal congestion, should be appropriately treated.

§ **110. II. Tonsillitis,** or inflammation of the tonsil, is met with clinically in three forms—(*a*) and (*b*) two varieties of *Acute Tonsillitis*, and (*c*) *Chronic Tonsillitis*. *Peritonsillitis* is sometimes described as a variety ; it is an inflammation of the connective tissue in the vicinity of the tonsil ; it accompanies catarrhal pharyngitis, and is sometimes due to decayed teeth.

The **two forms** of **Acute Tonsillitis** are as follows :

(*a*) **Acute Parenchymatous Tonsillitis** (Quinsy, Acute Suppurative Tonsillitis). The symptoms are pain, swelling and redness of the tonsils, coming on more or less suddenly with constitutional disturbance, the temperature varying from 101° to 104° F. One tonsil is usually more affected than the other, and there is pain, stiffness, and tenderness behind the angle of the jaw. The disease usually subsides in the course of a week ; if it lasts longer than this, suppuration has almost certainly occurred on one or other side. This is evidenced by the increased enlargement, by the swelling spreading along the soft palate, backwards, and downwards into the pharynx. The abscess usually bursts in the course of one or two weeks into the pharynx, but it occasionally points in other directions.

(*b*) **Acute Follicular Tonsillitis** is of a more superficial character. It is attended by the same symptoms as the foregoing, with the exception that abscess rarely occurs ; and the surfaces of the tonsils present numerous yellow points of thick purulent secretion, and perhaps ulceration. There are usually less fever and pain than in (*a*) above.

The *Diagnosis* of both these forms of tonsillitis from scarlet fever and diphtheria is sometimes a matter of considerable difficulty, but one of great importance. It is given in the form of a table (p. 193).

Etiology.—The function of the tonsils is still a matter of some uncertainty, and therefore it is not surprising that the etiology of tonsillitis is largely speculative. (1) Hereditary predisposition seems to play its

part, for tonsillitis often occurs in subjects having a gouty or rheumatic tendency. (2) Unhygienic conditions, and especially bad drainage, have been credited with causing the disease. (3) The tonsils become acutely inflamed in all cases of scarlet fever, in diphtheria, and in so large a proportion of cases of rheumatic fever that they are regarded as the portal of entrance of the virus of that disease. (4) Cold and damp weather are certainly conditions under which tonsillitis frequently arises. (5) Traumatism, such as drinking out of a boiling kettle. Fish-bones and bristles of a tooth-brush sometimes give rise to one-sided tonsillitis.

(*c*) **Chronic Tonsillitis** (chronic enlargement of the tonsils) occurs in two forms. That form chiefly seen in adults after repeated attacks of acute tonsillitis is due to a fibroid degeneration, and is known as relapsing tonsillitis or chronic fibroid tonsillitis. The other and commoner form is that seen in children, which consists of a *parenchymatous* hyperplasia. The condition is almost always associated with adenoids in the naso-pharynx, and consequently there are snoring and mouth-breathing.

Course and Prognosis.—Acute tonsillitis is a frequent, and sometimes very troublesome, but never fatal, disease. Sometimes the patient continues at work, but at others he is totally incapacitated. The chief consequence to be feared is frequent recurrence, which leads to chronic enlargement of the tonsils. Chronic tonsillitis is important, because it renders the patient so liable to repeated attacks of acute tonsillitis and coryza. It is a common source of recurrent pharyngitis, leading to otitis media and deafness. Enlarged tonsils met with in children occasionally disappear during adolescence ; but in some way, only imperfectly explained, the mental and physical development of children who have chronic enlargement of the tonsils is sometimes impeded. This is possibly explained by a defective hæmopoietic function, for these structures contain a large quantity of lymphoid tissue. It is, however, doubtful whether the development of the child is hindered unless there be concurrent adenoids, which interfere with the respiratory or oxidative processes of the body.

Treatment.—The indications are (*a*) to reduce the local congestion ; (*b*) to reduce the pyrexia ; and (*c*) in chronic tonsillitis to prevent relapse.

(*a*) Powdered sodium bicarbonate applied directly to the tonsils has been credited with aborting the disease. A cocaine spray (4 per cent.) relieves the pain. Cold compresses externally, steam inhalations, warm gargles of potassium chlorate, sodium bicarbonate, salol, and weak alum or carbolic acid (1 in 100) relieve the congestion (F. 15 to 19). In subacute or relapsing cases the tonsil may be painted with potassium iodide, gr. xv. ; iodine, gr. xii. ; oil of peppermint, ℥ii. ; glycerine ʒi.

(*b*) To reduce the pyrexia a brisk saline purge should be given at the outset. Tincture of aconite (1 minim doses) may be given every half-hour during the first few hours; then sodium salicylate, as in rheumatism, salol, or liquor ferri perchloridi. If the disease does not clear up in a week, one may be almost sure an abscess has formed, and should be incised. This is best done with a curved bistoury, round which lint is twisted to within an inch of the point, which should be directed *inwards and backwards* to avoid the internal carotid. Make a small incision; then insert a probe or dressing forceps, and push it in all directions.

(*c*) In chronic tonsillitis the most useful remedies are iron, quinine, cod-liver oil, and other tonics. Salicylic acid, guaiacum, and colchicum are used in the relapsing form. The chronic enlargement may be diminished by painting the throat with glycerine of tannic acid (a most nauseous preparation), or tincture of iodine (℥i. to ℥viii.), and other astringents (*vide supra*). But in most of these cases the question of tonsillotomy arises sooner or later. Parents sometimes raise objections on the score that it may "impair the voice" or "injure the health," but there is no reason to believe that this is so.

§ **111. III.** In **Scarlet Fever** the tonsil is generally the chief seat of inflammation in the throat. Both scarlet fever and acute tonsillitis start more or less suddenly, with constitutional symptoms, and thus the diagnosis is often one of considerable difficulty. There are four distinguishing features of scarlet fever—viz.: (i.) The diffuse *scarlet* colour of the soft palate and pharynx, with complete immunity of the larynx; (ii.) sudden onset of the illness with high fever; (iii.) on the second day the rash; and (iv.) about the third day the "strawberry" tongue (see Table X., p. 193, and Chapter XV.).

§ **112. IV.** The sore throat of **Diphtheria** may be recognised at once if there be an ashy-grey patch of exudation *upon the soft palate*. When this is absent it is chiefly with follicular tonsillitis that difficulties arise. In diphtheria the large size and the colour of the patches (which are grey with surrounding red areolæ), the difficulty of removing them, and the raw bleeding surface left, enable us to come to a conclusion. The onset is more insidious, the pyrexia less marked, but the prostration is greater in diphtheria. A muco-purulent or hæmorrhagic discharge from the nose is very characteristic of diphtheria. The occurrence of albuminuria is given by some as distinctive of diphtheria, but it is very frequently observed in acute tonsillitis also. When other diagnostic features are absent, the presence of *one* large patch on a tonsil, instead of several small patches, is in favour of diphtheria.

§ **113. V. Syphilitic Sore Throat** is very characteristic. This and the other *secondary* manifestations of syphilis come on about three to six weeks after the appearance of the chancre. (1) The tonsils may be inflamed, but the inflammation is more generalised, and the mucous

membrane presents greyish-white semi-translucent irregular patches (" snail-tracks "), on the fauces, tonsils, palate, and other parts of the buccal mucous membrane. Superficial ulceration may also be present, especially on the tonsils, with red punched-out edges and yellow-grey secretion. (2) Bilateral symmetry is a very characteristic feature of all these lesions.

TABLE X.

Tonsillitis.	*Scarlet Fever.*	*Diphtheria.*
	(*a*) LOCAL SIGNS.	
Swelling and redness chiefly confined to one or both tonsils. In the follicular form, tonsils covered with sticky mucus, with numerous small, separate yellow spots of secretion on one or both, which are easily removable. Nothing on soft palate.	Diffuse *bright* redness of throat generally. The tonsils swollen, and may be covered with mucus, and *sometimes* with multiple yellow points. Nothing on soft palate in ordinary cases.	Ashy-grey patch or patches on tonsils, uvula, *and soft palate* (latter situation is pathognomonic); patches *larger* than the follicular secretion in tonsillitis. Patches consist of membrane surrounded by red areolæ; difficult to remove, leaving raw surface. Klebs-Löeffler bacillus found in membrane. Sometimes a muco-purulent, acrid *nasal discharge*. Comparative absence of pain.
	(*b*) GENERAL SYMPTOMS.	
(i.) Onset moderately sudden, with moderate fever. (ii.) Temperature may be very high, but local symptoms are usually more troublesome than general symptoms.	(i.) Onset very sudden, with high fever. (ii.) Temperature very high. Local symptoms a subordinate feature. (iii.) Rash on second day. (iv.) Strawberry tongue about third day.	(i.) Onset insidious. Early and marked enlargement of cervical glands. (ii.) Temperature not so high at first, and may remain low during whole course. (iii.) Paralytic sequelæ sometimes.

Tertiary syphilitic ulcers may produce sore throat, their favourite position being the soft and hard palate, the tongue, and the fauces. They are usually preceded by gummatous swellings. (1) The ulcers are deep, with ragged floor, sharply cut edges, and covered with thick yellow-grey secretion. (2) They are progressive, and in course of time will destroy the hard palate or any other parts they invade. (3) They leave characteristic stellate cicatrices, which are indisputable evidence of the disorder.

The less frequent causes of Sore Throat are—Retro-pharyngeal Abscess, Phlegmonous Sore Throat, Neoplastic Ulcerations, *and* Acute Specific Fevers.

§ **114. VI. Retro-pharyngeal Abscess,** or inflammation of the lymphoid and areolar tissue between the pharynx and the spine, may come on insidiously, or it may be comparatively sudden. It is known by (1) the rigidity of the head, with difficulty of swallowing and alteration of the voice; (2) evidence of swelling in the posterior pharyngeal wall on inspection and palpation, by which it is diagnosed from croup in children.

Etiology.—Those cases with an *acute* onset are generally either part of a septic inflammation after fevers, or occur in rachitic children under four. Retro-pharyngeal swelling coming on *slowly* is generally due to pus burrowing from some adjacent structure, especially from caries of the vertebræ.

Prognosis and Treatment.—The acute condition is always grave, and requires prompt surgical interference, generally free incision; meanwhile, steam inhalations and warm fomentations relieve the symptoms.

§ **115. VII.** Phlegmonous Sore Throat—*i.e.*, Acute Septic Inflammation of the Pharynx and Larynx—or Angina Ludovici (when the inflammation is chiefly external, in the neck).[1]—This very severe disease may start *inside* the throat, with symptoms of sudden pain, accompanied by considerable swelling, leading to severe dyspnœa, stridor, aphonia, and complete dysphagia in a few hours. There is much œdema around the fauces, followed by a brawny infiltration of the skin of the neck, spreading from under the jaw to the tongue and larynx. Sometimes the infiltration starts *externally*, and rapidly invades the internal structures. There is great constitutional disturbance, and a temperature of 102° to 105° F., but unless pus forms, rigors and delirium are generally absent. Pus formation is further indicated by a widely and irregularly intermittent pyrexia. Mild cases begin with a stiffness and pain in the tissues around the jaw, and if recovery is to take place, the symptoms go no farther. But in many cases, and especially in alcoholic and debilitated subjects, the disease rapidly progresses, and death takes place in twelve to forty-eight hours from heart failure, coma, or asphyxia from œdema of the larynx. Suppurative forms are very fatal. Among the recognised complications are pneumonia, pericarditis, pleurisy, and meningitis. The disease is readily recognised by its sudden and severe onset and rapid progress, usually to a fatal issue.

Etiology.—The condition, happily, is rare, and the causes consequently obscure. (1) It sometimes arises in association with scarlet fever, erysipelas, and small-pox (in former times being a common cause of death in this disease), or other acute specific fevers. (2) Dental suppuration or an alveolar abscess often forms the source from which rapid infiltration starts. (3) It may arise in people apparently in good health, and has then been attributed to the entrance of a specific microbe by the tonsils, or through the socket of an extracted tooth.

Treatment.—The indications are to control the inflammation, and to keep up the strength of the heart. Quinine (4 or 5 grains) should be given every four hours. Iron and digitalis are recommended. Remove carious teeth or stumps.

[1] The disease has been variously described by the following names: Acute Inflammatory Œdema, Erysipelas of the Throat, Phlegmonous Cellulitis, Acute Infectious Phlegmon (a term applied by Senator when the inflammation was confined to the wall of the pharynx).

Free and early incisions should be made into the œdematous tissues, and the practitioner should be at hand to perform tracheotomy if the dyspnœa be increasing. Stimulants must be liberally administered.

§ 116. **VIII.** CARCINOMA frequently, and SARCOMA occasionally, affect the pharynx, either primarily or secondarily. Their diagnostic features are the same as those mentioned under The Tongue (§ 148).

§ 117. **IX.** TUBERCULOUS ULCERS of the pharynx are rare as primary lesions. (1) They resemble syphilitic ulcers, but there is pallor of the mucous membrane, and a characteristic " worm-eaten " appearance of the pharyngeal wall. (2) *Their course is not nearly so rapidly progressive.* (3) It may be possible to obtain the tubercle bacillus from the scrapings ; and (4) there are usually other manifestations of tubercle, especially in the lungs. For treatment, see Tuberculosis of the Larynx (§ 123).

§ 118. **X.** ACUTE SPECIFIC FEVERS other than those mentioned above, such as typhoid, give rise to inflammation and ulceration of the throat. In variola, for example, the pustules often form upon the palate, fauces, and buccal mucous membrane, leaving superficial circular ulcers. An examination of the throat is often useful as an aid to the diagnosis between measles, scarlet fever, and small-pox. The first-named always affects the *larynx*, rarely the pharynx ; scarlet fever always affects the *pharynx*, and very rarely the larynx ; whereas small-pox affects them *both about equally.* Patches of *Lichen planus* may be found on the palate when the disease exists on the skin, and the eruption of varicella may be found in that situation.

The Larynx.

§ 119. **Symptoms and Clinical Investigation.**—It will be remembered that the two cardinal SYMPTOMS of diseases of the throat (used in its widest sense) were (*a*) Sore Throat, and (*b*) Alterations of the Voice. Both of these may be present in disorders of the larynx, but it is the latter especially which indicates derangements of the organ of voice. Diseases of the larynx are also sometimes indicated by Cough, Hawking, Dysphagia, Dyspnœa, and actual Pain in the organ. But in some cases all of these may be absent ; there may, indeed, be pronounced disease of the larynx (*e.g.*, paralysis or papilloma) without any *subjective* symptoms.[1]

The CLINICAL INVESTIGATION of the larynx (laryngoscopy) is a procedure of considerable technical nicety, and requires some practice. The necessary appliances are a good steady light, a *reflecting mirror* mounted on a band or a spectacle frame for the operator's forehead, and a small circular *throat-mirror* mounted on a handle at an angle of 135°. The light should be placed on a level with, and a little behind, the patient's left ear. The operator takes his seat directly opposite ; and it is advisable that his seat should be a little higher than that of the patient. Having directed the patient to open his mouth and " breathe quietly in and out," the first step is to adjust the *reflecting mirror* in order to thoroughly illuminate the back of the pharynx. The

[1] Not long ago I met with the case of a well-known operatic singer who had a small papilloma just beneath one vocal cord. Her voice was in perfect order, and she could reach the highest notes with ease. The only defect was a hardly perceptible weakness in the middle register.

focal length of the head-mirror is generally 10 to 20 inches, and this should represent the distance of the mirror from the patient's pharynx. Having warmed the throat-mirror over the lamp, the next step is to pull the patient's tongue with the left hand gently out of the mouth with the aid of the corner of a towel or a piece of linen rag. Take the corner of the towel in the right hand, lay it on the patient's tongue, then grasp the tongue and towel firmly between the left thumb and finger. Take care not to hurt the under surface of the tongue against the teeth of the lower jaw. Then test the warmth of the throat-mirror against your cheek or the back of your hand, and, having pushed the patient's head a little backwards by pressing your right thumb against the upper teeth, introduce the mirror with the right hand, *taking care to avoid touching the top of the tongue* in so doing. Push the mirror obliquely upwards against the soft palate just over its junction with the uvula (Fig. 49, p. 186). This should give you a good view of the vocal cords by slightly lowering or raising the handle. In children and persons with very sensitive throats it is sometimes advisable to anæsthetise the pharynx before laryngoscopy, either by a spray of, or painting with, a 4 or 5 per cent. solution of cocaine, or by the administration of a few doses of bromide during the preceding twenty-four hours.

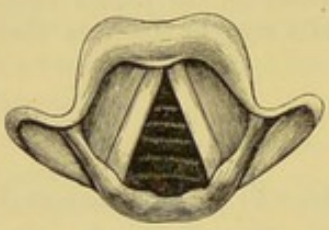

Fig. 50.—Quiet Inspiration.

Fig. 50*a*.—Forced Inspiration.

In normal conditions the *epiglottis*, which is in reality anterior, appears at the *upper part of the mirror*. The *vocal cords*, which are of a pearly white colour, are close together at their upper or epiglottic ends; and at their lower (really posterior) ends are widely divergent during quiet respiration. At their lower ends they appear to terminate in two prominent knobs seen at the lower edge of the mirror, which mark the position of the *arytenoid cartilages* (Figs. 50 and 50*a*). The The *ary-epiglottic folds* stretch on each side from the arytenoids to the sides of the epiglottis. In these folds, just external to the arytenoid on each side, may be seen a small prominence, the *cartilage of Wrisberg*. To the outer side of the cords lie the ventricular bands or false cords of mucous membrane. With a little practice, and under favourable circumstances, the bifurcation of the trachea may be seen.

In LARYNGOSCOPY there are FOUR MATTERS to be investigated:

(*a*) The presence of *congestion* or *pallor* of the vocal cords and the parts around. Congestion of the vocal cords is an evidence of LARYNGITIS, sometimes of ulceration or new growth.

(*b*) The presence of any *ulceration*. Ulceration occurring in a person below middle age is very often due either to SYPHILIS or TUBERCLE; in a person beyond middle life it is not infrequently MALIGNANT.

(*c*) The presence of a *nodule* or *new growth*. A nodule or new growth proves most frequently to be a PAPILLOMA.

(*d*) Whether there is any *paralysis* or *spasm* of the vocal cords, which is evidenced by the size, shape, and *mobility* of the aperture.

We shall deal with the disorders of the larynx in this order.

§ **120. Classification.**—As just mentioned, there may be no *subjective symptoms* even with pronounced disease of the larynx, and therefore it will be well to adopt as a basis of classification the *physical signs* discovered by laryngoscopy. However, when symptoms are present there is always some ALTERATION OF THE VOICE (except, perhaps, bilateral abductor paralysis, in which there may be dyspnœa and stridor without alteration of the voice). The principal diseases giving rise to such alterations (*i.e.*, the **causes of alterations of the voice**) may be grouped as follows :

I. LARYNGITIS—
  (*a*) *Acute Laryngitis*, including also—
    Œdema Glottidis, and
    Foreign Bodies in the Larynx or Trachea.
  (*b*) *Chronic Laryngitis*, including also—
    Perichondritis, and
    Infantile Stridor.

II. ULCERATIONS of the Larynx—
  (*a*) Tuberculous Ulceration,
  (*b*) Syphilitic Ulceration,
  (*c*) Malignant Ulceration.

III. NODULES and NEW GROWTHS—
  (*a*) Benign (warts and polypi),
  (*b*) Malignant Neoplasms.

IV. PARALYSIS of the Vocal Cords—
  Bilateral Abductor Paralysis,
  Unilateral Abductor Paralysis,
  Total (Ab- and Adductor) Bilateral Paralysis,
  Total (Ab- and Adductor) Unilateral Paralysis.

V. SPASM of the Vocal Cords—
  Laryngismus Stridulus or Croup.

VI. Diseases of the PHARYNX (§ 108) ; VII. Diseases of the NOSE (§ 129) ; VIII. Some severe PULMONARY affections ; and IX. Certain NEUROSES also cause alterations in the voice.

1. *The patient complains of huskiness or loss of voice, a comparatively dry cough, soreness on swallowing, and there are local signs of congestion of the vocal cords. The disease is* LARYNGITIS, *of which two varieties are met with,* ACUTE *and* CHRONIC.

§ **121. Acute Laryngitis** comes on somewhat rapidly, and usually runs its course in a week. As a rule it is not a serious affection, but in children it may be alarming. In children a slight laryngitis coming

on suddenly is a frequent cause of what mothers describe as "croup." Owing to the dryness of the cords, the child wakes up suddenly at night with loud inspiratory stridor followed by an attack of coughing. This symptom is technically known as *laryngitis stridulosa*, and is not to be confused with laryngismus stridulus (see Spasm of the Cords). Simple laryngitis is differentiated from membranous croup (laryngeal diphtheria) by the perfect general health of the child in the former.

Etiology.—The chief cause of acute laryngitis is exposure to cold—especially when combined with overuse and misuse of the voice (*e.g.*, actors, music-hall artistes, etc.). It is frequently a part of the "common cold." Diphtheria or measles may start in the larynx. Persons who suffer from chronic laryngitis (*q.v.*) or nasal obstruction are predisposed to attacks. A foreign body in the larynx or trachea is a cause of irritation which may produce symptoms resembling laryngitis.

Prognosis.—The affection is troublesome and apt to recur. When occurring during the course of the specific fevers the prognosis is less favourable, because Œdema Glottidis may supervene.

Treatment.—All use of the voice must be forbidden. The patient must be kept in a warm, moist atmosphere, and should use warm inhalations (such as tr. benzoin co. ℥i. to the pint of boiling water, and see also F. 110). Warm compresses or fomentations should be applied externally, and warm mucilaginous and alkaline drinks should be freely taken. The most efficacious medicine is one containing small doses of vinum antimonialis and potassium iodide. According to some authors, a strong solution of silver nitrate (20 grains to ℥i.) applied locally at the outset may cut short the disease. For laryngitis stridulosa, hot sponges should be applied to the throat, and vin. ipecac. in teaspoonful doses, with warm water, should be given every ten minutes or so until emesis ensues.

§ 121*a*. **Œdema Glottidis,** or œdematous laryngitis, consists of an œdematous swelling affecting the epiglottis and submucous tissue of the larynx, but the vocal cords are not involved. The onset is usually sudden, and attended by considerable dyspnœa, dysphagia, and inspiratory stridor. The diagnosis is usually simple, on account of the swelling which can be seen and felt on palpation at the back of the tongue. If this be absent, some difficulty may be experienced, but the sudden onset of laryngeal dyspnœa should bring the disease to our minds. It may arise either as a primary or as a secondary affection. As a primary disease it may come on as part of an acute septic inflammation of the throat, or it may be part of an œdematous angio-neurosis of urticarial origin (see Acute Œdema of the Tongue). It may occur as a *secondary* condition in association with (1) one of the various causes of acute or chronic laryngitis; (2) a general anasarca; (3) injury of the glottis by boiling or caustic liquids, etc. Its rapid onset is the chief source of danger, but if the patient does not shortly succumb to asphyxia, recovery generally takes place in a few days.

The *Treatment* consists in the administration of emetics, and ice internally and externally. In severe cases, if a 20 per cent. cocaine spray fail, scarification of the epiglottis must be resorted to; and if this be unsuccessful, tracheotomy must be performed without delay.

§ 121*b*. **The Swallowing of a Foreign Body,** and its passage into the larynx or trachea, has always to be borne in mind in children suffering apparently from acute laryngitis, for the history is often wanting. *Paroxysms of dyspnœa* or of *coughing* in a child without obvious cause should make us suspect it. Unless it has passed into the bronchus (usually the right), a foreign body may be seen by laryngoscopic examination. On the other hand, when a foreign body passes into the bronchus, it may cause so little cough or disturbance at the time that the patient may imagine he has swallowed it, or he may be unmindful of the incident. Some obscure cases of unilateral bronchiectasis are probably due to such causes. When the presence of a foreign body is suspected, a skiagram should be taken.

§ **122. Chronic Laryngitis** is a troublesome affection on account of the perpetual hoarseness and liability to acute laryngitis. Its causes are (1) repeated acute attacks ; (2) excessive speaking, singing, teaching, and overuse of the voice (actors, clergymen, school-teachers, etc.) ; it also affects masons, fustian-cutters, and others exposed to dusty atmospheres ; (3) nasal obstruction and mouth-breathing ; (4) tubercle, syphilis, and new growths, the evidences of which should always be sought in cases of intractable laryngitis. These usually go on to ulceration, under which they will be described. (5) Spread of inflammation from adjacent parts. Many cases of chronic laryngitis depend upon a granular condition of the pharynx. (6) Rheumatic and gouty diatheses predispose.

Treatment.—The indications are to avoid the cause and to relieve the local congestion. The removal of the cause is most important, and often most difficult to accomplish, for a large number of the patients are singers, teachers, and others whose living depends upon the daily excessive use of the voice. The avoidance of tobacco and alcohol will aid, and residence in a dry climate will often accomplish a speedy cure. Much may be done to prevent or relieve the condition by proper voice-production and respiration. This affection is extremely common among our board-school teachers, owing chiefly to faulty voice-production, and they ought to be specially trained to obviate this defect. Locally, painting with strong astringent remedies, such as zinc chloride (30 grains to the ounce) or silver nitrate (20 grains to 60 to the ounce), are useful. These strong applications should not be made more than twice a week ; weaker solutions can be applied more frequently. The patient himself may use sprays of alum (5 grains) or zinc sulphate (2 grains to the ounce) for five minutes twice daily, or inhalations of turpentine, creosote, iodine, menthol, etc., for fifteen minutes three times a day.

§ 122*a*. **Perichondritis** is an inflammation of the perichondrium of the laryngeal cartilages. Opinions differ as to its frequency. If considerable, it may lead to necrosis of the cartilages and abscess of the larynx. The differential features, besides loss of voice or hoarseness, are dull aching pain and acute tenderness. This may be accompanied by swelling in the neck. As regards its *Etiology*, apart from traumatism, it is rarely a primary malady. It more often occurs secondary to syphilitic or tubercular laryngitis. Syphilis is its commonest cause. It also follows enteric fever.

Prognosis and Treatment.—It is a serious affection, for even in the mild forms the voice is rarely restored. Great stenosis of the larynx may result. If there be much swelling, the dyspnœa is very marked, and the patient may die from pneumonia or gangrene of the lungs, or, in the suppurating forms, from pyæmia. Abscess and fistula may follow.

§ 122*b*. **Chronic Infantile Stridor** is a term applied to more or less continuous inspiratory dyspnœa, accompanied by a croaking sound, occurring in infants. It may be more or less constant up to the age of two, or occur only at intervals during that period of life. It is believed to be caused by a folding of the epiglottis, possibly due to some malformation. It is usually attended by a certain amount of laryngitis and hoarseness, and passes off without need for operative interference.

II. **Ulcerations** *of the larynx are met with chiefly in tubercle and syphilis, and in persons past middle life malignant disease may be a cause. The simple erosions present in catarrhal laryngitis hardly amount to ulceration. Ulceration is also found in the later stages of Lupus and Leprosy, usually when cutaneous lesions are present.*

§ 123. **Chronic Tuberculous Laryngitis** should always be suspected when delicate patients complain of constant hoarseness. This form of laryngitis is recognised by (1) the general pallor of the mucous membrane, accompanied by a thickening or swelling most marked over the arytenoids or the aryteno-epiglottic folds; (2) the occurrence of irregular, slowly growing ulcers, usually bilateral; and (3) the history or presence of pulmonary tuberculosis.

The *Prognosis* is always grave, and until recently recovery when the larynx was involved in tuberculosis was practically unknown. The course of the affection depends more upon the condition of the lungs (§ 94) than that of the larynx.

The *Treatment* at first is largely constitutional—*e.g.*, creosote in doses of 1 to 5 minims is recommended. Locally, menthol, one part to five of olive oil, used as paint, or an insufflation of menthol (8 grains) with iodoform and boracic acid (of each 1 drachm), is valuable. When ulceration has occurred, after being swabbed with cocaine and curetted, the parts should be thoroughly brushed with lactic acid, 10 to 60 per cent. This is a very favourite application. For the pain, which may be severe enough to cause dysphagia, morphia (¼ grain) with starch (½ grain), may be blown into the larynx; or it may be sprayed with 10 per cent. cocaine. Rest, a warm, dry climate, and sanatorium treatment, are indicated (§ 94).

§ 124. **Chronic Syphilitic Laryngitis.**—The laryngitis accompanying secondary syphilis may resemble simple catarrh, with the addition of whitish patches (§ 113). But that which occurs in the later stages nearly always takes the form of ulceration. The intensity of hyperæmia, the irritability, and the profuseness of the purulent discharge, are features of syphilitic ulceration. It is distinguished from the tuberculous ulceration by (1) the bright red coloration of the mucous membrane; (2) the presence of a deep, *rapidly growing ulcer*, with bright yellow surface, regular edges, often undermined, sometimes unilateral. If the ulcers invade the upper surface of the epiglottis, this is said to be pathognomonic of syphilis. (3) The presence of a syphilitic history.

Prognosis and Treatment.—This form of laryngitis is twice as rapid as, and far more destructive than, the preceding, and is liable to involve the cartilages (*vide* Perichondritis). Even when arrested considerable stenosis may result. The usual constitutional treatment must be carried out, full doses (60 to 100 grains) of potassium iodide being given. Local applications of iodoform, or a spray of perchloride of mercury (1 in 1,000), are employed.

(*c*) **Malignant Disease** and (in other countries) **Leprosy** give rise to ulceration of the larynx (see below.)

III. **Nodules and New Growths.**—*Flat localised thickenings of the mucous membrane are spoken of as warts, nodes, or nodules. When they*

are pedunculated they are spoken of as polypi. In either case they begin most frequently as a unilateral thickening on or near one of the vocal cords. In the early stage they are extremely difficult to distinguish from syphilis or tubercle, and sometimes this can be accomplished only by the history. With one important exception (singer's node) nodules are UNILATERAL, *and this feature of asymmetry distinguishes them from the thickening which may result from chronic laryngitis. The practical point of prime importance is the distinction of benign from malignant growths, often a task of considerable difficulty.*

§ 125. **Benign New Growths** begin most frequently as **warts,** nodules, or thickenings, the surface of which is smooth, although congested. They may give rise to no symptoms for a considerable time, unless they happen to be on the free edge of the cord. Perhaps the commonest of these growths is what is known as a **singer's node.** This lesion very often affects the under surface of the vocal cord, and hence may be overlooked for a long time. It is distinguished from other nodules by its frequent involvement of both sides symmetrically. A projection on one cord at the junction of the *anterior with the middle third* is probably a Singer's Wart; one situated at the junction of the *posterior with the middle third* is probably pachydermia laryngis. In the latter case there is a nipple on one cord which fits into a crater on the other. **Pachydermia Laryngis** is a localised chronic laryngitis (§ 122), usually most marked over the vocal processes. *Benign* nodules, as a class, are differentiated from *malignant* by the absence of pain and the paucity of symptoms of any kind. A pedunculated benign growth (**polypus**) of the larynx has the same clinical features, but is accompanied by very characteristic attacks of paroxysmal dyspnœa. **Leprosy** may affect the larynx.

§ 126. **Malignant Growths** start as thickenings of the mucous membrane, which may be like those of the preceding class, or they may be greyish-white, or they may have a ragged surface. In the course of a few months they invariably go on to erosion and ulceration. *Malignant* are differentiated from the *benign* growths by (1) the age of the patient and the family history; (2) the early onset of hoarseness, and sometimes pain, which may have preceded any other symptom for months; (3) their rapid and progressive nature, giving rise in the course of a few months to foul breath and hæmorrhage; and (4) the presence of secondary enlargement of glands in the neck. A diffuse unilateral congestion or *unilateral paralysis* in a person over middle age is always suggestive of malignant disease.

Prognosis and Treatment.—Benign growths often cause but little inconvenience. They are generally removable, without ulterior damage, by snares or cutting forceps. The *malignant* growths constitute one of the most rapid and fatal forms of the disease. Early extirpation of the larynx offers the best prospect, and, as in the case of Montagu Williams (whose larynx is now in St. Thomas's Hospital Museum), may afford immunity for two or three years.

IV. **Paralysfs of the Vocal Cords** *can be detected only by carefully inspecting both the* POSITION *and the* MOBILITY *of the cords during* (i.) *rest,* (ii.) *phonation, and* (iii.) *deep inspiration.*

§ 127. **Paralysis of the Vocal Cords.**—The chief actions of the larynx are (i.) Abduction (glottis-opening), which is performed by the posterior crico-arytenoids, and (ii.) Adduction (glottis-closing), which is performed by the lateral crico-arytenoids and the arytenoideus muscle. The cords are rendered tense by the crico-thyroids (external tensors), and are relaxed and shortened by the thyro-arytenoids (internal tensors—*i.e.*, the muscle which lies in the vocal cord). The larynx is supplied by two nerves, the superior laryngeal and the recurrent laryngeal branches of the vagus. The former supplies the crico-thyroid or tensor muscle and the mucous membrane of the larynx, while the recurrent laryngeal supplies all the other muscles. In progressive lesions of the recurrent nerve the abductors are paralysed first, and later on the adductors.

The Signs of Laryngeal Paralysis.—It is very rarely that a single muscle is paralysed; the paralysis nearly always affects a physiological group of muscles—*i.e.*, the glottis-openers (abductor paralysis) or glottis-closers (adductor paralysis on one or both sides. Paralysis is often accompanied by more or less catarrh, which modifies the appearance somewhat, but the evidences of laryngeal paralysis depend upon the position and mobility of the cords during phonation and respiration. The symptoms are given in Table XII. (p. 203).

Normally, during rest the cords are midway between open and closed (Fig. 51); during phonation they are approximated so that practically no space is left between them (Fig. 53); during deep inspiration they are widely opened (Fig. 50*a*).

Table XI.

Name of Muscle.	*Nerve Supply.*	*Action.*	
		Phonation.	*Respiration.*
Crico-thyroid or external tensor.	Superior laryngeal.	Tense and elongate the vocal cords.	—
Thyroid-arytenoid proprius[1] *internal tensor in cord itself.*	Recurrent laryngeal.	Adjusts edges of the cords.	—
Posterior crico-arytenoid.	Recurrent laryngeal.	—	Abduct—*i.e.*, open glottis.
Lateral crico-arytenoid.	Recurrent laryngeal.	—	Adduct—*i.e.*, close glottis.
Arytenoideus.	Superior laryngeal and recurrent laryngeal.	Close the glottis (posterior third chiefly).	—

When the cords are normal during phonation, but do not move out on inspiration, there is bilateral paralysis of the glottis-openers—*bilateral abductor paralysis* (Fig. 54). If both cords move during phonation, but one of them fails to move out fully during inspiration, there is *unilateral abductor paralysis* (Fig. 55).

When the cords neither move to the middle line with attempted phonation,

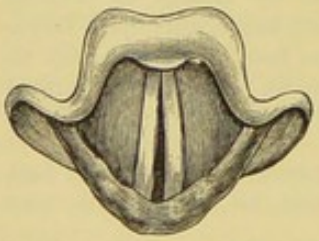

Fig. 51.—Moderate Adduction.—The appearance seen during rest.

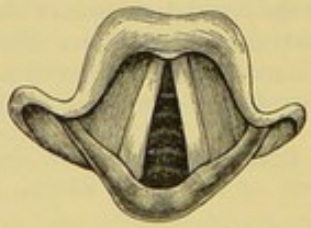

Fig. 52.—Cadaveric Position of cords.

Fig. 53.—Typical position during Phonation of high notes.

nor move as far outwards as normal during deep inspirations, but remain midway between the two in the cadaveric position (Fig. 52), there is *total bilateral paralysis of* adductors and abductors (Fig. 56).

If during phonation and inspiration one cord remains immobile, there is *total unilateral paralysis.*

[1] Lateral thyro-arytenoid is the lateral part of this muscle.

If there is aphonia, and on laryngoscopic examination the cords do not meet properly during attempted phonation, although they move outwards with inspiration, there is *bilateral adductor paralysis* (Figs. 57 and 58).[1]

TABLE XII.—LARYNGEAL PARALYSES.

(From Gowers, slightly modified.)

Lesion.	*Symptoms.*	*Signs.*
Bilateral abductor (opener) paralysis.	Voice little changed; cough normal; inspiration difficult and long, and attended with loud stridor.	Both cords near together; not separated during inspiration, but even drawn nearer together.
Unilateral abductor (opener) paralysis.	Symptoms inconclusive; little affection of voice or cough. Brassy cough sometimes.	One cord near the middle line not moving during inspiration, the other normal.
Total bilateral paralysis.	No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless (*i.e.*, the cadaveric position).
Total unilateral paralysis.	Voice low-pitched and hoarse; no cough; stridor absent or slight whilst breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.
Bilateral adductor (closer) paralysis.[2]	No voice; normal cough; no stridor or dyspnœa.	Cords normal in position, and moving normally during respiration, but not brought together on an attempt at phonation.

The *Etiology* of laryngeal paralyses differs considerably in the various forms. They may arise from ORGANIC or FUNCTIONAL conditions, but each is so characteristic that it can be readily identified. Thus *hysterical paralysis* is always double, and very nearly always due to adductor paralysis. Abductor paralysis is generally —and if unilateral is always—organic in origin. If the left vocal cord cannot be abducted, it is almost certainly due to pressure on the left recurrent laryngeal, and this in nine cases out of ten is due to *aneurysm of the aorta.*

(*a*) BILATERAL ABDUCTOR PARALYSIS (Fig. 54) may be due to—

(i.) The earlier stages of *pressure* upon both recurrent laryngeal nerves, as by mediastinal tumour, or pericardial effusion (§ 54).

(ii.) *Peripheral* neuritis from toxins (such as diphtheria, alcoholism, influenza), certain drugs (*e.g.*, lead, arsenic), or simple catarrh.

(iii.) *Central Causes*, as in lesions affecting the medulla or base of the brain, bulbar paralysis, cerebral tumours or syphilis, hæmorrhage into the bulb, tabes dorsalis, disseminated sclerosis, meningeal conditions, etc.

(*b*) UNILATERAL ABDUCTOR PARALYSIS (Fig. 55) is due to the same causes acting on one side only. Thus, if on the *left side*, it is due in nine cases out of ten to *aneurysm of the aorta*, although no other signs of that condition may be present. Malignant tumour of the œsophagus may also affect the left recurrent laryngeal. Thickened right pleura may be the cause of a paralysed right recurrent laryngeal. Pressure upon the vagus in the neck, as by an enlarged thyroid, or cervical glands, may affect one or both sides.

[1] Further particulars of the actions of the various muscles may be found in a study of laryngeal paralyses since the introduction of the laryngoscope, by Sir Felix Semon (*Brain*, 1892, vol. xv., p. 471).

[2] *Unilateral* adductor paralysis is practically unknown, though tubercular or rheumatic ankylosis of the crico-arytenoid joint may cause immobility of one cord.

(*c*) Total (Ab- and Adductor) Bilateral Paralysis (Fig. 56) is practically always of organic origin, but it may (rarely) be due to catarrh or hysteria. It may arise from any of the causes mentioned under Bilateral Abductor Paralysis, but is most frequently of *central* origin. It occurs later in the disease than abductor paralysis, the abductor fibres in the nerve being the first to be affected.

(*d*) Total (Ab- and Adductor) Unilateral Paralysis is due to the same causes as mentioned under unilateral abductor paralysis—*i.e.*, usually pressure upon the recurrent laryngeal. This condition, however, occurs at a later stage in the case, unilateral abductor paralysis being a feature of the earlier stage. Total paralysis is sometimes called "recurrent paralysis," because it is due to paralysis of the recurrent laryngeal.

(*e*) Bilateral Adductor Paralysis (Figs. 57 and 58) is always *functional* (viz., unconnected with *gross lesions*); (1) hysterical; (2) simple catarrh, or over-use of the voice; (3) general weakness, as in anæmia. But the first of these is by far the most common.

Prognosis.—Laryngeal paralysis is generally only a minor element in the case. When occurring alone, however, the prognosis in adductor paralysis is good, because it is always of functional origin. Paralysis arising from syphilis is remediable if treated early. In all forms, however, the prognosis depends upon whether the cause is removable or not.

Fig. 54.—Bilateral Paralysis of the Glottis-openers (Bilateral Abductor Paralysis).—The patient is able to oppose the cords during phonation, but *the cords do not move outwards during deep inspiration* (as in Figs. 50 and 50A).

The same appearance as the above is sometimes produced by acute laryngeal catarrh, but the cords would be pink instead of white.

Treatment.—Hysterical paralysis should be treated on lines laid down elsewhere. Strong faradisation or static electricity to the larynx is indicated, the patient being instructed to call out loudly. In organic paralyses the prognosis depends upon the cause. Potassium iodide should receive a fair trial. Strychnine and electricity are useful. In organic cases, if dyspnœa be severe, tracheotomy must be performed.

Illustrations of Laryngeal Paralyses.—It should be remembered, in studying these illustrations, that to test the motor power of the vocal cords it is necessary to make the patient INSPIRE deeply to OPEN the cords, then to PHONATE, so as to CLOSE the cords, for a given position of the cords conveys no information unless it is first known which of these acts the patient is performing.

In laryngeal paralysis it is very important to decide whether a functional or organic cause is in operation, and the following hints should be remembered:

1. Glottis-closer (adductor) paralysis is generally functional; glottis-**O**pener (abductor) paralysis generally **O**rganic.
2. Bilateral paralysis is generally functional; **O**ne-sided paralysis is generally **O**rganic.
3. Left **A**bductor (glottis-opener) paralysis suggests **A**neurysm.

V. Spasm of the Laryngeal Muscles *and consequent* Inspiratory Dyspnœa, *is not a very common occurrence, except in the form of Laryngismus Stridulus, a disease almost confined to childhood. It may arise when a foreign body passes into the larynx, and may occasionally occur in adults who are the subjects of acute laryngitis. Inspiratory dyspnœa may also arise in Bilateral Abductor Paralysis.*

§ **128. Laryngismus Stridulus or Nervous Croup**[1] is a form of paroxysmal inspiratory dyspnœa. It consists of a sudden spasmodic closure

[1] Synonyms: Spasmus glottidis, spasmodic croup, child-crowing, spasm of the larynx.

of the glottis, followed by a long noisy inspiration which produces a crowing sound, and is due to spasm of the adductors. It is a nervous affection, and appears to be due to some irritation of the vagus or of its recurrent laryngeal branch. The whole attack lasts from a few seconds to a minute or two. The child may become cyanosed or the spasms may spread to other muscles and give rise to general convulsions. Occasionally it terminates fatally. The attacks come on either during sleep, or in the waking state. They are very apt to recur, and the severity of the attacks may increase at each recurrence. On the other

PATIENT'S RIGHT.

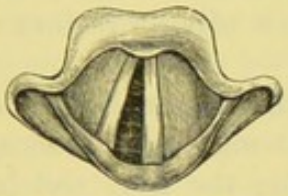

Fig. 55.—LEFT ABDUCTOR, or glottis-opener, paralysis.—DURING INSPIRATION the *left cord* remains fixed, instead of moving outward like the right cord does. This occurs in early paralysis of the recurrent laryngeal nerve of ORGANIC ORIGIN—*e.g.*, aneurysm.

Fig. 56.—TOTAL BILATERAL paralysis.—DURING INSPIRATION and DURING PHONATION *both cords* are immobile, and remain in what is practically the cadaveric position. Nearly always of ORGANIC origin, and frequently central.

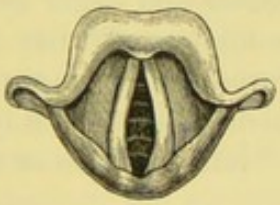

Fig. 57.

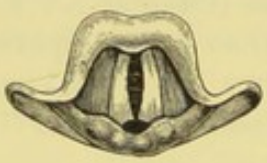

Fig. 58.

Figs. 57 and 58.—PARTIAL BILATERAL ADDUCTOR, or glottis-closer, paralysis.—It is the condition commonly met with in hysterical or FUNCTIONAL aphonia. DURING PHONATION the cords close anteriorly and posteriorly, but leave an elliptical space between them. The glottis is closed by two muscles—the crico-thyroid in front, and the arytenoideus behind. If the CRICO-THYROID is mainly affected, the condition depicted in Fig. 57 is seen, and it is met with in functional aphonia and exhaustion. The ARYTENOIDEUS closes the posterior angle, and when this is paralysed the posterior angle remains open (Fig. 58). Both of these forms are met with in acute and chronic laryngitis, and are generally independent of any actual nerve lesion, excepting perhaps peripheral neuritis and some rare cases due to a local lesion affecting the recurrent laryngeal nerve of both sides.

PATIENT'S LEFT.

hand, if the attacks are slight, they may gradually disappear as the child grows older. In the intervals the child is free from cough or hoarseness, and the larynx appears healthy.

The *Etiology* is obscure. It is practically confined to children of from four months to two years old, and nine-tenths of these are rachitic—that is to say, children in whom infantile convulsions and tetany are also apt to arise. It is twice as common in boys. It is more frequent in the spring time, and it is often hereditary. In older subjects laryngeal spasm and inspiratory dyspnœa occur sometimes in tabes dorsalis, when it forms the laryngeal crisis of that disease. Its rarer causes are epilepsy, hysteria, tetany, chorea, reflex irritation of the vagus or its

recurrent laryngeal branch from mediastinal growths, a growth or foreign body in the larynx, or too long a uvula.

The *Diagnosis* is not difficult, though it is well to bear in mind the possibility of a foreign body in the throat, larynx, or trachea. There are, however, three pathological conditions to which the term "croup" is loosely applied, and which consist essentially of a PAROXYSMAL INSPIRATORY DYSPNŒA.

1. *Nervous Croup*, or laryngismus stridulus, is the non-inflammatory nervous affection described above. This is recognised by the absence of cough, hoarseness and other symptoms referable to the larynx in the intervals between the attacks. There is often a history of similar attacks.

2. *Catarrhal Croup*, or spasmodic laryngitis, consists of attacks of dyspnœa, coming on for the most part at night, in children suffering from a little cough and hoarseness during the day, and lasting for an hour or so. This is due to the collection of thick secretion, or to the sticking together of the edges of the glottis from slight laryngeal catarrh. It should not be confounded, as it often is, with nervous croup.

3. *Membranous Croup*, or laryngeal diphtheria.—This is true diphtheria, and is attended by the constitutional and other symptoms of that disease (Chapter XV.). However, some (*e.g.*, Whitla and others) maintain that a non-diphtheritic membranous croup may occur. A severe injury (*e.g.*, drinking out of a boiling kettle) may certainly result in a membranous or "diphtheritic" inflammation of the mucous membrane.

Treatment of Nervous Croup.—(*a*) *For the Attacks.*—Cold water may be dashed in the face, or the patient plunged into a hot bath, or alternately hot and cold, or cold water douches applied. The inhalation of chloroform relieves it promptly. Artificial respiration is often of great service, and it may restore, even after apparent death. In the rare cases in which the spasm is prolonged and continuous, tracheotomy may be necessary.

(*b*) *For the Intervals.*—The patient should be kept very quiet, and irritation of the surface or the application of any stimuli conducive to an attack should be avoided. Reflex causes of irritation should be sought in the gums (*e.g.*, teething), in alimentary canal (*e.g.*, worms or gastric disorder), in the lungs and elsewhere (*vide* causes). The general treatment of rickets should be adopted, and it is worth bearing in mind that children taken into the country very often cease to have these attacks. Sponging with cold water twice or three times a day is of value; and as to medicine, bromides and chloral in small doses allay the irritability of the nervous system, on which the condition mainly depends. Faradisation of the pneumogastric is sometimes useful.

VI. and VII. **Diseases of the Pharynx** (*ante*) **and of the Nose** (*post*) *are generally attended by a certain amount of hoarseness and alteration of the voice.* The latter give to the voice a peculiar nasal twang, which is very characteristic.

The Nasal Cavities.

§ **129. Symptoms and Physical Examination.**—Diseases of the nose will be considered under three cardinal SYMPTOMS : *Inodorous discharge* from the nose (Rhinorrhœa) ; *foul discharge* from the nose (Ozœna) ; *mouth-breathing* and snoring (Obstruction of one or both Nostrils). *Bleeding* from the nose also occurs in some nasal disorders, but it is *not* a cardinal symptom. It is perhaps more generally associated with some constitutional or general derangement. *Sneezing*, *tickling* in the nose and *sniffing* may also be present ; and the quality of the *voice* may be altered, particularly in nasal obstruction. The sense of *smell* is always disturbed to some extent in nasal disorders. In some instances, headache, vertigo, and other nervous derangements are met with in association with disorders of the nose, especially when the free transit of air through the nasal passages is interfered with, and the atmospheric pressure within the tympanum disturbed.[1] Various constitutional symptoms may result from septic conditions of the nose or the adjacent sinuses, and not infrequently a patient suffers from listlessness and general debility for a long time before our attention is directed to the true source of his troubles.

Clinical Investigation.—Rhinoscopy or examination of the nose may be effected through the anterior nares (anterior rhinoscopy), and the posterior nares (posterior rhinoscopy) ; and by digital examination posteriorly.

Fig. 59.—NASAL SPECULUM.

ANTERIOR RHINOSCOPY.—First examine the anterior nares for any obvious disorder, such as fissures, ulcers, scars from ulcers, any narrowing of the nares, or a deviation of the septum ; secondly, introduce a speculum (Fig. 59), using either a direct light or one reflected from a mirror on the forehead, as in laryngoscopy. In this way an examination of the inferior turbinate bone can be made, to see if it be hypertrophied. The inferior or middle meatus can be thus examined for polypi or alteration in the mucous membrane. If, as frequently happens, the anterior part of the inferior turbinate is hypertrophied, and hides the view, this may be reduced by swabbing out with a cotton-wool pledget soaked in a 10 per cent. solution of cocaine.

POSTERIOR RHINOSCOPY is effected by precisely the same procedure

[1] A notable instance in my own experience was that of a lady of thirty-five who suffered from most troublesome tinnitus aurium and occasional giddiness, which was not relieved until the middle turbinate bone was removed by Dr. Scanes Spicer (see the Author's " Clinical Lectures on Neurasthenia," fourth edition).

as in laryngoscopy (§ 119), using the smallest of the mirrors, and turning it upwards. It is convenient to have a special mirror for this purpose mounted on a curved handle, the stem being hinged at its extremity, so that it can be turned to any desired angle. It is important to avoid touching either the dorsum of the tongue or the posterior wall of the pharynx. The patient should be instructed to breathe gently all the while through the nose. By moving the mirror slightly in different directions we are able to examine the posterior nares and turbinated bones, the inner end of the Eustachian tube for any swelling, and Luschka's tonsil (*cf.* Fig. 49). The pharyngeal or Luschka's tonsil is a mass of lymphoid tissue on the pharyngeal roof and posterior wall above and between the Eustachian tubes; when in a condition of hyperplasia it forms the cushion-like growth of post-nasal adenoids (§ 109).

A great deal of information may be derived by passing the finger behind the soft palate, but for this purpose it is generally necessary to spray the pharynx with cocaine (10 per cent.).

Our *first* inquiries concerning any given case of suspected disease of the nose should be relative to the LEADING SYMPTOM, especially whether there be any nasal discharge, and whether it is inodorous or foul smelling. We cannot depend upon the patient's statement on this point, because very often the same disease which causes a foul discharge may blunt the sense of smell. *Secondly*, we must investigate the HISTORY, and whether any of the other symptoms above mentioned were present. *Thirdly*, we must proceed to the PHYSICAL EXAMINATION by testing whether the patient can breathe freely through each nostril separately; by examining the anterior, and, if necessary, the posterior nares.

Classification.—Diseases of the nose, like those of the throat, are best classified by the PHYSICAL SIGNS met with on examination—viz., **nasal discharge, nasal obstruction, epistaxis**—and their **causes.**

(*a*) ACUTE INODOROUS DISCHARGES (Acute Rhinorrhœa)—the causes of which are—

I. Acute Rhinitis; II. Syphilis (snuffles); III. Diphtheria, and other fevers; IV. Coryza; V. Hay Fever; VI. Glanders.

(*b*) CHRONIC INODOROUS DISCHARGES (Chronic Rhinorrhœa)—the causes of which are—

I. Chronic Simple Rhinitis; II. Chronic Hypertrophic Rhinitis; III. Cerebro-spinal Rhinorrhœa; IV. Ulcerations of the Nose, Polypi, and occasionally Catarrh of the Sinuses.

(*c*) CHRONIC OFFENSIVE DISCHARGES (Ozœna), which have for causes—

I. Ulcerations and Bone Disease—Syphilis, Tubercle, and Lupus; II. Atrophic Rhinitis; III. Empyema of Antrum and other Sinuses; IV. New growths and polypi breaking down, and impacted foreign body.

(*d*) Nasal Obstruction (Snoring, and mouth-breathing)—the causes of which are—

I. Pharyngeal Adenoids; II. Polypi; III. Deviated Septum; IV. Hypertrophy of Turbinate; and V. Foreign body and neoplasms in adjacent parts.

(*e*) Epistaxis, the causes of which may be Local or General.

§ **130. Acute** (or recent) **Inodorous Discharge from the Nose (Rhinorrhœa).**—*Discharge is a frequent symptom when disease of the nose is present, and we should endeavour to ascertain if this be odourless or offensive, although these are, of course, only relative terms, and the two groups cannot be sharply defined. Among the causes of* Acute Inodorous Discharge, *congenital* Syphilis *should be suspected in infancy;* Diphtheria *in childhood;* Coryza *in adults.*

I. **Acute Rhinitis** may be set up by *irritation* of any kind, as the vapour or dust of some trade, or by any injury. For instance, a profuse discharge from one nostril in a child should always make us suspicious of his having inserted a pea, marble, or other *foreign body*, although the history may be wanting. But its commonest cause is "a cold" (see Acute Coryza below).

II. "**The Snuffles.**"—In newly-born children, congenital syphilis is almost invariably attended by a profuse nasal catarrh, and is known familiarly as the "Snuffles." The other features of nasal syphilis will be referred to under Ulcerations.

III. **Diphtheria** and other fevers.—In diphtheria a profuse nasal discharge excoriating the upper lip, with slight elevation of temperature, and prostration, coming on suddenly in a child or young person previously healthy, is so characteristic that the disease may be almost diagnosed from these features alone.

IV. In **Acute Coryza**, "catarrh," or "cold in the head," there is profuse muco-purulent discharge attended by sneezing, running from the eyes, and febrile symptoms with frontal headache, extending over a few days. It is usually attributed to some exposure to cold ("a chill"); but it frequently prevails in an epidemic form, and is then of microbic origin. It is predisposed to by cold and damp weather, by adenoids, and the other causes of chronic rhinitis. It is not a serious disorder, but its repeated occurrence may lead to middle-ear catarrh, or to bronchitis by extension.

Treatment of "Catarrh."—In severe cases it is advisable for the patient to keep in bed. At the outset a full dose of Dover's powder given at night, or a mixture of tr. aconiti ♏ i., liquor ammoniæ acetatis, with other salines, every two hours, may cut short the disease. Locally, sprays of cocaine (2 to 4 per cent., applied with caution, occasionally), or equal parts of boracic acid and borax dissolved in water, or camphor and menthol (gr. 8 to ℥i. of paroleine), may abort the disease.

Ferrier's snuff[1] is also useful. Inhalations of camphor, menthol, or vinegar taken at night are reputed to be efficacious.

V. **Hay Fever,** or, as it is sometimes called, **Hay Asthma,** is a severe catarrh of the nasal mucous membrane and conjunctivæ, coming on fairly regularly in the summer or autumn of each year, presumably connected with the pollen of flowers. It is accompanied by the symptoms of severe coryza, just described, which come on somewhat suddenly in a person predisposed, who has been outdoors (usually in the hay-making season), and are attended by a certain amount of constitutional disturbance. There appear to be two clinical varieties of this disease—(i.) where the symptoms are chiefly constitutional ; and (ii.) where the symptoms are chiefly local. In the latter there is generally hypertrophy of the inferior turbinate, which constitutes an important predisposing factor.

The *Etiology* of this disease is somewhat obscure, but it evidently is connected in some way with pollen, especially that of grasses. There are some people, usually those with a marked neurotic taint, who cannot go within a couple of miles of a hay-field in the summer without developing the disease. It is diagnosed from simple coryza chiefly by its seasonal occurrence. It resembles asthma in some respects, especially in its periodicity, but differs in that the nasal, instead of the bronchial, mucous membrane is involved. The malady is not a fatal one, but causes serious discomfort and inconvenience. Sometimes people get rid of it as they get older, but in others it continues throughout life.

Treatment.—The first indication is the avoidance of the cause. This may be accomplished by a sea-voyage, residence at the seaside at a high altitude, or by living indoors in the city, taking care that no plants or flowers enter the house. But there is no rule in this respect ; for some do better at a high altitude, others at a low one ; some get better at the seaside, others in a town. Quinine, arsenic, iron, or belladonna, may be taken before the attack is expected. Antipyrin (15 grains) has been credited with cutting short an attack. If the disease extends to the bronchi, asthma papers and cigarettes should be employed. Locally, means should be taken to prevent the pollen reaching the mucous membrane. For this purpose Brunton recommends smearing the nostrils with zinc oxide ointment, which not only allays the irritation, but, by remaining longer unmelted, is more efficacious than other ointments. Antiseptic sprays destroy the pollen. Of these quinine, ½ grain to the ounce, dissolved in normal saline solution, as being less irritating than water, gives good results. Sir Andrew Clark recommended swabbing out with hyd. perchlor., gr. i. ; quin. hydrochlor., gr. ii. ; glyc. ac. carbol., ℥ii. To relieve the discomfort, cocaine tabloids (¼ grain), inserted in the nose, sprays of cocaine (4 per cent.) or menthol (20 per cent.) are used. The mucous membrane, if thickened, must be treated as in hypertrophic rhinitis. Pollantin has been greatly recommended.

VI. **Glanders.**—The copious discharge of viscid semi-purulent matter from the nostrils is one of the earliest symptoms of Farcy, or Chronic Glanders (Chapter XV.).

§ 131. In **Chronic Nasal Discharges** *it is still more difficult to draw the line between odorous and inodorous discharges, since many of the conditions, though odourless at the outset, become offensive later on, and it will generally be necessary to pass in review all the conditions mentioned in this section and* § 132 below. The following are the chief causes of INODOROUS DISCHARGE :

I. **Chronic Rhinitis** is a chronic inflammatory condition of the mucous membrane of the nose, attended by increased secretion, and usually by thickening. It occurs in three forms : (*a*) SIMPLE ;

[1] Bismuth Subnitrate, ℥vi. ; Morph. Hydrochlor., gr. ii. ; Pulv. Acac., ℥ii.

(*b*) HYPERTROPHIC (*infra*) ; (*c*) ATROPHIC (§ 132). The first two give rise to an *inodorous*, but the ATROPHIC to an *odorous* discharge.

CHRONIC SIMPLE RHINITIS consists of a chronic congested, and sometimes, later on, a hypertrophied state of the mucous lining of the nose, attended by a continuous mucous or muco-purulent discharge. There is generally a certain amount of nasal obstruction, giving rise to altered voice and snoring.

Etiology.—(i.) It is predisposed to by cardiac and pulmonary disease, alcoholism, and the strumous diathesis. It may be *determined* by (ii.) recurrent attacks of neglected coryza over a long period of time ; (iii.) the injury caused by an unsuspected foreign body, in which case the condition is generally confined to one side ; or (iv.) the constant irritation of dust and noxious vapours—*e.g.*, masons, fustian-cutters. (v.) It is often associated with adenoids, enlarged tonsils, and other causes of obstruction to the nasal respiration.

Prognosis. The disease is chronic, and requires prolonged treatment. The chief fear is that middle-ear catarrh may result from the extension of the inflammation up the Eustachian tube. Even apart from this, it is very important to treat these cases in strumous children, because the condition interferes with the respiratory functions of the body.

Treatment.—In the early stages alkaline washes—bicarbonate of sodium, gr. xv., and borax, gr. v., or carbolic acid, gr. iii. to $\bar{3}$i.—sniffed up or given by the nasal douche.[1] This is followed later on by a spray of menthol and eucalyptol (gr. xxx. to $\bar{3}$i. of aquol or paroleine), or an ointment of cocaine and thymol (gr. x. to $\bar{3}$i. of white vaseline), or by the use of the ammonium chloride inhaler. Constitutional treatment is necessary, by means of tonics, cod-liver oil, and malt. Alcohol should be avoided, and a high and dry climate should be sought. In the later stages, the only satisfactory method of treatment is applying chromic acid (gr. v. or x. to $\bar{3}$i.), or, still better, the galvano-cautery.

II. **Chronic Hypertrophic Rhinitis** is a special form distinguished from the preceding by the fact that there is considerable hyperplasia of the nasal mucous membrane, especially over the inferior turbinate bone at its anterior and posterior ends. It presents the same symptoms as the preceding, but in a greater degree. Even in slight cases it is apt to be accompanied by headache and mental depression. It is frequently associated with adenoids. The *Prognosis* is on the whole less favourable. The *Treatment* is much the same, but more active measures are indicated, and especially treatment by the thermo-cautery.

III. **Cerebro-spinal Rhinorrhœa.**—Dr. St. Clair Thomson[2] has shown that the cerebro-spinal fluid sometimes escapes from the skull, probably through the peri-

[1] A nasal douche is always preferable, but it requires practice for its use. A very imperfect substitute is to fill the palm of the hand with the liquid, and sniff it up the nose.

[2] "The Cerebro-spinal Fluid : Its Spontaneous Escape from the Nose," by St. Clair Thomson (Cassell and Co., London, 1899) ; and see also *Brit. Med. Journ.*, September 23, 1892, p. 794.

neural sheaths of the olfactory nerves, and probably owing to some pathological change leading to increased intracranial pressure. It is *recognised* by: (1) The constant andlong-continued escape from the nostril of a perfectly clear watery fluid. This alone should arouse our suspicion. (2) The fluid has no odour, taste, nor sediment; is free from albumin and mucin, and reduces Fehling's solution. This last is absolutely distinctive.

IV. **Ulcerations of the Nose, Polypi, Disease of the Sinuses,** occasionally produce inodorous discharges, but the discharge is more often offensive (see below). CHRONIC FRONTAL SINUS EMPYEMA is, however, attended by (i.) a purulent, *non-fœtid* nasal discharge,[1] (ii.) frontal or supra-orbital headache or feelings of discomfort, and (iii.) more or less well-marked nasal obstruction, caused by inflammatory enlargement of the middle turbinated body, or by polypi. Headache only occurs from retention, and not when drainage is free. There may be tenderness on pressure over the affected side.

§ **132. Ozœna** or a **Chronic Offensive Discharge** *from the nose may occur in the later stages of* MANY OF THE CONDITIONS *mentioned in the preceding section. But the chief causes of foul discharge from the nose are as follows; the commonest and foullest of these being* SYPHILITIC DISEASE *in middle life, and* CANCER *in the aged.*

Foreign bodies (*which have already been referred to*) *and* Polypi, *both of which may cause one-sided ozœna, will be described under Nasal Obstruction* (§ 133) *which is their leading symptom. It will be necessary to give some detailed account of*—Ulcerations (and Bone disease); Atrophic Rhinitis; and Empyema of the Sinuses.

I. **Ulcerations and Bone Disease** attacking the nose are mostly of syphilitic, occasionally of tuberculous, origin. Neoplasms in the later stages ulcerate, but in the earlier stages give rise to Rhinitis or Nasal Obstruction (§ 133).

(*a*) **Syphilitic Rhinitis.**—In the early stages of syphilitic infection we may get an acute catarrh with superficial ulceration, which is the condition found in children with congenital syphilis, known as "snuffles." In the later stages gummata form in various situations, which *rapidly involve the bone* and other parts; the discharge then becomes very foul. The ulcers have the same character as those affecting the throat (*q.v.*).

(*b*) **Tuberculous Ulceration** more often involves that part of the nose near the orifice, but otherwise the ulcers much resemble the preceding. They are differentiated from them by their very much slower progress, as well as by their site. The bones are rarely attacked, and consequently the discharge may be more or less inodorous; and there is rarely the falling in of the bridge of the nose, which so frequently occurs in tertiary syphilis. The ulceration of *Lupus* differs but little from the true tuberculous ulceration, except that lupus vulgaris usually involves also the skin of the alæ nasi, *whence it has probably spread.*

Atrophic rhinitis is distinguished from these ulcerations by the pallor and thinning of the mucous membrane, the absence of visible ulcers,

[1] Dr. Herbert Tilley, the *Lancet*, July 14, 1900.

and the absence of a history or evidences of syphilis or tubercle respectively.

The *Prognosis* of nasal ulceration is fairly good if the patient come under treatment early, but if not it leads to considerable destruction of tissue. Tuberculous ulceration may slowly lead to the destruction of the *alæ* of the nose, but syphilis results in the most extensive destruction of the *bones* both of the septum and the palate ; the bridge of the nose falls in, and the anterior nares may be represented by a single gaping orifice. It is this extensive and rapid destruction which is so pathognomonic of nasal syphilis.

The *Treatment* should be much more prompt and vigorous in ulceration of the nose than in chronic rhinitis and similar affections, because of the destruction which ensues. Carbolic and astringent sprays are useful palliatives, but surgical measures are called for if the bone is involved. All dead bone must be removed. Tuberculous ulcers should be scraped. Large doses of potassium iodide lead to rapid healing of syphilitic ulcerations.

II. **Atrophic Rhinitis,** also known as idiopathic or true ozœna, is characterised by (i.) a thick, foul discharge, which is sometimes profuse, sometimes scanty; (ii.) the nasal cavities are often large, and the bridge of the nose broad and sometimes depressed. The mucous membrane is thin, pale, and covered with crusts, hard, adherent, and decomposing. Sometimes it is unilateral—*e.g.*, in cases of deviated septum. A certain amount of chronic pharyngitis is usually present. (iii.) The breath has a foul odour, which is not detected by the patient, as the sense of smell is blunted. It is *Diagnosed* from the other causes of ozœna by the absence of ulceration, the presence of atrophied mucous membrane, and wide cavities.

Etiology.—(i.) It is commoner in the young and in women. It usually starts before sixteen years of age. (ii.) Unilateral atrophic rhinitis is mostly due to some local cause, such as a deviated septum or sinus disease, the narrower side being healthy. (iii.) The exciting causes of bilateral atrophic rhinitis are obscure : it has been said to follow chronic rhinitis in strumous children ; (iv.) in some cases it is a sequence of hypertrophic rhinitis.

Prognosis.—Prolonged treatment is necessary for its cure, and even this is not very hopeful if the disease be advanced. The disorder is generally most marked at about twenty years of age ; it becomes less troublesome at middle age, and, as it gradually disappears with advancing years, we may presume that it tends slowly to spontaneous cure.

Treatment.—Alkaline and antiseptic douches and sprays are indicated, as in § 131. To stimulate the mucous membrane, nasal tampons of cotton-wool, soaked in glycerine, are used. These are useful in unilateral rhinitis, as they insure respiration through the narrower cavity. The nose may be swabbed out with silver nitrate (10 grains to the ounce), or with trichloracetic acid (5 to 20 parts in 1,000), which removes the smell. Constitutional treatment is also advisable.

III. **Chronic Empyema of the Antrum** and other sinuses is a term applied to a chronic suppurative inflammation of the lining membrane, though the term " empyema " should be reserved for cases in which there is retention. The most constant and cardinal symptom is a purulent or sero-purulent discharge from *one nostril*, which is generally offensive or sickly. It may arise as an extension of nasal catarrh, or various suppurative nasal conditions (syphilis, tubercle, bone disease, etc.). A probe cannot be introduced if the orifice communicating with the nasal cavity is blocked.

Empyema of the Antrum may be due to irritation from a tooth. Many of the patients have had decayed teeth in the upper jaw. It is recognised by the dis-

charge being intermittent, returning usually about the same time each day, and flowing freely when the head lies on the opposite side, or is lowered between the knees. The discharge may be seen coming from beneath the middle turbinate. If a bright light is held in the mouth, the cheek of the affected side remains darker than the other (trans-illumination).

Discharge from the *frontal* or *anterior ethmoidal* sinuses flows best when the patient is upright. It comes from under the middle turbinate, and there is often pain in the brow and orbit (see also p. 212).

Discharge from the *posterior ethmoidal* and *sphenoidal* sinuses flows over the middle turbinate and down into the pharynx. There may be exophthalmos, ptosis, strabismus, etc., with disease in this locality.

Various constitutional symptoms have recently been recognised as being associated with sinus disease, due probably to the toxæmia which results from septic absorption.[1] Lassitude, headache, occasional elevations of temperature, and numerous nervous and vaso-motor symptoms are among the commonest. They generally present a periodic or paroxysmal character. Trifacial neuralgia may also result from sinus disease. If overlooked or neglected, empyema may excite middle-ear catarrh (with tinnitus, deafness, etc.) recurrent nasal catarrh, and nasal polypi.

Prognosis and Treatment.—Sinus empyema is chronic and intractable, but very rarely fatal. The treatment is based on surgical principles, but the chief indications are free drainage, stimulation of the chronic inflammation until it takes on a more healthy action of repair, or scraping.

IV. **Neoplasms and Polypi** (§ 133), and **Impacted Foreign Body** (§ 130, I.), are referred to elsewhere.

§ **133. Nasal Obstruction, Snoring, and Mouth-breathing.**—*Nasal obstruction may be partial or complete, and it may exist on one or both sides It is met with in a greater or less degree in nearly all of the various nasal conditions previously discussed, and it is a marked feature in* Hypertrophic Rhinitis (p. 211). *Its commonest cause in children is* Pharyngeal Adenoids (p. 188). *It is also a cardinal symptom in* Nasal Polypi, Deviation or Spur of the Septum, Alar Collapse, Foreign Bodies, Neoplasms, *and* Abscesses.

Effects.—Apart from the inconvenience of snoring, nasal obstruction renders the individual prone to pharyngitis, stomatitis, bronchial catarrh, and other consequences due to the entry of cold air into the lungs without being properly warmed by its passage through the nose. Among the other consequences are a nasal quality of the voice, distortion of the chest (when arising early in life), and impeded respiratory functions of the body generally. These disorders consequently assume an importance quite out of proportion to the degree of local mischief.

I. **Pharyngeal Adenoids** are of very frequent occurrence. They constitute one of the forms of granular pharyngitis, and the disease has been referred to under that condition (§ 109). It is the most frequent cause of mouth-breathing and snoring in children. It is often overlooked by parents, a circumstance greatly to be regretted for three reasons. In the first place, it is one of the most potent causes of chronic otitis media and deafness in after-life; secondly, it impairs the respiratory

[1] See also "A Treatise on Nasal Suppuration," by Dr. Ludwig Grünwald, translated by Wm. Lamb. London, 1900.

functions of the body, as just mentioned; and thirdly, the open mouth and vacant aspect, which are so characteristic, produce an appearance of backward intelligence, which in point of fact sometimes also exists.[1]

II. **Polypi,** or pedunculated tumours, are the most frequent new growths in the nose. Polypi are of three kinds: (*a*) GELATINOUS; (*b*) FIBROUS; and (*c*) MALIGNANT.

(*a*) GELATINOUS or MUCOUS POLYPI are the most common form of polypi. They usually consist of myxomatous tissue, and are generally associated with inflammatory disease of the subjacent bone. They are often multiple, and most often grow from the muco-periosteum of the upper and middle turbinated bones. Their detection is not difficult, for in addition to the feeling of "stuffiness" and the watery discharge (which may be intermittent), they are easily seen through the nasal speculum as pale grey glistening bodies. They are apt to recur after removal, but are not malignant in other respects.

(*b*) FIBROUS POLYPI grow from the roof of the naso-pharynx. By their growth they displace the parts around and are apt to give rise to "frog-face." The discharge is often foul, and may be hæmorrhagic. They sometimes become malignant (fibro-sarcoma). They may occur at any age.

(*c*) MALIGNANT POLYPI may be either fibro-sarcomatous or carcinomatous. They are known by their rapid growth, and the resulting deformity of the face, "frog-face," and by the offensive and hæmorrhagic discharge. Sarcomatous growths are chiefly met with in the young; carcinomatous in the aged.

Prognosis and Treatment.—The benign polypi are not dangerous to life, but are liable to recur. Malignant growths give rise to a condition of considerable gravity. Occasionally fibrous tumours atrophy. Gelatinous polypi, arising as they do from the anterior part of the cavity, can generally be removed by means of a nasal snare or forceps; but the other varieties, springing usually from the posterior parts and infiltrating the tissues around, may require an operation of some magnitude.

III. **Deviated Septum and Nasal Spur.**—The nasal septum is rarely quite in the median line, but the displacement is often considerable. Sometimes it results from injury. Various consequences may ensue, such as hypertrophied turbinate on one side, atrophic rhinitis on the other. When an angle is formed in the septum nasi, it is spoken of as a "spur," and this is most readily dealt with by sawing it off.

IV. **Hypertrophied Turbinate** is met with usually either as part of, or a consequence of, chronic hypertrophic rhinitis. It may occur on one or both sides, and in either case, in narrow nostrils, produces partial obstruction, snoring, and mouth-breathing. It is removable by turbinectomy; sometimes the thermo-cautery is employed (see Hypertrophic Rhinitis, p. 211).

V. **Foreign Bodies** within the nose, **Neoplasms,** and **Abscesses** in adjacent parts may also produce *unilateral* nasal obstruction.

[1] This result is not altogether explicable by faulty respiratory processes of the body; nevertheless, I am satisfied that the intelligence in some children has been dull until the pharyngeal defect has been remedied.

§ **134. Epistaxis** (bleeding from the nose) may be a symptom of nasal disorders, but if in any appreciable quantity it is usually an evidence of some general disorder. Not infrequently both general and local causes are in operation, and the *nasal cavities should always be carefully examined*. The bloodvessels give way in this situation (sometimes as a kind of safety-valve) merely because they are thin-walled, numerous, and near the surface. So much is this the case that the diminished atmospheric pressure to which mountaineers are subjected is sufficient to produce nose bleeding when they reach great heights. The *Causes* may be divided into two groups—Local and Constitutional.

(*a*) Local Causes, in which the hæmorrhage consists usually of little more than streaks, may arise from any marked congestion of the mucous membranes, such as that which accompanies adenoids, acute rhinitis, or worms in the nose ;[1] or as a consequence of mechanical violence, applied either directly to the nose or to the base of the skull. Any serious destructive disorder—such as new growths, especially malignant, syphilitic, tuberculous, or other ulcerations (which if small are *very apt to be overlooked*)—may be attended by a certain amount of recurrent bleeding. In these circumstances the hæmorrhage is usually an intermittent and subordinate feature. The diagnosis rests on the characters already given. When small in quantity the blood often passes backwards into the throat, and is swallowed, or it may be expectorated or coughed up, and be mistaken for hæmatemesis or hæmoptysis.

(*b*) With Constitutional Causes the bleeding is usually, although not always, of larger quantity, and it may, indeed, be so profuse as to endanger life. The blood in this group comes from a spot near the anterior part of the septum. Among the *predisposing causes* none is more frequent than an idiopathic tendency which exists in certain individuals to bleed upon slight provocation, a tendency which runs in families. Without amounting to hæmophilia, certain persons undoubtedly present some inherent quality which renders them more liable to bleed from their mucous surfaces, with or without a wound. It may exist in only one member of a family, but more often in several brothers and sisters. I have often noticed that such a predisposition may exhibit the phenomenon of atavism and skip a generation. Epistaxis is more frequent in children, especially in boys. It is also met with in the aged, but only when vascular disease and some of the other conditions about to be mentioned exist. The constitutional causes may be grouped under (*a*) *Alterations in the Cardio-vascular System*, and (*b*) *Altered Blood States*.

(*a*) Epistaxis occurring for the first time in an apparently healthy

[1] Dr. Manasseh relates the case of a child with epistaxis in whom a leech was found in the nose. This had gained entrance by the child drinking at springs in a district where leeches abounded in the water.—*Lancet*, September 16, 1899, p. 785.

person over forty years of age should always give rise to the suspicion of chronic Bright's disease. It affords us, moreover, an indication for the treatment of this malady, of which advantage may sometimes be taken, for it relieves the vascular tension which would otherwise seek relief in some less favourable situation. For instance, I have observed several patients who, after repeated admissions to hospital for epistaxis, have finally come in to die of cerebral hæmorrhage. Epistaxis is a frequent consequence of cardiac valvular disease, emphysema, chronic bronchitis, and cirrhosis of the liver. It may also be an evidence of lardaceous or other disease of the vessels. Finally, epistaxis is one of the forms of vicarious menstruation, and, like the bleeding which may take place in hysteria and other conditions where the vaso-motor system is disordered, we must regard this as an extreme effect of disease of the sympathetic nervous system.

(*b*) Concerning *Altered Blood States*, it may occur with purpura, hæmophilia, scurvy, leukæmia, anæmia (simple, and especially pernicious), and the specific fevers, especially typhoid, rheumatism, and the hæmorrhagic forms of the exanthemata. It is in children a not infrequent prodromal manifestation of whooping-cough and similar microbic disorders.

Prognosis.—Slight epistaxis in children is of no consequence, but occurring for the first time in persons at or past middle life should receive our serious attention, and its cause should be carefully investigated. Inquiry should always be made as to whether it has occurred previously in the life of the individual, because, as above mentioned, certain persons have this tendency, and in these the symptom may not be of much importance.

Treatment.—The indications are—first, to check the hæmorrhage if profuse ; and secondly, to ascertain the cause.

The epistaxis which accompanies Bright's disease, and the congestion of cardiac and pulmonary disease should not be checked unless the amount be profuse. In such cases the epistaxis is usually preceded by headache, and is accompanied by high arterial tension. It may be one of Nature's methods for the relief of congestion, as evidenced by the fact that the headache and the high arterial tension are relieved by the hæmorrhage. In all cases of epistaxis, the first thing to do is to examine the arterial tension. So long as this remains high or moderate no harm can accrue from the epistaxis.

(*a*) The treatment of *the attack* resolves itself into checking the hæmorrhage. The patient should be kept perfectly quiet, with the head erect, and chin forward, the head being cool, the feet warm, with hot bottles if necessary. The arms may be raised above the head and ice applied to the lower cervical spine. A homely substitute for the latter has long been in vogue in the form of the front-door key. Some recommend pressure to the anterior part of the septum by the

thumb and forefinger externally. More extreme measures consist of the application of liquor ferri perchloridi or adrenalin to the site of the hæmorrhage, if this can be discovered, or the use of styptic sprays of hamamelis, catechu, vinegar, lemon-juice, etc. Finally, if all these fail, the posterior nares must be plugged by means of Belloc's cannula, or a simple makeshift in the form of a loop of malleable wire.

(*b*) *Between the attacks* a very *thorough investigation* of the nasal and post-nasal cavities must be made. Minute lesions, quite sufficient for epistaxis, are very easily overlooked. The treatment of recurrent epistaxis is not always an easy matter, for the cause is often obscure, and we are often driven to regard the case as belonging to the idiopathic group above referred to. In a good many cases iron is efficacious in warding off the attacks; and calcium chloride by increasing coagulability.

THE THYROID GLAND.

This gland is anatomically connected with the upper respiratory passages, but is physiologically quite separate. There is still much for us to learn concerning its functions, both in health and disease. In health it is concerned in what is called "internal secretion," and it either adds to or modifies the blood in some way that is necessary to the well-being of the individual. This is proved experimentally,[1] and is evident from the gravity of the symptoms which arise when the gland is diseased or is removed by operation.

Symptomatology.—There are two opposite clinical conditions which may arise from disease of the thyroid gland. In one there is a *diminished* thyroid action, a condition of *Athyroidism*, the symptoms of which (lethargy, lowered vitality, and impaired growth and development) are identical with those of Myxœdema and Cretinism. These effects also arise, as Horsley has shown, when the gland is extirpated. The other condition is one of *increased* (or diseased) thyroid action or *Thyroidism*, the symptoms of which resemble Graves' disease; and these, with the exception of the proptosis, can be produced by the internal administration of thyroid gland or extract in large doses. It is important to remember that the size of the gland is not always a guide to which of these two sets of symptoms we may expect in a given case, for enlargement of the gland is consistent with atrophy of the glandular elements and diminution of function; on the other hand, a small gland may be functionally active. It depends on the nature of the histological changes, and these need further elucidation.[2]

[1] Sir Victor Horsley, *Clin. Journ.*, March 8, 1899, and Trans. Roy. Soc.

[2] True hypertrophy, with increased functioning of the thyroid gland, is indicated microscopically by great diminution of the colloid material and increase in the secreting cells which line the vesicles. See also Victor Horsley, *Clin. Journ.*, 1899, and Trans. Roy. Soc. in the previous years.

§ **135. Physical Examination and Classification.**—There are but two physical signs referable to the thyroid gland—viz., enlargement or diminution of volume. The size of the gland can never be gauged with precision, and when the alteration of volume is only slight it is difficult, if not impossible, to estimate it with accuracy, because it is partially covered by muscles, and so intimately connected with the trachea and other deeper structures. The patient should be instructed to let his head fall forwards and *to swallow* whilst we endeavour to gently grasp the gland. The thyroid rises during deglutition as no other neck tumour or organ does. Some idea may be obtained of the progress of a case by measuring the neck from time to time, always exactly at the same level.

Classification.—In general, *enlargement* is attended by a condition of thyroidism (*e.g.*, Graves' disease), and a *diminution* by a condition of athyroidism (*e.g.*, myxœdema) ; and there are two well-marked types of disease which are *usually* associated with enlargement, and two with diminution in volume of the thyroid gland.

(*a*) The two diseases (besides cancer and other neoplasms) in which **enlargement of the thyroid** is—at some stage of the malady—the essential or pathognomonic feature[1] are—

I. Graves' Disease—or Exophthalmic Goitre—is the term applied to that form of enlargement of the thyroid which, coming on in adult life, is attended by proptosis, and by numerous cardio-vascular and nervous symptoms, with marked disturbance of the general health. These general symptoms collectively constitute thyroidism. They are often present long before there is any visible enlargement.

II. Simple Goitre or "Bronchocele" is the term applied to a simple increase in size of the thyroid gland, either congenital or coming on in childhood or early adult life, generally unattended by increase or diminution of function, and unaccompanied, therefore, by any symptoms other than the mechanical effects of enlargement. In other words, there is in most cases a condition neither of thyroidism nor athyroidism.

II*a*. Bronchocele of Adolescence is the name under which I propose to include the slight, usually tender, but painless enlargements which are not uncommon in young women, transitory (a few weeks) in duration, probably inflammatory in origin, and usually unattended by any general symptoms.

(*b*) There are also two diseases in which **atrophy of the thyroid**—or at any rate a diminution of its function (and usually of its size)—is the essential feature of the malady :

I. Cretinism is the term applied to the condition of stunted growth

[1] In Acromegaly (Chapter XVII.) the thyroid is sometimes slightly enlarged or diminished in size, but it has generally been regarded as a subordinate feature in this rare and strange disease. In some cases of Cretinism (which is included in group *b*) the thyroid gland is considerably enlarged, but *deficient* thyroid action constitutes the chief factor of the disease.

(both in mind and body) of the individual, due to congenital atrophy or absence of the gland function, although the gland itself may be either enlarged or diminished in size. It is a condition of congenital ATHYROIDISM. The disease is endemic in certain districts.

II. MYXŒDEMA is the term applied to the group of symptoms (lethargy, low vitality, etc.) which, coming on in adult life, accompany *atrophy* of the thyroid gland. This is a condition of acquired ATHYROIDISM, or sporadic adult cretinism.

It is legitimate to infer from the foregoing that—

1. Increased or disordered thyroid secretion gives rise to profound disturbance of the general health, and neuro-vascular irritation (Graves' disease).

2. An innocent enlargement of the thyroid, unaccompanied by increased or disordered thyroid secretion, has no effect on the economy (as in most cases of bronchocele).

3. Simple absence or diminution of the thyroid secretion results (*a*) when it is congenital or comes on in early life, in deficient development, mental and physical, (*i.e.*, cretinism); and (*b*) when it supervenes in adult life, in lethargy and deficient vitality (myxœdema).

Diseases usually attended by thyroid ENLARGEMENT—*viz.*, GRAVES' DISEASE *and* BRONCHOCELE.

§ **136. Graves' Disease** (Synon.: Exophthalmic Goitre, Basedow's disease) has been defined on the preceding page. Usually the onset is very insidious. There are *five* groups of symptoms, and the varieties of the disease depend on which of these predominate.

Symptoms.—(1) *Cardio-vascular* disturbances are among the earliest and most important symptoms. They are never absent, and may exist for months before any other evidence appears: (i.) Palpitation. (ii.) The increased frequency and tumultuous action of the heart is accompanied by a rapid and sometimes feeble pulse, up to 150 or more on the slightest exertion or emotion. The rate may sometimes be reduced by absolute rest by 40 or 50. (iii.) Paroxysmal dyspnœa and a distressing sense of suffocation, produced and relieved by the same means as the preceding. (iv.) Evidences of cardiac disease, such as the murmur of dilatation (said to be present in two-thirds of the cases, § 44), are frequently present. The commonest murmur is a systolic, heard loudest over the second left costal cartilage, and sometimes propagated up the vessels of the neck. (v.) Sometimes slight dropsy and occasionally albuminuria are observed.

(2) *Nervous* disturbances are always present, and very closely resemble the nervous-vascular phenomena of neurasthenia. They are very variable: thus (i.) there may be nervousness, irritability, insomnia, depression alternating with excitement, hysterical attacks, melancholy, or mania. (ii.) Hyperæsthesia, perverted sensations, neuralgic headache, vertigo, tinnitus aurium, and hallucinations of sight or hearing. (iii.) Other fairly common symptoms are fine and rapid vibratile tremors of the hands, or of the lips (causing stammering); choreïform movements or paresis, usually transient. (iv.) Vaso-motor disturbances of

many different kinds, such as " flushings and shiverings," and various other subjective phenomena. All these symptoms, as well as the cardio-vascular symptoms above mentioned, are of a paroxysmal character, and vary from hour to hour.

(3) *Thyroid Enlargement* is always present at some stage of the disease, though it is rarely the first symptom noticed by the patient, probably because there are no means of detecting slight enlargements. Therefore in the early stages we have to rely upon the other symptoms. The enlargement varies considerably in different cases, and is by no means proportionate to the other symptoms, because the symptoms depend more upon the histological element of the gland which is involved than the degree of enlargement (p. 218). Mechanical effects of thyroid enlargement may be present (see Bronchocele), and occasionally alteration in the voice from this cause is the first symptom noticed by the patient.

(4) *Exophthalmos* (proptosis or protrusion of the eyeballs) is present in a varying degree, though sometimes not until late in the disease (Fig. 2, p. 20). It is best detected by seating the patient in a chair, standing behind him, and looking down his forehead. As a rule no changes can be detected in the fundi. Later on ulceration of the cornea occasionally takes place, either from neuro-trophic causes or from deficient protection.

Four signs of Graves' disease referable to the eyes bear the names of different physicians. *Von Graefe's* sign is a condition in which the upper eyelid does not follow the eyeball when this makes a downward movement. *Mœbius's* sign is an insufficiency of convergence of the two eyes when looking at a near point. *Stellwag's* sign is an absence or deficiency of blinking as an involuntary act. *Abadie's* sign in this disorder is an involuntary twitching or spasm of the levator palpebræ superioris. All except the first are present only in advanced cases, and are not therefore of very great value in the diagnosis.

(5) The *general health* of the patient is always disturbed. Anæmia is pronounced, and is usually in proportion to the severity of the other symptoms. Progressive weakness is always present. The *varieties* of the disease depend on which of these five groups of symptoms predominates.

Etiology.—(i.) Upwards of 95 per cent. of cases are females. (ii.) Nearly all are young adults between the ages of fifteen and thirty. (iii.) Locality has no known influence, but Russell Reynolds believed that a low temperature, continuous rainfall, and little sunshine may be determining factors. (iv.) Heredity has not been traced so far as the disease is concerned, but the family often show neuroses in the shape of epilepsy, chorea, hysteria, insanity, etc. (v.) Fright, anxiety, and mental overwork are potent factors in determining the disease.

Diagnosis.—The four cardinal symptoms are : (i.) thyroid enlargement, (ii.) proptosis, (iii.) rapid cardiac action, and (iv.) nervousness. The early stage of the disease is often mistaken for Neurasthenia or for Tachycardia, but (i.) and (ii.) are wanting.

Prognosis.—The duration of the disease varies from some six months to many years. It may certainly shorten life, but many very severe cases have recovered under modern methods of treatment. The mortality has been variously stated as from 10 to 50 per cent.; probably more modern statistics would give from only 5 to 10 per cent. Death may occur from cardiac failure, from some intercurrent disease, or with a condition termed Acute Thyroidism, in which there is a great aggravation of the symptoms above described, accompanied perhaps by maniacal excitement.

Treatment.—The early recognition of the disease is very important, for a great deal can be done in the early stages. Many drugs have been tried. *Iodides* in increasing doses (10 or more grains *ter die*) undoubtedly reduce the exophthalmos and thyroid. *Bromides*, especially the ammonium salt, are useful if nervous symptoms predominate. Digitalis, strophanthus, and belladonna are very valuable for steadying the heart; by beginning with small doses and persevering, recovery has been effected in some cases. Iron, arsenic, and other nerve tonics are certainly useful. The orthophosphates of sodium and potassium, as recommended by Kocher, or phosphoric acid are of value. Rest of body and freedom of the mind from anxiety are of great importance, combined with other treatment, which must depend on which group of symptoms predominates. Mere sojourn in a hospital will of itself do good, and sea-voyages are of use in the same way. One of my cases improved under opium, 4 minims *ter die;* another under suprarenal extract. Thymus gland extract (10 to 30 grains *ter die*), or ℥ss. to i. raw gland three or four times a week, has been tried very extensively, sometimes with success.[1] Extirpation of the thyroid[2] has been successfully adopted as a remedial measure, and is indicated when that structure is greatly enlarged. Excision of part of the diseased gland when very large, and division of the isthmus or excision of the sympathetic cervical ganglia, in cases where the gland is not much enlarged, have been recommended.[3]

Galvanism (descending) is often useful, and small doses of X rays have given good results in some cases. Parathyroid gland has been administered, but without convincing results. The discovery that the thyroid is a protective gland, which neutralises circulating toxins, has led to the trial of various sera. Thus Mœbius has injected a serum prepared from the blood of thyroidectomised sheep, and Murray a serum from rabbits, fed on increasing doses of thyroid extract. *Rodagen*, the desiccated milk of dethyroidised goats, has given good results, but all these recent methods require much longer trial to estimate their value.

§ 137. Bronchocele (Simple Goitre) is another form of enlargement of the thyroid gland. It may affect the whole organ, or only one of its lobes, or the isthmus. *Anatomically*, the enlargement may be due chiefly to parenchymatous increase, to cystic enlargement, or to a vascular increase. The enlargement may be so great

[1] David Owen, Brit. Med. Assoc. Meeting, *Lancet*, August 22, 1896. Also *Brit. Med. Journ.*, January 7, 1899, article by W. Rushton Parker, M.A., M.D., Cantab.
[2] *Lancet*, 1894, vol. ii., p. 1045; and 1895, vol. i., p. 1077.
[3] *Brit. Med. Journ.*, 1899, vol. ii., p. 899, Polland and Lake.

that the organ amounts almost to the size of an infant's head. It is worthy of notice that the healthy gland may slightly enlarge during pregnancy.

The *Symptoms* which attend the disease are entirely those due to the mechanical pressure of the tumour, and it is by the absence of the cardio-vascular, nervous, and other symptoms that this condition is distinguished from Graves' disease. The chief pressure symptoms are referable to the larynx and trachea. The voice is modified early in the disease, and vertigo, due to pressure on the vessels, may be present. The general health may be good, but the patient is usually somewhat anæmic, otherwise there are usually no symptoms of thyroidism nor of athyroidism. It may be *Diagnosed* from other tumours in the neck by the fact that it invariably rises with the larynx during deglutition. The enlargement generally increases steadily, but it is only rarely that there is any danger from tracheal obstruction and asphyxia.

The *Etiology* of the condition is not well known. More often females are affected, and it sometimes starts during pregnancy, or a catamenial period, but it is most prone to start during adolescence, between eight years of age and puberty. It never starts after forty. The disease is endemic in certain districts, and these for the most part are valleys which have a calcium or a magnesium and limestone subsoil, together with a large amount of sulphate and carbonate of lime in the drinking water. That it is not entirely due to the last-named condition is shown by the fact that in districts where this permanent hardness of water exists bronchocele does not occur. Captain McCarrison[1] has shown that the endemic goitre of Chitral and Gilgit is due to amœboid organisms. Dogs, horses, and mules suffer also from the condition.

Treatment.—The patient should, of course, leave the district; or, if this is impossible, the water used for drinking purposes should be boiled. When these conditions can be fulfilled, recovery may result. As regards remedies, potassium iodide in gradually increasing doses has the greatest reputation, and in a less degree ergot, arsenic, and the local application of blisters. Inunctions of mercury or iodine paint have been employed with considerable success. Radical cure consists in the removal of the tumour. The operation of tapping and injecting iodine and other remedies has almost fallen into disuse.

Diseases in which the thyroid is usually DIMINISHED *in size—viz.*, I. CRETINISM, II. MYXŒDEMA. *The latter is described elsewhere, since the leading symptom is General Debility* (*Chapter XVI.*).

§ 138. **Cretinism** is a condition of dwarfism and deformity attended by mental imbecility, due to an absence or perversion of the thyroid secretion, and is endemic in certain districts. In advanced and typical cases the face is characteristically broad and flat, the tongue protrudes from the mouth, the eyes are wide apart, and the head is brachycephalic (*i.e.*, broad transversely). The skin and hair are dry and coarse, and the mental condition is extremely backward. In severe cases the body may be so dwarfed that a person of twenty is the size of a child of five. The limbs are shortened, the neck stunted; pads of fat are present above the clavicles; the hands are short and square (spade-like), the abdomen prominent, and an umbilical hernia is often present. The thyroid may be enlarged, small, or absent (see Figs. 6 and 7, § 19). Rushton Parker[2] distinguishes three *varieties*, both etiologically and pathologically. In one, the thyroid is embryologically not developed, or only partially developed, the cause being presumably akin to that which brings about other embryological deficiencies, such as acardia, acephalia, etc. In a second, the thyroid undergoes the same changes as in endemic goitre, and doubtless from the same cause, any differences being due to loss of thyroid function. In a third, the thyroid, after performing its functions healthily for a time, atrophies, doubtless from causes akin to those of adult myxœdema.

[1] Paper read before the Royal Society, December, 1908.

[2] "Acquired Cretinism, or Juvenile Myxœdema," *Brit. Med. Journ.*, May 29, 1897.

Etiology.—Cretinism is endemic in certain districts—*e.g.*, the valleys of Switzerland. Cases occur also in certain parts of England, especially in the valleys of the Lake District, and Derbyshire. Sporadic cases are found in healthy families. The causes are unknown. Some attribute it to consanguinity of the parents, to alcoholism, tubercle, syphilis, or to maternal worry during pregnancy. It is associated with deficient thyroidal function, and hence may be regarded as congenital myxœdema. In slight cases of cretinism the diagnosis from other forms of mental deficiency may be difficult. The condition of the skin and hair are valuable diagnostic features.

Prognosis.—The patient may grow up capable of doing light manual work, or may remain an idiot. Under treatment begun early, the child may recover completely, but in other cases, although the body is greatly improved, the mind does not improve in proportion.

Treatment.—Thyroid extract, beginning with ½ grain doses (5 grains of raw gland), causes a rapid and remarkable change. The skin becomes soft, the general conformation normal, and, if the treatment has not been too long delayed, the mind assumes its natural vigour. The patient must *continue* to take the thyroid all his life, or else he will relapse. A case showing the remarkable efficacy of this treatment is figured in § 19.

CHAPTER VIII

THE MOUTH, TONGUE, AND GULLET.

The Mouth.

(Lips, Breath, Saliva, Teeth, and Gums.)

We often regret that we cannot investigate the internal organs more thoroughly, but how seldom do we avail ourselves of the instructive information afforded by a thorough examination of the mouth? Many of the indications of syphilis, hereditary or acquired, may be so revealed; several other constitutional conditions produce symptoms in this locality, such as anæmia and lead-poisoning; and a good idea of the general condition of the patient can be obtained from a careful inspection of the tongue. Many of the disorders special to the mouth are comprised among the "causes" of stomatitis. For the diagnosis of these disorders it is necessary to make a thorough examination of the Lips, the Breath, the Saliva, the Teeth, and the Gums. We will consider the symptoms referable to these structures in that order.

§ **139. The Lips.**—*Dryness* of the lips is often one of the most conspicuous evidences of indigestion, and it is a very useful one, because this disorder has so few physical signs to assist us. The lips are *pale* in anæmia, they are *cyanosed* in advanced bronchitis with dilated right heart, and in other conditions where there is obstruction in the circulation. This cyanosis is especially marked in congenital heart disease.

Fissures around the lips are an almost infallible sign of syphilis, especially when surrounded by a reddened infiltration. This infiltration helps us to distinguish a syphilitic fissure from the "cracked lip," which is the only condition liable to be mistaken for syphilis. *Cracked lip* occurs mostly in nervous children who lick and bite their lips and are exposed to cold winds. It can generally be remedied by the application of some simple ointment, such as zinc ointment or cold cream, whereas the syphilitic fissures do not yield to this treatment. By pressing the corner of the mouth inwards and forwards when the patient opens it, we may often detect a mucous patch surrounding a syphilitic fissure inside the mouth. The *scars* left by syphilitic fissures are also a useful indication of a previous attack, or still more frequently

that the patient has had congenital manifestations. They are white and stellate.

§ **140. The Breath** should be normally quite free from any kind of odour. Offensiveness of the breath may arise from four sources: (1) A want of cleanliness *in the mouth*, particles of decomposing food, and the presence of decayed teeth, may give rise to a very offensive odour of the breath. (2) *Dyspepsia, constipation*, and other conditions of the *alimentary canal*, and the derangement of digestion in fevers, may also produce a bad smell of the breath. (3) Some *diseases of the nose;* thus it always accompanies ozœna. (4) A large cavity in the *lungs*, especially if *bronchiectatic*, produces a putrid odour. The odour of bronchiectasis is very foul, and is characterised by being intermittent; it comes on suddenly, lasts a day or two, and disappears gradually. Certain general conditions are attended by a more or less characteristic odour of the breath. Thus, in *diabetes* it is sweet; in acute *alcoholism* it is alcoholic or ethereal. In *uræmia* it is said to be urinous. Certain *drugs* give rise to a very characteristic odour in the breath—*e.g.*, turpentine (a resinous odour), chloral (odour of chloroform), bismuth (odour of garlic), and opium (odour of the drug). Alcohol, ether, chloroform, and many other volatile substances are partly excreted by the breath.

§ **141. The Saliva** may be *increased* (i.) in mouth inflammations; (ii.) in chronic gastritis, in which there is such a profuse flow of saliva during the night that it gives rise to the impression that the patient, in the morning, is vomiting clear alkaline fluid (water-brash or pyrosis); (iii.) in pregnancy, and in mania, hydrophobia, and some other diseases; (iv.) after the administration of mercury, pilocarpine, bitters, and, according to some, alkalies and acids. The saliva is *decreased* (i.) in certain febrile states, (ii.) in diabetes, (iii.) severe diarrhœa, (iv.) chronic Bright's disease, and (v.) during the administration of atropine or daturin. A condition known as "dry mouth" has been described by some authors,[1] in which there is a constant deficiency of saliva. The cause of this is obscure.

§ **142. The Palate** may be "cleft" from childhood, otherwise a hole in this situation is practically always an evidence of past syphilis. The *soft* palate shares in the diseases of the fauces (§ 108). It is a favourite position for the membrane of diphtheria, which in this situation forms an important means of differentiating the disease from follicular tonsillitis, the exudation of which never affects the palate. The *hard* palate is sometimes involved in the diseases of the floor of the nose. A swelling may appear here in abscess of the antrum, or in abscess dependent on disease of the lateral incisor tooth. The latter is the commonest cause of swelling in this situation according to Tomes.[2]

[1] Clin. Soc. Trans., 1885.

[2] "System of Dental Surgery," Charles S. Tomes, p. 680. London, 1897.

Thirst accompanies all febrile conditions and inflammatory conditions of the gastric mucous membrane. It is met with also in diabetes, after diarrhœa and vomiting, and after a diet excessively salted.

§ **143. The Teeth** are subject to a certain amount of variation, even in health. The *average* dates of the eruption of the temporary and permanent teeth are as follows :

Temporary or "Milk" Teeth.	*Permanent Teeth.*
About 6th to 8th month, lower central incisors.	About 6th year, first molars.
About 8th to 10th month, upper incisors.	,, 7th ,, central incisors.
About 12th to 14th month, first molars.	,, 8th ,, lateral incisors.
About 18th to 20th month, canines.	,, 9th ,, anterior bicuspid.
About 2 to 2½ years, posterior molars.	,, 10th ,, posterior bicuspid.
	,, 11th to 12th year, canines.
	,, 12th to 13th ,, second molars.
	,, 17th to 25th ,, third molars.

One quarter of the mouth may be represented diagrammatically thus :

Teeth ..	I.	I.	C.	M.	M.
Month of eruption.	6	9	18	12	24

Teeth ..	I.	I.	C.	B.	B.	M.	M.	M.
Year of eruption.	7	8	11	9	10	6	12	24

The normal order of eruption of teeth may be represented thus : MILK teeth 6, 9, 18, 12, 24 MONTHS ; and PERMANENT teeth 7, 8, (11), 9, 10 ; 6, 12, 24 YEARS. These details are worth remembering, because defective or deficient teeth are nowadays an extremely frequent cause of faulty digestion.

The presence of decayed teeth is one of the commonest causes, if not the commonest, of the dyspepsia of modern times, and it is an ominous feature that a very large proportion of the candidates for the Army and Navy Services are rejected because of bad teeth. They decay early in rickets, in cretinism, and in some other constitutional conditions.

The teeth are altered in shape after stomatitis in early life, which may be due to mercury, etc. (*vide* § 146). In these circumstances the teeth present transverse and vertical ridges, with or without alteration of shape. "Hutchinson's teeth" show alterations in the shape of the permanent teeth, due to hereditary syphilis, and present a valuable means for the identification of this disorder, as they are of very frequent occurrence in that disease, and bear lifelong testimony. They are both pegged and notched—that is to say, the transverse measurement is smaller at the free edge than the part near the gum, and on the edge of each tooth there are one or two notches (see Fig. 3, p. 21).

§ **143*a*. Toothache** (odontalgia) is caused most frequently by decay (caries) of the teeth ; but there are OTHER CAUSES, the chief of which, as given by Mr. C. S. Tomes (*loc. cit.*), are as follows :

1. Morbid conditions of the *tooth-pulp*, including irritation, acute and chronic inflammation of the pulp, pressure from confined matter in the pulp cavity, and deposit of secondary dentine in its substance.

2. Exposure of sensitive dentine, with or without caries, is probably the commonest cause of toothache—a "hollow tooth," as it is called.

3. Morbid conditions of the *alveolar periosteum* and exostosis, including inflammation of the periosteum, acute and chronic alveolar abscess, lesions manifested by alterations of the roots of the tooth (such as roughening by absorption or increase by exostosis).

4. Morbid conditions of the *periosteum of the jaws—e.g.*, traumatic, rheumatic, strumous, or syphilitic periostitis.

5. Irritation of the *dental nerves* by causes not productive of visible local lesions. This may include malposition of wisdom teeth, retarded eruption of wisdom teeth, and pressure due to insufficient space for the teeth.

6. *Inflammations and ulcerations of the mucous membrane* and submucous tissue—*e.g.*, that consequent upon difficult eruption of wisdom teeth, and other conditions (see Causes of Stomatitis, *below*).

This is merely an approximate classification. The character and degree of the pain is greatly modified by the condition of the patient. The pain is generally more or less of an intermittent type. It is often absent at periods of full vigour—*e.g.*, after breakfast or dinner. Pain due to irritation, or to chronic local inflammation of the pulp, partakes of a neuralgic character, and the patient is often unable to point out the affected tooth or teeth. The suffering induced by acute inflammation of the pulp is excessive, particularly if there be no exit. It ceases more or less abruptly from the consequent death of the pulp. The recumbent posture or active exercises serve to aggravate the pain by increasing the vascular supply.

The *Treatment* belongs to the dental surgeon, but a good deal of temporary relief may sometimes be obtained by constantly rinsing the mouth with hot carbolic lotion (1 in 100). A formula for drops to apply to a hollow tooth is given in F. 23 at the end of this book.

§ **144. The Gums.**—The pallor of anæmia, the purple line of lead-poisoning, the red and ulcerated condition in stomatitis, the sponginess in mercurialism and scurvy are all useful local indications of some general condition. A swelling of the gums with greenish discharge suggests Actinomycosis. *Bleeding from the gums* is apt to occur in scurvy, purpura, the hæmorrhagic diathesis, and even in apparent health, when the teeth are covered with tartar and the gums recede. In some people the gums very readily bleed, and sucking them may produce bleeding, which enables malingerers and hysterical persons to simulate diseases of the lung or stomach. It is detected by being only in small or moderate quantity, and by its intimate mixture with saliva. There is one disease of the gums—Pyorrhœa Alveolaris—the importance of which has only recently been recognised.

§ **145. Pyorrhœa Alveolaris** (Riggs' disease or Suppurative Gingivitis) is a raw ulcerating condition of the gums around the sockets of the teeth or stumps. When tartar is allowed to collect upon the teeth, it gradually pushes the gum back; and by degrees a pocket or fossa is formed around the neck of each tooth, and there is considerable sero-purulent and often blood-stained discharge from the pockets thus formed, which not only imparts an offensive odour to the breath, but, being continually swallowed, is absorbed, and sets up a chronic toxæmic condition, which it is now recognised may produce a large number of

troublesome symptoms.[1] Dyspepsia, even apart from difficulties of mastication, invariably ensues sooner or later. But even before the dyspepsia becomes established the patient is listless, languid, and unfit for work, and complains of a great variety of functional nerve symptoms. A large proportion of my out-patients at the Nerve Hospital who complain of functional neuroses owe their troubles to pyorrhœa alveolaris. Among the symptoms due to this cause I may mention headache, neuralgia, pains or tingling in the limbs and prostration, attacks of flushing, shivering, or giddiness; a feeling of heaviness, and swelling of the limbs which is sometimes attended by actual œdema of the ankles, wrists, and other parts, which differs from ordinary anasarca in requiring longer pressure to produce the pit. Great depression is usual, and even melancholia may result. One of my patients committed suicide.

Treatment.—All these symptoms may arise when, for instance, stumps are left beneath an artificial plate. If the pyorrhœa, as frequently happens, be not identified as the cause and removed, no treatment is of much use. The tartar must be removed and the suppurating pockets carefully dressed with sulphate of copper, iodine, or peroxide of hydrogen. This must be done at intervals of a week or so, and later on of a few months, by a skilled person. The only radical cure is the removal of the stumps or teeth if these are at fault. Recently good results have been obtained in obstinate cases by the inoculation of vaccines prepared from organisms cultured from the gums.

§ **146. Stomatitis** is a generalised inflammation of the mouth, evidenced by redness, swelling, tenderness, and pain of the mucous membrane, swelling and protrusion of the lips in severe cases, offensive odour of the breath, and usually, but not always, excess of saliva. This, the simplest form of stomatitis, such as occurs in dentition or the application of caustics, is known as (*a*) *Catarrhal* or *Erythematous Stomatitis*. (*b*) *Aphthous Stomatitis*, also known as *Vesicular* or *Herpetic Stomatitis*, occurs in badly-fed children, and it presents, in addition to the above features, small grey patches, with a red base and sharply defined circular margin, resembling vesicles, which are very painful to the touch. (*c*) *Ulcerative Stomatitis* occurs in a mild, and also in a severe form. In this we find, in addition to the features belonging to variety (*a*), irregular ulcers, especially on the gums, which recede from the teeth, so that the teeth become loosened. In the severe form there is great fœtor of the breath, considerable enlargement of the glands, submaxillary and cervical, and constitutional disturbance; and the teeth may drop out of the ulcerating gums. The ulcers often have a yellowish or grey coating, resembling a membrane, and it is therefore sometimes called "pseudo-membranous stomatitis," or "phagedenic gingivitis." (*d*) *Gangrenous Stomatitis* (Cancrum Oris, Phagedena Oris, Noma Oris) is a gangrenous inflammation starting at one spot, usually on the cheek or on the lips. At first there is acute pain, but as this passes off a black spot forms (usually both internally and externally), which spreads, and leads to perforation of the cheek. The inflammation may spread to the gums, and the teeth become loosened. This is a severe disease, attended by considerable prostration, and, at first, a subnormal temperature. It is apt to

[1] This subject was the topic of an interesting discussion at Roy. Med. Chir. Soc., June, 1900. The blood-stained discharge is liable to be mistaken for serious disease of the stomach or lungs, as in cases mentioned at that discussion.

follow measles or other exhausting illnesses in weakly children exposed to bad hygienic conditions.[1] (*e*) *Pyorrhœa Alveolaris* has been separately described above (§ 145).

Etiology of Stomatitis.—(1) Certain *local conditions*, of which the commonest are dentition, tartar, and a want of cleanliness, the local irritation of a jagged tooth, excessive smoking, dirty feeding-teats in children, the application of hot fluids and caustics, new growths (simple or malignant), and gummata. In most of these cases the stomatitis takes the form of (*a*) or (*b*) above. Mouth-breathing and chronic gastric catarrh are said also to give rise to stomatitis occasionally, and necrosis of the jaw may lead to an ulcerative stomatitis.

(2) Certain *drugs and chemical substances* are apt to cause stomatitis. Chief amongst these is mercury, which gives rise to a very characteristic ulcerative stomatitis, with spongy gums and great fœtor of the breath. Arsenic and iodides may produce catarrhal stomatitis. Phosphorus produces ulcerative stomatitis, with necrosis of the jaw. The blue line of lead may be attended by a certain amount of catarrhal stomatitis.

(3) Chief among the *constitutional conditions* which cause stomatitis is (i.) the lowered vitality met with in phthisis and other wasting disorders, or in badly-fed children, in whom the stomatitis may be aphthous or ulcerative. Thrush often accompanies catarrhal stomatitis in these circumstances. (ii.) Syphilis is accompanied by a special variety of the catarrhal form, and is attended by whitish, semi-transparent patches on the tongue and mucous membrane, resembling "snail-tracks." Later on ulcerations may occur (§ 113, Throat). (iii.) Measles and other acute specific fevers are apt to be followed by cancrum oris in children exposed to bad hygienic conditions. Diphtheria is attended by both stomatitis and rhinitis when the membrane affects the mouth and nose. (iv.) Scurvy and purpura are attended by swollen and spongy gums and ulcerative stomatitis. (v.) Gastro-intestinal derangement, as in dyspepsia and fevers, leads not infrequently to catarrhal and sometimes aphthous stomatitis. (vi.) A lowered state of health, with insanitary environment, gives rise to epidemics of ulcerative stomatitis, sometimes taking a fatal form, in jails, hospitals, and camps. Occasionally this condition is met with in individuals in private life.

(4) Certain *skin lesions* may invade the mucous membrane of the mouth, such as the rashes of small-pox, chicken-pox, measles, and herpes iris. In measles certain spots, first described by Koplik,[2] appear on the inner sides of the cheeks, opposite the bicuspid or molar teeth, before the skin eruption occurs. They most often take the form of a greyish-white stippling on a slightly raised purplish base, and afford considerable aid in the early diagnosis of the disease. Lichen ruber planus may affect the mucous membrane of the mouth and tongue, and it may be present there even before it appears on the integument. In this situation it has a whitish appearance, much resembling secondary syphilis, for which it has sometimes been mistaken. Lupus may affect the palate.

Prognosis of Stomatitis.—As a rule, stomatitis is not a serious disease, except that form known as phagedenic stomatitis, in which the mortality is 80 per cent. Catarrhal, aphthous, and ulcerative stomatitis generally end in recovery in a week or two. Those cases due to constitutional conditions are, as a rule, far more serious

[1] Microbes probably play a more important part than is at present recognised in the pathology of stomatitis, and the entry of these organisms into the mouth, where they may perhaps lodge on some chance abrasion, might possibly account for some of those hitherto inexplicable cases of the disease. For instance, it seems highly probable that that extremely serious condition, *Cancrum oris*, may be connected with one of the group of bacteria which has lately attracted attention, and which includes the *Bacillus aërogenes capsulatus*, *B. œdematis maligni*, *B. emphysematosis*, and other microbes found in connection with "acute spreading traumatic gangrene," malignant œdema, *gangrene foudroyante*, etc. See, *e.g.*, a paper by Corner and Singer on "Acute Emphysematous Gangrene," the *Lancet*, November 17, 1900; and Discussion at the Path. Soc. Lond., in the *Lancet*, 1900, vol. ii., p. 1651.

[2] Koplik, *Med. Record*, New York, April, 1898.

and obstinate than those due to local or removable conditions. The stomatitis of mercury may be extremely severe, but is, happily, only rarely seen nowadays. When aphthous stomatitis occurs in adults, accompanying a lingering disease, it is very obstinate, and is, in itself, a very grave omen. The prognosis is grave in the epidemic form, which is probably of microbic origin. The complications of the phagedenic form are diarrhœa, broncho-pneumonia, and gangrene in other parts of the body, especially the organs of generation (noma pudendi).

Treatment.—In all varieties, the indications are, first, to remove the cause; secondly, to alleviate the local inflammation; and, thirdly, to attend to the general health. Any tartar present should be removed, and a wash for the mouth used, consisting of carbolic (1 in 100), or mercuric chloride (1 grain to 6 ounces), sodium bicarbonate (1 in 20), or chlorate of potash, or boracic acid (1 in 30). Chlorate of potash may be also administered with iron internally. In all pronounced cases milk and slop diet is indicated, on account of the difficulty and pain of mastication.

Aphthous and Ulcerative Stomatitis are best treated by touching the sore places with solid nitrate of silver or sulphate of copper. For the pain, a solution of cocaine (½ to 3 per cent.) may be used. In the ulcerative form chlorate of potash is especially useful. Antiseptics should be applied—*e.g.*, swabbing or painting the throat with glycerine and mercuric chloride (1 in 3), and a mouth-wash of 1 per cent. potassium permanganate. In the *Gangrenous* form (cancrum oris) prompt measures are necessary to avert a fatal issue. The modern treatment of this is averse to the application of strong caustics, such as were formerly used. The superficial sloughs should be gently removed by swabs, forceps, and scissors, and the parts beneath swabbed with perchloride (1 in 1,000) or carbolic (1 in 100); and this procedure should be *repeated every hour*, night and day, when the patient is awake. Abundance of stimulants and nourishment is called for.

The Tongue.

The alterations to which the tongue is liable will be referred to under six headings: (*a*) Furring of its Surface; (*b*) Ulceration; (*c*) White Patches; (*d*) Acute Swelling; (*e*) Chronic Swelling (Hypertrophy), and Atrophy; (*f*) Warts, Fissures, and Cicatrices. A mother sometimes speaks of her child being "*tongue-tied*" when the frenum is too short. In some cases this is really so, or the structure may be attached to the tongue too far forward, but it exists much less frequently than parents suppose.

§ **147. Furring of the Tongue.**—The appearances of the dorsum of the tongue used to be looked upon by older authors as an indication of the state of the stomach, and with certain reservations it is still regarded as some aid in the investigation of that organ (§ 195), though it is a better guide in the prognosis of fevers and other grave constitutional disorders. Five varieties of tongue have been described by authors: (1) The *pale, large, flabby tongue*, with broad tip and indented edges, and a uniform thin white coating, is the commonest abnormality. It is met with after alcoholic excesses, in atonic dyspepsia, in anæmia, and in gouty persons. (2) A *red tongue*, with sharp red tip and edges, in which the hyperæmic papillæ contrast strongly with the slight white coating in the centre, is found in subacute gastritis and irritable dyspepsia. (3) The *coated tongue*, with a uniform white layer over the surface, is found in acute gastritis, feverish conditions, anæmia, and

nervous depression. Two forms of this tongue have been described: (i.) The strawberry tongue, having a slight white coating through which the fungiform papillæ protrude at the tip and edges, is very typical of scarlatina and other highly febrile states. (ii.) The plastered tongue, where the coating is considerably thicker. The amount of coating on a tongue varies directly with (*a*) the amount of dryness of the mouth—that is to say, the deficiency of salivary secretion (*e.g.*, in fevers and profuse perspiration); and (*b*) with the immobility of the tongue, owing to eating food that does not require mastication. The plastered tongue may pass on to—(4) the *furred tongue.* The coated papillæ stand out separately, giving a shaggy appearance. It is met with in states of marked prostration—*e.g.*, coma, abdominal cancer, advanced phthisis, profound anæmia, and other asthenic states. The prognosis is grave when the tongue becomes encrusted and its dryness increases. From any cause it may become dry, brown, and crusted, and then pass on to—(5) the *denuded red tongue*, which generally follows the preceding as the crust falls off. This tongue is red, shiny, smooth, and often cracked. It is found in advanced states of the preceding conditions, in diabetes, and other severe chronic ailments. The appearance of this tongue in a disease is of very grave prognosis. Aphthous stomatitis may supervene.

There is no doubt that large individual peculiarities in the character of the tongue exist apart from disease. On this account some[1] go so far as to say that the tongue is of little importance as a clinical indication. Undoubtedly we should make sure in any given case that the tongue condition before us is not due to these personal peculiarities, to smoking in excess, or to previous disease.

As regards *Treatment*, it is an old saying that a red tongue requires alkalies, and a white tongue acids. The former of these is true to some extent, but not the latter. With the exception of diabetes, a dry tongue indicates no appetite, and deficient gastric secretion; therefore the patient should be fed on fluids, animal soups, and other things requiring no great digestive power; (4) and (5) call for alcohol and other stimulants. In the prognosis of enteric fever the tongue is a valuable indication.

§ 148. Ulcers of the Tongue may be Simple, Syphilitic, Malignant, or Tuberculous.

I. Simple Ulcers of the tongue are known by their superficial character, by the presence of some local cause, such as a jagged tooth or other local irritation (see also Ulcerative Stomatitis). The *frenum* is apt to be ulcerated in whooping-cough. This is probably of mechanical origin, but it is a useful aid in diagnosis.

II. Syphilitic Ulcers are of two kinds: (*a*) superficial, (*b*) deep.

(*a*) *Superficial Syphilitic Ulcers* of the tongue are met with usually

[1] *E.g.*, Sir Jonathan Hutchinson, *Med. Press and Cir.*, July, 1883.

at the side, or in the form of fissures on the dorsum (see § 113 for description), or superficial circular " punched-out " ulcers.

(*b*) *Deep Syphilitic Ulcers* are preceded by the formation of a roundish nodule (a gumma) which ulcerates. They are recognised by (i.) their site, which is usually on the centre of the dorsum ; (ii.) their raised, ragged, and sometimes undermined edges ; (iii.) the yellow slough which covers the base ; and (iv.) the fact that they leave deep stellate scars. Syphilitic ulcers are usually multiple ; the chief difficulty arises in the case of a single ulcer as to whether it be syphilitic or cancerous. Syphilitic ulceration is differentiated by (1) the relative absence of surrounding induration, and consequently there is less interference with the movements of the tongue ; (2) the site of the ulcer on the dorsum ; (3) there is less glandular enlargement, and the glands have a shotty feel ; (4) the age of the patient, malignant ulcers rarely occurring before forty ; and (5) there is a history of syphilis, and the disease *heals under iodide of potassium*.

III. Malignant Ulcer of the tongue is known by (i.) its site, which is chiefly on the side of the tongue ; (ii.) its hard, raised, everted edges, and its uneven warty base, with foul discharge and tendency to hæmorrhage ; (iii.) the induration around, and the early involvement of the glands ; and (iv.) the early impairment of the movements of the tongue with great pain. These are the characters in an advanced case when diagnosis from syphilis is relatively easy. In an early stage it may be very difficult. In that stage a cancerous ulcer has flat sloping edges and scanty secretion, *its progress is very slow*, and it does not yield to iodides.

IV. Tuberculous Ulcers are not common. They are superficial, with a yellowish discharge, and there is generally a history of tubercle in the lung or throat. The tubercle bacillus may be found in the scrapings.

Prognosis.—Simple ulcers are easily dealt with, but other ulcers of the tongue are dangerous chiefly from their liability to hæmorrhage, and because of the important structures around. The diagnosis of syphilitic from malignant lesions is as important as it is difficult, for however advanced the former may be, they yield to appropriate remedies, but the latter are necessarily fatal, being certain to recur sooner or later after removal.

The Treatment consists of the usual surgical measures. In syphilitic ulcers iodide should be given in large doses. It is rarely given in sufficient quantity.

§ **149. White Patches** are not infrequently met with on the tongue, and may result from : I. Thrush ; II. Leucoplakia ; III. Aphthous Stomatitis (§ 146) ; IV. Syphilitic Patches (§ 113). The two last are described elsewhere. The stellate cicatrices so characteristic of syphilitic lesions must not be confused with any of these.

I. In Thrush (parasitic stomatitis) there are white membranous patches, like

milk curd, sometimes with an areola round them. They are distinguished from other similar affections by (i.) leaving a bright bleeding surface when they are scraped off, and (ii.) by the detection of the fungus *Oidium albicans* (*Saccharomyces albicans*, Fig. 60) on microscopical examination. It usually starts on the tongue, but may invade the lips and the whole of the interior of the mouth. The disease occurs chiefly in infancy, also in the later stages of exhausting diseases in adults. In the adult it only occurs at the end of wasting disorders, and not infrequently forms one of the indications of approaching death. In infancy it generally arises in hand-fed children under bad hygienic conditions, and is often attended by diarrhœa. It is contagious from child to child. In children it has no very great significance, and readily yields to glycerine and borax, or weak carbolic lotion (1 in 500). The diet and methods of feeding should always receive attention in such cases. The patches may be removed by cotton-wool swabs soaked in weak carbolic. In such children it sometimes happens that excoriations are noticed around the anus, and the mother thinks the " thrush has gone through the child "; but these are more frequently due to congenital syphilis or eczema intertrigo.

II. Leukoplakia Linguæ (synonym, Ichthyosis Linguæ) is a term applied to flat, whitish, horny-looking, silvery patches on the tongue, due to a heaping up and condensation of the epithelium. The disease generally involves a considerable area of the tongue. In a later stage the tongue becomes red and glazed. The patches themselves are often cracked, and form a pavement-like surface, which has the appearance of ichthyosis of the skin. They give rise to a great deal of discomfort and tenderness. It is most frequently met with in tertiary syphilis, and, according to most observers, it is always an evidence of that disorder. But others attribute the condition to excessive smoking, jagged teeth, drinking, and dyspepsia. I cannot say that I have met with a case which could not be attributed to syphilis. The *Treatment* is, as a rule, very unsatisfactory, unless the disease be met with in the early stages. A mouth-wash, consisting of bicarbonate of soda (20 grains to the ounce), or a saturated solution of chlorate of potash, sometimes relieves the symptoms. But the best treatment, in my experience, is the local application of chromic acid (5 to 10 grains to the ounce, gradually increased), painted on daily. It should be accompanied by anti-syphilitic remedies, though they do not have a very marked effect. Some say that strong or irritating applications should be avoided.

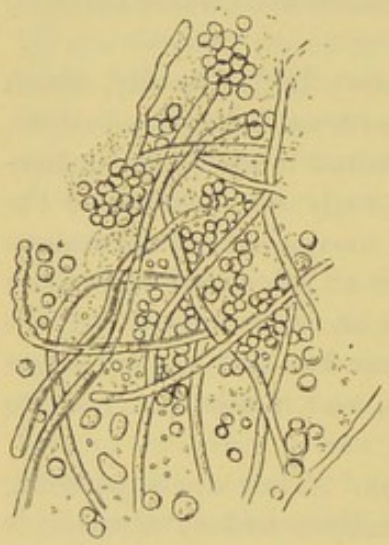

Fig. 60.—Oidium Albicans, or Thrush Fungus.

§ 150. Acute Swelling of the Tongue—*i.e.*, swelling of the tongue coming on rapidly—may be due to either (*a*) *Acute Glossitis* or (*b*) *Acute Œdema*. In both of these the tongue rapidly enlarges, and may even protrude beyond the teeth. A great deal of pain is present, and there is a difficulty of swallowing and speaking.

(*a*) Acute Glossitis may be due to various local causes—*e.g.*, the sting of an insect, a septic wound, biting of the tongue, acute ulcers—or it may be due to constitutional conditions—*e.g.*, mercurial salivation, and, according to some, acute specific diseases, such as erysipelas. The onset of acute glossitis is rapid, though rather less so than acute œdema; the swelling rarely extends beyond the tongue, and the glands are more markedly involved. *Treatment* consists of the use of mouth-washes, especially chlorate of potash, painting with a cocaine solution (10 per cent.), the administration of chlorate of potash, iron, and bark internally, with purging and antiphlogistic remedies generally. Free incisions may be necessary.

(*b*) Acute Œdema of the Tongue is a serious disorder, because of its liability to involve the glottis. It may accompany urticaria, or it may be, like the angina

Ludovici (§ 118), of an erysipeloid nature. The œdema comes on suddenly, and in the course of a few hours the tongue may protrude from the mouth. The swelling rapidly extends to the throat, nose, and down the œsophagus and trachea. It is attended by an inability to speak, to swallow, and sometimes even to breathe. Its *Causation* is obscure, but it is said to be usually of an urticarial nature, and to occur in those who have had urticarial attacks. This condition is *Diagnosed* from simple acute glossitis by (i.) its rapid advent in the course of an hour or two; (ii.) the rapid extension to the throat and other parts; (iii.) the presence sometimes of an urticarial rash, or a history of attacks of angioneurotic œdema. Without the last feature the diagnosis is difficult.

Prognosis and Treatment.—The disease comes on rapidly, and runs a very rapid course, subsiding in the course of twenty-four hours, unless the patient die in the meantime. It is apt to cause suffocation. Prompt measures are necessary. A strong purge should be given at once (croton oil, 1 minim, if it can be swallowed) or a turpentine enema. Cocaine (5 or 10 per cent.) should be kept constantly painted on the tongue. Scarifications and leeches may be required, and the practitioner should be prepared to perform tracheotomy if necessary.

§ 151. Chronic Swelling, Hypertrophy and Atrophy of the Tongue.

I. *Chronic Glossitis* is a chronic inflammation of the tongue, in which either the surface or the substance is mainly involved. The *surface* is covered with irregular, red, raw, tender patches and cracks (unless it be secondary to, or attended by, leucoplakia). If the *substance* be affected, the organ is enlarged, indented by the teeth, and in course of time it becomes indurated. It is more frequently due to some local irritation, such as a jagged tooth or an ulcer, in which case the enlargement is generally limited to one part of the tongue. Glossitis may arise from alcoholism, syphilis, chronic dyspepsia, or excessive smoking, and many of the other causes of stomatitis (*q.v.*). The treatment is directed to the removal of the cause, and the employment of chlorate of potash and astringent mouth-washes.

II. *Macroglossia* is due generally to a congenital overgrowth of the connective tissue, accompanied by a dilatation of the lymphatics of the tongue. Its causes are obscure. Persistent application of mild caustics or the galvanic cautery to the tongue is the only remedy.

III. *Atrophy of the Tongue* (microglossia) may arise from nerve lesions. It may occur in bulbar paralysis, and is then usually bilateral. In unilateral cases the lesion is either situated in the nucleus or trunk of the twelfth nerve of one side (*vide* Chapter XIX., § 622).

§ 152. Warts, Fissures, and Cicatrices.

Warts are simple or syphilitic. *Simple* warts are distinguished by the fact that they are soft; they are raised, and often pedunculated, and there is but little secretion. The glands are not shotty to the touch. *Syphilitic* warts are hard, with infiltration; they are never pedunculated, secretion is present, and the glands of the neck and elsewhere are shotty.

Fissures are also divided into simple and syphilitic. The *simple* can generally be accounted for by some such cause as the irritation of a ragged tooth, and are never infiltrated. On pinching *syphilitic* fissures between the fingers, infiltration is found to be present.

Cicatrices.—Simple ulceration rarely leaves a scar, but if so, it is never hard. Hard, stellate scars are invariably indicative of syphilis.

The Gullet.

§ 153. Symptomatology.—Diseases of the œsophagus have practically one symptom which is common to all—namely, *dysphagia—i.e.*, a difficulty in swallowing. There are certain features about this symptom which it is important to investigate:

First, does the difficulty apply to both liquids and solids? This

gives us an idea of the *degree* of the obstruction. *Secondly*, does the food return ? and if so, after what interval ? This is sometimes a guide to the *seat* of the obstruction. Obstruction within the *œsophagus* has to be distinguished from obstruction at the pyloric end of the *stomach* (i.) by the easy way in which the food regurgitates as compared with the vomiting which accompanies pyloric stricture ; and (ii.) by the absence of acidity in the material returned. *Thirdly*, is there any pain ? What is its situation, and is it only present after the ingestion of food? Constant pain is a feature of malignant disease. *Fourthly*, what is the duration of the dysphagia ? Has it been persistent, and become progressively and steadily worse ? The last named is the leading feature of organic, as distinguished from functional, dysphagia, which is frequently intermittent, and by no means progressive. *Fifthly*, is there any regurgitation through the nose ? This feature implies paralytic dysphagia, with paralysis of the soft palate. *Sixthly*, is there any emaciation, or are there any symptoms referable to other organs ? Marked emaciation coming on early in a patient beyond middle life is characteristic of carcinoma.

§ **154. Physical Examination.**—(*a*) A careful *inspection* of the throat should be made, because the dysphagia may arise from tonsillitis or other pharyngeal conditions. The paralysis of the palate which succeeds diphtheria may thus be detected. Any swelling should be carefully examined, such as retro-pharyngeal abscess or tumour or a foreign body in this situation. I have known the bristle of a toothbrush entangled in the pharynx give rise to very serious difficulty in swallowing.

(*b*) In cases of dysphagia of any duration *the passage of an œsophageal bougie*, or, at any rate, a soft stomach-tube, should always be made.[1] The solid bougie is preferable, both for purposes of diagnosis and of treatment, but if carcinoma be suspected, great care must be exercised. The chest should always first be examined for aneurysm, and if this be found, the bougie should be avoided. The bougie must first be dipped in hot water in order to make it more flexible, and glycerine if necessary for lubrication. There is not much fear of it entering the larynx, provided the tube be passed to one or other side, and instruction given to the patient to put his head horizontally forwards and swallow during the operation. As the entrance to the stomach—from the teeth to the cardiac orifice—is a distance approximately of 16 inches, it is a good plan to tie a thread round the bougie 16 inches from its point ; then one can tell when it has reached the stomach. The œsophagus starts at the cricoid cartilage, opposite the sixth cervical vertebræ, and ends opposite a point between the ninth and tenth dorsal vertebræ, a dis-

[1] Dr. J. S. Bristowe, with characteristic candour, narrates a case showing the consequences which arose from a neglect of this procedure in his "Clinical Lectures and Essays," p. 43. The case was really one of dilatation of the œsophagus, which remained undiscovered until after death.

tance of 10 inches. The presence of acute *pain* during the passage of the instrument indicates ulceration, either simple or malignant. The presence of *blood*, and perhaps cancer cells adhering to the end of the tube, should be looked for as having the same significance as the foregoing. The presence of *dilatation* may be suspected when the end of the tube is not gripped, but is loose and easily movable. Occasionally a diverticulum or saccule of the œsophagus is formed, which by its pressure on the gullet above or below it causes obstruction. In such cases a bougie, which could not be passed before, may be passed after vomiting has occurred.

(*c*) *Auscultation* affords a valuable means of detecting both the presence and position of an œsophageal stricture. Place the chest end of a binaural stethoscope over the interval between the xiphoid cartilage and the left costal arch. Two gurgling sounds can be heard in this situation if the patient swallows *one* gulp of fluid ; the first is when it passes from pharynx to œsophagus, the second is when it passes from œsophagus to stomach. The normal interval between these two is *six seconds*, but if there be any obstruction in the gullet this interval becomes increased. If the first sound cannot be distinctly heard, the moment of its occurrence can be judged by looking at the throat. Again, by placing the stethoscope on the left side of the neck in a healthy person a gurgling sound will be heard during the act of swallowing. This normal sound may be *traced round and down the back* on the left side of the vertebral spines as low as the tenth dorsal vertebræ. But if a stricture be present it will be delayed or *absent below the seat of stricture.*

§ **155. Causes of Dysphagia.**—"*When a patient complains of difficulty in swallowing, or that the food returns to his mouth, the practitioner should first think of thoracic aneurysm, secondly of cancer, and thirdly of some other kind of ulceration.*"[1] The COMMONER CAUSES are—

I. A tumour pressing upon the gullet from the outside.
II. Epithelioma (cancer) of the gullet.
III. Simple or non-malignant stricture.
IV. Spasm.
V. Foreign bodies, acute œsophagitis, and simple ulcer.

LESS FREQUENT CAUSES are—

VI. Paralysis of the gullet.
VII. Dilatation of the gullet.

§ **156. A Tumour** pressing upon the gullet from without is perhaps the commonest cause of dysphagia, although malignant or simple stricture and muscular spasm are regarded by many as of equal frequency. Any intrathoracic tumour may, by its pressure, narrow the lumen of the gullet, and undoubtedly the commonest of these is aneurysm of the aorta. Other tumours are cancer of a neighbouring viscus, retro-

[1] Bryant, quoted by Fagge and Pye-Smith, "Prin. and Pract. of Med.," vol. ii., p. 316, second edition.

pharyngeal abscess or tumour, enlargement of the bronchial glands, lympho-sarcoma or other mediastinal tumour, goitre, pericardial effusion, and diverticula of the gullet filled with food (§ 162). The features common to all such tumours are the slowly progressive character of the dysphagia, the symptoms of pressure on other viscera, and sometimes, although usually not until late in the case, the physical signs of the tumour in question. For the rest, the differential features vary according to the nature and position of the tumour. In *aortic aneurysm* the amount of dysphagia is rarely very great at any time, although it is slowly progressive. Rest in bed will generally ameliorate the dysphagia. Difficulty of swallowing is only one of the pressure symptoms in this disease, and others should be looked for—*e.g.*, dyspnœa, abductor paralysis of the left vocal cord, and inequality of the pupils. The physical signs of aneurysm are commonly wanting in such cases on account of its deep-seated position.

§ **157. Malignant Disease** of the œsophagus is due in the large majority of cases to an epitheliomatous growth in the wall, usually primary, which goes on to ulceration, and forms a stricture from 1 to 4 inches long. Rarely the deposit is sarcomatous. The diagnostic features of epithelioma of the œsophagus are : (i.) The patient is past middle life. It is said to be more common in males. (ii.) The dysphagia becomes steadily and progressively worse. At first a difficulty only exists with solids, but later on fluids also are returned. The duration of the whole illness rarely exceeds twelve to eighteen months. (iii.) Emaciation and other evidences of cachexia occur quite early in the illness. There may be evidences of cancerous deposit elsewhere, especially within the abdominal cavity. (iv.) Pain and hæmorrhage, those frequent accompaniments of all malignant growths, are usually present and the pain is persistent and independent of, although aggravated by, food. (v.) The passage of a bougie is attended by considerable difficulty. The favourite sites of malignant stricture are opposite the cricoid cartilage, 6 inches from the teeth ; opposite the bronchi, 9 inches ; and at the cardiac orifice, 16 inches from the teeth.

Fibroma and *Myoma*, and other benign growths in the œsophagus, sessile, or in the form of polypi, are very rare. They may simulate simple or malignant stricture, and there may be hæmorrhage ; but the absence of any cachexia and the long duration without any increase of symptoms are the only means of suspecting the condition.

§ **158. Simple or Non-Malignant Stricture** of the œsophagus is most frequently caused either by the narrowing due to a syphilitic infiltration or the contraction which it subsequently leaves. It may also arise from the cicatrisation which follows a simple ulcer of the gullet or stomach ; or, thirdly, as the result of swallowing a corrosive liquid. Dilatation may take place above the stricture. The differential features of this condition are : (i.) The dysphagia comes on gradually, and, having reached a certain degree, is apt to remain stationary ; the

patient may be unable to swallow solids, but lives for many years on liquid food. (ii.) The passage of bougies gradually increased in size is possible, and this procedure gives some relief. (iii.) The patient may be young, or he may be of any age; the cachexia and emaciation of cancer are wanting; and pain is not a prominent feature in the case. (iv.) The gullet is apt to dilate above the stricture, and the food returns after an interval, which becomes progressively longer as the dilatation becomes greater. (v.) There is nearly always a history of one of the three causes above mentioned.

§ **159. Spasm of the Pharynx or Œsophagus** is, in the author's experience, one of the commonest causes of dysphagia. It is not infrequently associated with hysteria and other functional neuroses. Its differential features are fairly characteristic: (i.) The dysphagia is never progressive. It may come on somewhat suddenly, dating perhaps from an emotional shock or trouble, and it is very often intermittent, the patient being well enough in the intervals. Sometimes solids can be taken, while fluids are regurgitated, or *vice versa*. (ii.) It is unaccompanied by emaciation or cachexia; indeed, the patient sometimes appears to be in perfect health, a feature in which it differs from all other causes of dysphagia. There is usually little or no pain, and never any bleeding. (iii.) The dysphagia may last intermittently for a considerable time. I have known cases persist in varying degree for seven, twelve, and sixteen months. (iv.) The passage of a bougie, or flexible stomach-tube, is possible with a little steady pressure, and with the patient under chloroform it is easily done. This procedure generally results in curing the condition, at any rate for a time. (v.) The patient is most frequently of the female sex, and often presents other evidences of hysteria. It certainly occurs also in males, and gout or rheumatism are said to predispose to it.

§ **160. Foreign Bodies, Acute Œsophagitis, and Simple Ulcer.**—The symptoms of these conditions are much alike. Acute œsophagitis occurs after traumatism, as after swallowing corrosive fluids,[1] or in a localised form from the presence of foreign bodies. It sometimes occurs in the course of the specific fevers, and in infants at the breast from unknown causes. A slighter degree of *localised* inflammation arises by no means infrequently when a fish-bone, needle, pin, bristle of a toothbrush, or other solid particle, sticks in the folds of the œsophagus. This dysphagia takes the form of a difficulty and pain during the act of swallowing, at one particular spot. The symptoms here start suddenly and reach a maximum at once. This source of trouble is very apt to be overlooked when the patient has forgotten the incident which led to the lodgment of the foreign body. When the inflammation is *generalised*, there is great pain, with consequent spasm and regurgita-

[1] Fluids which are simply irritating, such as beer contaminated by the substances used to clean the pewter pots, may cause the condition.

tion on attempting to swallow. Thirst and, if the condition be severe, feverishness are present. Mucus, pus, and blood may be vomited if ulceration ensue.

Simple Ulcer of the gullet is very rare. It is sometimes due to syphilis. Acute pain and tenderness are prominent features, with spasm on swallowing or attempting to pass bougie. But the affection cannot be diagnosed with certainty.

We now turn to the **rarer causes of Dysphagia.**

§ **161. Paralysis of the Gullet.**—Paralysis of the upper part of the gullet—*i.e.*, of the *pharyngeal constrictors*—is not uncommon as an accompaniment and complication of diphtheria. Difficulty of swallowing under these circumstances may be one of the first evidences of diphtheritic paralysis. It also occurs in Bulbar Paralysis, and at the end of some slowly progressive exhausting diseases. All these differ from the other causes of dysphagia by being attended by regurgitation of fluids through the nose, owing to the paralysis of the soft palate. Paralysis of the gullet *below the pharynx* is a much rarer condition. It may sometimes accompany and be due to the same causes as the above. It also arises as an occasional complication of general paralysis of the insane, cerebral tumour, diseases of the nuclei in the medulla, and lesions of the vagus. The dysphagia in these cases is not absolute, the normal œsophageal sound on auscultation is absent, and *a bougie passes* without hindrance. The condition can only be distinguished from simple dilatation when there is no regurgitation or pseudo-emesis of food.

§ **162. Dilatation** or **Diverticulum of the Gullet** is not frequent, and the causes are obscure. It may take the form of (*a*) a *generalised dilatation* of the whole tube; (*b*) a *fusiform dilatation* above a stricture. (*c*) It may occur as a *diverticulum*, or sac, which is said to be formed in one of two ways: (i.) A *pressure* diverticulum or saccule, due either to weakness of the wall after injury, or sometimes to congenital weakness of some part of the tube, with consequent hernia of the mucous, through the muscular, wall; and (ii.) a *traction* diverticulum, due either to adhesions between the œsophagus and neighbouring glands, or other structures, pulling out the œsophageal wall as they contract.

(*a*) A general dilatation has but few or no symptoms. (*b*) The symptoms of variety (*b*) are masked by those of the stricture below. The occurrence of dilatation (with stricture) is evidenced by the regurgitation of food at shorter or longer intervals. On this account such cases are very apt to be mistaken for the vomiting of pyloric obstruction (*vide* feature 2, § 154). There is an unusual mobility in the bougie just before it reaches the obstruction. (*c*) The diverticular varieties are very rare, but, as far as we know, their symptoms are as follows: (i.) There is regurgitation of food after an interval varying from a few minutes to a few hours after ingestion. It is apt to be mistaken for persistent vomiting, but the ease with which the food is returned, and the absence of acid in it, should make us suspect this condition. (ii.) The regurgitation gradually increases in amount, and the breath is foul from the decomposition of food in the gradually enlarging pouch. (iii.) In cases of pressure diverticula a bougie which could not be passed before can be passed after vomiting, because the sac full of food forms a swelling that presses upon the gullet and so leads to obstruction. (iv.) Sometimes the pouch forms a definite tumour in the neck.

§ **163.** Prognosis and Treatment of Dysphagia.—Dysphagia is in most cases a symptom of considerable gravity, and in severe cases it commonly enough results in death by starvation. Of all causes, malignant stricture is the most serious, and, in spite of the means which modern surgery has placed at our disposal, patients rarely live more than a year or eighteen months. The length of time depends on the

maintenance of the nutrition of the individual. Next in order of gravity come tumours pressing on the œsophagus, when the prognosis depends on the nature of the tumour and its amenability to treatment.

Patients with simple stricture, and with dilatation, may live for many years on fluid diet, with or without gastrostomy, but diverticula are much more serious. Of all causes functional spasm is the most curable, although it is apt to return.

The cause of death in dysphagia is usually starvation or a low form of pneumonia. This may arise from perforation into the bronchus, or by the food passing into the glottis. In either case death is expedited by the lowered vitality of the patient. Perforation may occur in other directions—*e.g.*, a case of malignant disease of the gullet under my care died from hæmorrhage consequent upon perforation into the aorta.

Treatment of Dysphagia.—The indications are to remove the cause of the obstruction, to maintain the strength and nutrition of the patient, and to relieve any concurrent symptoms. The question of three surgical procedures may arise in these cases: the passage of bougies of different sizes, the use of Symonds' tubes, and gastrostomy. If possible a bougie should be passed in all cases, not only for purposes of diagnosis, but also as part of the treatment. It may be of little use in malignant stricture, but simple stricture may be dilated or prevented from further contracture by this method. Symonds' tube, a funnel-shaped tube with a string attached to prevent it slipping down, changed every three weeks or so, undoubtedly prolongs life both in malignant and advanced simple strictures. In malignant stricture if, when the case comes under treatment, debility is very marked, complications are present, and there are evidences of cancer elsewhere, neither Symonds' tube nor gastrostomy are of any use, and the only treatment available is rectal feeding. In addition to the above treatment, the only indication in *malignant* stricture is to soothe the pain by morphia, opium, or cocaine.

In *simple* stricture, bougies of gradually increasing size should be passed and left in for some hours at a time. Force must not be used in so doing. In very narrow strictures a Symonds' tube would be better. If syphilis be suspected as the cause, potassium iodide must be given. In *functional* spasm a bougie should be passed, and cold douches given along the neck and the spine. The general condition must be treated, valerian in hysteria, combined with special diet in cases with gastritis. Electricity may be useful. In *paralysis* and *dilatation*, especially the diverticular type of dilatation, the patient must be fed by a stomach-tube. If the diverticulum is high up in the neck, the surgeon may be able to remedy it. In *acute œsophagitis* the pain must be soothed by morphia hypodermically, by cocaine lozenges, or by opium given with tragacanth. Thirst may be allayed with spoonfuls of iced water, in which small doses of opium, cocaine,

and milk may be administered. During the acute stage the patient may require nutrient enemata. *Foreign bodies* in the gullet need prompt attention but very careful measures, else they may pierce the tube and injure the aorta or other structures around.

Feeding by a stomach-tube is a measure available in a fair proportion of cases especially in Causes I., III., IV., VI., and VII. (*supra*). The only apparatus necessary consists of a long flexible rubber tube (5 feet long) one end of which is blunt, with the " eye " at the side or the end (according to choice) and the other end tied to a funnel. The method of passing the tube is either the same as that used in passing a bougie (§ 154) or it is passed through the nose. In the latter case the size must be smaller. The operator then pours into the funnel the fluid food, previously prepared, from a jug.

CHAPTER IX

THE ABDOMEN

THE abdomen contains a large number of very important organs and structures, but just as their physiology and pathology are in many instances obscure, so also are the means at our disposal for their thorough clinical investigation imperfect. However, it is in this region that we have to deal with symptoms which on the one hand may be of quite a trivial order, or on the other may be of extreme gravity; symptoms and conditions the issue of which will largely depend on the promptitude, knowledge, and skill of the medical man in attendance, and upon his adequate comprehension of their true meaning.

PART A. SYMPTOMATOLOGY.

§ **164. Local Symptoms.**—The symptoms referable to disease situated within the abdominal cavity are necessarily of the widest and most varied kind, but there are only three which are sufficiently constant to be regarded as cardinal symptoms, all of which are local—viz., ABDOMINAL PAIN, GENERALISED ENLARGEMENT, and LOCALISED TUMOUR.

VOMITING is a fairly constant accompaniment of all acute abdominal conditions, whether the stomach is involved in the lesion or not. Its causes are discussed in § 191.

The presence of DIARRHŒA and CONSTIPATION depends very largely on whether the intestinal canal is affected, and these are fully dealt with in Chapter XI. The other symptoms also depend largely upon which of the abdominal organs is affected, with one important exception—viz., "INDIGESTION." In all chronic abdominal disorders, no matter which organ is affected, we are often consulted for "INDIGESTION"; in fact, nausea and all the other symptoms of pronounced dyspepsia may be due to disease quite unconnected with the stomach, and located, for instance, within the uterus, kidneys, liver, spleen, or pancreas. Some cases of "dyspepsia," after resisting treatment for months or years, have been cured by the stitching up of a dislocated kidney.

Abdominal Pain, if acute and sudden, is a medical emergency of the most important kind; if chronic it presents many difficult questions for diagnosis. It will therefore merit the most careful study and analysis (§ 168). The diseases *outside the abdomen* which may give rise to it are only three in number:

1. *Diaphragmatic pleurisy*, or a basal pleuropneumonia, may give rise to acute abdominal pain of sudden onset and to abdominal rigidity and other symptoms of acute peritonitis, which can only be differentiated by the pulse-respiration ratio and the concurrent symptoms.

2. *Neuralgia* of the intercostal and other spinal nerves may be referred to the abdomen. In this way spinal caries, especially in children, may be mistaken for various abdominal diseases.

3. An *abscess* in the abdominal wall may be similarly mistaken, but this ought to present no difficulty.

Abdominal Enlargement and Abdominal Tumour are fully considered in Part C.

§ **165.** The **General** or **Remote Symptoms** met with in abdominal disorders are, as just mentioned, of an extremely varied nature, and our endeavour should be to correctly associate these symptoms with the abdominal organ which is affected.

Collapse and Pulse-Temperature Ratio.—In connection with the general symptoms of abdominal diseases, one fact needs special mention —(1), the profound collapse which is so apt to be associated with all acute abdominal conditions. A blow on the abdomen may result in fatal collapse, and so also may perforative peritonitis. A person with endocarditis, pleurisy, or even pneumonia, may walk to the doctor, but with any acute abdominal condition he takes to bed instinctively. This tendency to collapse possibly finds an explanation in the fact that the chief centre of the sympathetic is situated within the abdomen. Now, a subnormal temperature is one of the symptoms of collapse, and for this reason the temperature rarely ranges very high even in the gravest abdominal inflammations, especially in their earlier phases. In acute peritonitis, for instance, an extensive inflammatory process affects the peritoneum, which acting alone might produce a temperature of 105° F. or more, but by reason of the collapse it is rarely more than 102° or 103° F. (2) *In the pulse*, however, *we find our best guide to the severity of mischief within the abdomen.* In all acute diseases, other than abdominal, we find a rough general proportion between the height of the temperature and the rate of the pulse. Thus, a temperature of 100° F. will correspond roughly with a pulse of 100, 101° with 110, 102° with 120, 103° with 130, and so on—an increase of about 10 for every 1° F. But in acute abdominal conditions this is not so. The pulse-temperature ratio is disturbed, for although the pulse rate increases with the severity of the abdominal mischief, the temperature never increases proportionately. Indeed, in many of

the worst cases, the temperature is one or more degrees below normal. The pulse, however, is an almost infallible guide, and one may say (1) that if the pulse remains under 100 nothing very serious is happening within the abdomen ; and (2) that the rate of the pulse and the pulse-temperature ratio are great aids to the diagnosis, and in some sense measures, of acute abdominal disorder, especially when that disorder has reference to the peritoneum.

PART B. PHYSICAL EXAMINATION.

§ **166.** In the examination of the abdomen we must proceed systematically, as in the examination of the thorax, by INSPECTION, PALPATION, PERCUSSION, MENSURATION, and occasionally auscultation ; though of all these measures palpation by the educated hand is at the present time the most valuable means we have.

1. **Careful inspection of the abdomen** should on no account be omitted ; much can be learned in this way. The best point of view is that from the foot of the bed, or by bending over the patient's feet, so as to view the abdomen from below. The mere fact of enlargement may thus be verified, and whether the enlargement be a generalised and uniform one, or whether it be localised or asymmetrical. Notice whether the umbilicus is centrally situated, and also whether the surface presents dilated veins, such as occur in abdominal cancer, or when the portal vein or vena cava is obstructed. Dilatation of the abdominal veins is met with chiefly in three conditions : (1) In liver cirrhosis, these veins being part of the conservative collateral circulation which gradually becomes established ; (2) the veins without being much dilated or prominent are *unduly apparent* in cases of abdominal carcinoma. It is a sign of considerable value and constancy. (3) Extreme dilatation and varicosity of the superficial veins occurs only when the inferior vena cava is obstructed. This is generally due to a gummatous deposit in or around the posterior border of the liver where the vena cava passes through it. The veins of the legs and testes generally share to a less extent in the dilatation.[1] Notice also whether there is any thickening or infiltration round the umbilicus such as occurs in cancer and tuberculous peritonitis. An abdominal enlargement due to the presence of air or gas is rounded anteriorly, but when due to fluid it is usually flattened in front and the flanks bulge ; when it is due to the presence of a solid tumour it is irregular. Incidentally you may notice the presence or absence of the white lines (lineæ albicantes) left by a previous pregnancy, the knowledge of which may be medically useful. The amount of movement of the abdominal wall with inspiration should be noticed, for diminished or absent movement

[1] A case is recorded by Dr. W. Chapman, Clin. Soc. Trans., 1899 and 1900, and *Lancet*, December 2, 1899.

constitutes an important sign of peritonitis. If the peritonitis is local, the abdominal wall over that area may move badly, whilst elsewhere abdominal respiratory movement is normal. Pulsation may sometimes be seen, most often in the epigastrium, and may be due to the right ventricle or an engorged liver secondary to heart failure. Sometimes aortic pulsation is unduly visible, especially in neurotic dyspeptic women, or it may be transmitted by a pyloric tumour. Rarely the pulsation is due to an abdominal aneurysm. Visible peristalsis should also be looked for, and if present its position and direction should be noted.

The Regional Anatomy of the Abdomen is important as a guide to the seat of disease (Fig. 61).

2. Palpation.—Considerable experience is necessary for satisfactory abdominal palpation. The hand should be warmed and always laid *flat* on the abdominal wall; then by gently dipping in the fingers, by flexing the metacarpo-phalangeal joints, we have the most ready method of ascertaining (1) the presence of any tumour; (2) the boundaries of some of the solid organs. The patient should lie on his back with the knees drawn up and the shoulders somewhat raised, so as to relax the abdominal muscles. Do not use the tips but only the pads of the finger, for they stimulate the recti muscles to contract, and thus to simulate a tumour where none exists. Many patients offer considerable involuntary or voluntary resistance, and this must be overcome by placing them in an easy posture and distracting their attention, or asking them to "let the breath go." The palpation and percussion boundaries of the different organs are described in later chapters.

3. Percussion of the abdomen is done with the same precautions as in the case of heart and lungs, and the student will now find it very convenient to be able to percuss with either hand indifferently. Normally the anterior surface of the abdomen is resonant (when the stomach and intestines are empty) as far upwards as liver and spleen, downwards as far as the pubes, and outwards as far as the outer border of the colon. By this means we ascertain the presence of solid and fluid, which are dull, or of gas, which is resonant.

4. By measurement we ascertain the amount of increase in size. As a general rule, horizontal measurement should be taken at the level of the umbilicus, and it should be recorded for future reference. In order to ascertain whether the enlargement is symmetrical, we measure from the umbilicus to the ensiform cartilage above and the pubes below, and from the umbilicus to the anterior superior spine on each side. These four measurements should be approximately equal. From these data we ascertain very slight deviations from symmetry.

The fallacies of abdominal enlargement are: (1) *Fat in the omentum* is referred to under fluid enlargement (§ 185). (2) *Phantom tumour*

is described under abdominal enlargement due to gas (§ 183). (3) *Pendulous abdomen*, so frequent in elderly women, is often thought by the patient to be a " tumour," but it is due only to weakness of the muscles

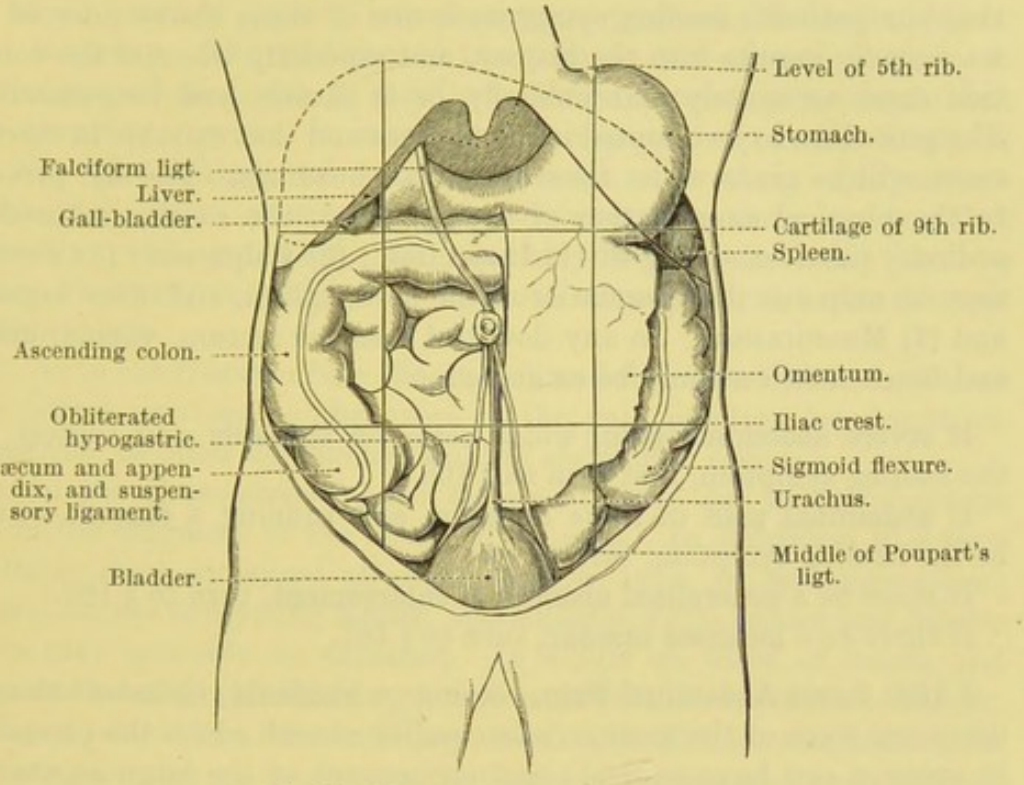

Fig. 61.—Regions of the Abdomen.

For purposes of convenience the abdomen is divided into nine regions, which are bounded by two imaginary lines running vertically upwards on each side of the abdomen, from the *middle* of Poupart's ligament to the costo-chondral articulation above, and two horizontal lines running round the abdomen on a level with the end of the ninth costal cartilage and the anterior superior spines respectively. Their names and the organs they contain are as follows :

Right Hypochondriac.

The right lobe of the liver and the gall-bladder, the duodenum, pancreas, hepatic flexure of the colon, upper part of the right kidney, and the right suprarenal capsule.

Right Lumbar.

Ascending colon, lower part of the right kidney, and some convolutions of the small intestines.

Right Iliac.

The cæcum, appendix cæci, and ureter.

Epigastric Region.

The middle and pyloric end of the stomach, left lobe and lobulus Spigelii of the liver, and the pancreas.

Umbilical Region.

The transverse colon, part of the great omentum and mesentery, transverse part of the duodenum, and some convolutions of the jejunum and ileum.

Hypogastric Region.

Convolutions of the small intestines and the bladder in children and in adults when distended, and the uterus during pregnancy.

Left Hypochondriac.

The splenic end of the stomach, the spleen and extremity of the pancreas, and the splenic flexure of the colon, upper half of the left kidney and the left suprarenal capsule.

Left Lumbar.

Descending colon, part of the omentum, lower part of the left kidney, and some convolutions of the small intestines.

Left Iliac.

Sigmoid flexure of the colon and ureter.

of the abdomen and of the intestinal tube. (4) *Pregnancy* is referred to among the localised enlargements (§ 188). (5) In *rachitic children* the liver and spleen may be pushed down by the deformity of the costal arches, and so produce the appearance of an enlarged abdomen.

PART C. ABDOMINAL DISORDERS: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **167. Routine Procedure and Classification.**—Having *first* ascertained that the patient's leading symptom is one of those above referred to, we *secondly* inquire into the history, and especially whether the condition came on acutely and suddenly, or is chronic and long-standing. The procedure to be adopted in acute cases and that suitable in chronic cases, will be given under these respective headings. *Thirdly*, proceed to the physical examination of the abdomen, the routine method in ordinary cases consisting of (1) Inspection; (2) Palpation; (3) Percussion, to map out the boundaries of the liver, spleen, and other organs; and (4) Mensuration. In any doubtful case the rectum, vagina, urine, and fæces must certainly be examined.

If **severe abdominal pain,** which came on **suddenly** and acutely, be the leading symptom, first turn to § 168.

If **abdominal pain** of some duration and running a **chronic** course be the leading symptom, turn to § 174.

If there be a **generalised abdominal enlargement,** turn to § 182.

If there be a **localised tumour,** turn to § 187.

§ **168. Acute Abdominal Pain,** coming on **suddenly,** includes amongst its causes some of the most serious conditions with which the physician or surgeon can have to deal; and on account of the large number of organs contained in the abdominal cavity, these causes include many pathological processes situated in various and often unsuspected positions.

The *causes* of abdominal pain may be conveniently classified for clinical purposes into nine groups:

A. ABDOMINAL PAIN coming on **suddenly, with collapse.**

I. Perforation of some organ or cyst (perforative peritonitis) ..	§ 169
II. Acute peritonitis due to causes other than the preceding ..	§ 170
III. Acute intestinal obstruction (hernia, intussusception, internal strangulation, and appendicitis)	§ 228
IV. Displaced enlarged (or gravid) uterus; V. embolism of the mesenteric artery; VI. Pancreatic hæmorrhage	§ 171

B. ABDOMINAL PAIN coming on **suddenly, without collapse.**

VII. Colic (Intestinal, renal, biliary)	§ 172
VIII. Appendicitis (some cases); floating kidney; splenic embolism; and some other obscure organic affections ..	§ 173
IX. Visceral Neuralgiæ	§ 173

In the first SIX the acute abdominal pain is usually ATTENDED BY COLLAPSE, but not in the last three. This, however, is only relative, and in any doubtful case the whole should be passed in review.

In order to ascertain which of these causes is in operation, and in view of the gravity of some of these cases, it will be desirable to consider the METHOD OF PROCEDURE in some detail.

(1) Regarding the *cardinal* or *leading* symptoms, inquire carefully, as in all cases of "pain," concerning its position, character, degree and intensity. The position of the pain is not always a guide to the organ affected, for it rapidly tends to become generalised; but the direction in which it is radiated is of great help in the diagnosis of the three kinds of colic. Whenever the three symptoms—ABDOMINAL PAIN, VOMITING, and CONSTIPATION—come on together suddenly, with COLLAPSE, the condition is very probably due to either PERITONITIS (which may be due to PERFORATION), or INTESTINAL OBSTRUCTION.

2. As to the *History of the Illness*, it is useful to note if there had been any illness previous to the onset of the pain pointing to ulceration, dyspepsia, or other derangement of the abdominal organs.

3. In the *Examination of the Patient*—(i.) his *age* is an important aid in the diagnosis of the cause of the pain. In childhood it is very probably some intestinal affection, such as colic, or intussusception; in adolescents and young adults, appendicitis, while cancer and tabetic crises may probably be excluded. In adults we think of hernia and ulcer of the stomach; in old age or after middle life we think of cancer, or if the patient is a female, biliary colic. (ii). The *sex* may aid us, for in young females we may suspect an ulcer of the stomach even without previous symptoms; and in older women the rupture of an ectopic (extra-uterine) pregnancy, a condition which is frequently overlooked, or gall-stones. (iii.) The presence or absence of *tenderness* is of considerable aid. If it is quite absent peritonitis may be excluded, and its position will aid us in localising the lesion more than does the seat of the pain, as, for instance, MacBurney's point in appendicitis. (iv.) *All the organs* of the abdomen must be as carefully and as thoroughly examined as circumstances will permit. Never forget to examine per rectum and vagina, because stricture of the former or a pelvic tumour may throw considerable light upon the case. (v.) The patient's *general symptoms* must also be carefully investigated. If the temperature *and the pulse* be normal, we may exclude inflammatory conditions. The temperature alone is not a sufficient guide in this respect (see § 165), but in general terms no serious acute abdominal condition exists without the *pulse rate* exceeding 90 or 100. If the patient is much emaciated, in adults we must bear in mind obscure malignant disease, and in children the presence of tubercle.

If the pain, which is severe and has come on suddenly, is **attended by marked collapse,** first turn to § 169. If it is **unattended by collapse,** turn first to § 172. It must be remembered, however, that any severe pain will cause a certain amount of prostration.

I. *The patient complains of* **acute abdominal pain,** *which has come on* **suddenly,** *with symptoms of severe* **collapse,** *attended by* VOMITING *and* CONSTIPATION ; *the pulse is rapid* (*over* 100). *The case is probably one of three conditions*, PERFORATION into the peritoneum, ACUTE PERITONITIS, *or* ACUTE INTESTINAL OBSTRUCTION.

§ **169. Rupture of a Cyst, Abscess, Organ, or Perforation of the Alimentary Canal** (which shortly develops into Perforative Peritonitis). The *cysts which may rupture* are hydatid or simple cysts of the liver, kidney, pancreas or other organs, ovarian and parovarian cysts, and the abscesses those of the liver, gall-bladder, kidney or other organs, and perityphilitic abscess (§ 175). *Rupture of an Organ*, with consequent extravasation of blood, causes similar symptoms, and of such may be mentioned ruptured Fallopian tube (in cases of extra-uterine pregnancy), ruptured abdominal aneurysm, rupture of the liver or kidney (following injury). *Perforation* of the alimentary canal may at any time occur when an ulcer is present. These ulcers are, in order from above downward, simple ulcer of the stomach (which is usually met with in young anæmic women), simple ulcer of the duodenum (which occurs in males, or may follow burns), ulcer of the lower part of the ileum (due to tuberculosis or enteric fever), ulcer of the cæcum or appendix, ulcer of the large intestine, especially the sigmoid flexure (usually cancerous, dysenteric, or syphilitic).

Symptoms.—Pyrexia at first is absent, and the temperature may be subnormal because of the collapse. The pulse is thready, feeble, and rapid. The pain is probably very severe, and the ashen pallid face, with its cold, clammy sweat and sunken eyes, are very characteristic. Vomiting is rarely absent ; it may be incessant, distressing, and even stercoraceous. A certain amount of constipation is generally present on account of the paralysis of the bowel consequent on the pain, and by this feature these cases are distinguished from corrosive poisoning. Perforated *gastric ulcer* is perhaps the commonest of the conditions above mentioned, and may be taken as a type. We should inquire for a history of dyspepsia and other symptoms (§ 207), but in not a few cases rupture has occurred without previous symptoms of any kind whatever. On examination there is tenderness most marked in the epigastrium, rigidity of the muscles, and a tympanitic note over the whole abdomen. The disappearance of the liver dulness in a case presenting these symptoms has been considered pathognomonic of ruptured gastric ulcer. After a few hours there is a deceptive period of repose, during which all symptoms of discomfort are diminished. On examination several hours later, however, symptoms of collapse with acute peritonitis (§ 170) are found, *generalised*, in a case where there were no adhesions at the site of rupture, or *localised*, where adhesions were present. The symptoms of perforated *duodenal ulcer* may be the same as those of gastric ulcer, but the condition occurs usually

in men. The symptoms of perforation of another part of the intestine, or rupture of a cyst, are much the same, and one can only hazard a diagnosis as to its situation by the site of the pain and tenderness, and the previous history. There are three degrees of severity met with when perforation of the intestine occurs : (*a*) When there are adhesions the peritonitis may be localised or partial ; (*b*) when there are no adhesions, but a small leakage, it may be only moderately sudden in its onset ; (*c*) when the leakage is large it will be extremely sudden and severe in its onset.

The *latent period* which ensues shortly after an acute onset deceives many clinical observers. The pain may subside, all symptoms decrease, and the temperature become normal or subnormal. But (1) the *pulse remains persistently high*, and (2) in blood-films the polynuclear cells, and perhaps the total leucocytes, will be markedly increased. These are sufficient to indicate immediate exploratory abdominal section. Perforative peritonitis may have to be diagnosed from diaphragmatic pleurisy and pneumonia of the base, where the pulse-respiration ratio is disturbed, but not the pulse-temperature ratio.[1]

Treatment and Prognosis.—Laparotomy should be performed at once. If deceived by the period of repose into thinking the patient is recovering, in a few hours general peritonitis will have set in, and operative interference will be too late. In cases where patients have been operated upon within the first twelve hours 79 per cent. have recovered ; if after twenty-four hours only 29·4 per cent. have recovered (Goffe). The after-treatment depends on the cause. In the case of rupture consequent on injury, internal hæmorrhage takes place with a fatal result, but even in such cases laparotomy has been performed with satisfactory results.[2]

II. *The patient complains of severe* **abdominal pain,** *extreme* **prostration,** *and* VOMITING ; *there is* THORACIC RESPIRATION, *and the* TEMPERATURE IS ELEVATED. *The disease is* ACUTE PERITONITIS.

§ **170. Acute Peritonitis** (General Peritonitis) is an acute inflammation of the peritoneum. It is rarely a primary disease, but its onset is usually sudden.

Symptoms.—(1) The aspect is very characteristic ; the countenance has an anxious pinched look, the cheeks flushed, and the skin cold and clammy. (2) The pain is severe and constant, but liable to exacerbations on account of the intestinal peristalsis and the passage of wind along the bowel.[3] It is also increased by any kind of movement, even by the respiratory movements. Consequently (3) the respiration is

[1] See Report of Clin. Soc. Lond., the *Lancet*, April 19, 1902.
[2] *E.g.*, in case of ruptured kidney (Bland-Sutton, Clin. Soc. Trans., 1899-1900).
[3] The acute peritonitis which complicates enteric fever is of a latent character, and unaccompanied by pain. This and puerperal peritonitis are the only exceptions.

thoracic, and careful inspection will show that (4) the abdominal walls are immobile and rigid. There is acute tenderness on pressure, so much so that the weight of the bed-clothes can hardly be borne. (5) The posture of the patient is very characteristic as he lies on his back with legs drawn up to relax the abdominal muscles. (6) Pyrexia, ushered in suddenly with rigors, and attended by a small, wiry, rapid pulse of 100 to 104 per minute. The temperature is elevated only 2° or 3° F. above normal, and maintained there continuously, unless pyæmia be present, in which case there are rapid variations of wide range. In some cases—*e.g.*, perforation—it may be subnormal at first (*vide supra*). There is marked prostration, as in all abdominal inflammations, and a great tendency to collapse, even from the beginning. (7) The bowels are constipated, and there is persistent vomiting. Hiccough is often present, and if persistent is a very bad sign, as in all abdominal disorders. There is diminution of urine, which may amount to suppression. The urine is abundantly charged with indican. Death occurs from collapse or asthenia, and the mind remains quite clear until the end in uncomplicated cases. Peritonitis is seldom a primary affection, and careful inquiry should reveal the cause.

The *Causes* of acute peritonitis may be grouped under six heads: (i.) *Injury or Operation.*—In cases occurring in women without obvious cause, the possibility of criminal procedure for abortion should always be remembered. As regards surgical operations on the belly, modern experience has shown that it is not so much the actual injury as the introduction of septic organisms, which produce the peritonitis, and that if these be excluded mere damage to the peritoneum will not cause a generalised peritonitis. (ii.) *Extension* of inflammation from the thorax, or from various organs of the abdomen—*e.g.*, appendicitis, gonorrhœal salpingitis, inflammatory conditions of the intestines (typhoid, dysenteric, and other). (iii.) *Blood Infections* of various kinds—*e.g.*, streptococcal, staphylococcal, and gonorrhœal. Peritonitis is apt also to complicate scarlatina and the other acute specific fevers. *Puerperal Peritonitis* is due to the introduction of a pyogenic infection through the raw uterine surface. A chronic form of the disease arises in *uræmia.* (iv.) *Idiopathic Peritonitis* was the name formerly given to the disease when no cause could be discovered. But there are good grounds for believing that such cases are due to the entrance of micro-organisms into the blood, probably through the intestinal wall previously rendered pervious by damage or disease. Some are due to pneumococci and *Bacillus coli communis*, which may produce peritonitis either as part of a septicæmia, or primarily. (v.) *Chill*, under certain conditions, such as bathing during the menstrual period, is sometimes included as a cause, though this usually leads to a chronic localised peritonitis (perimetritis). Apart from this it seems probable that the true explanation of peritonitis after a "chill" is to be found

under heading (iv.)—viz., microbic infection. (vi.) *Rupture* of some abdominal cyst, such as ovarian cyst, or an abscess of the liver, or rupture of the gall-bladder, etc. (§ 169). Rupture of a Graafian vesicle may give rise to a monthly peritonitis, but this is usually localised and less serious. (vii.) *Perforation of some part of the alimentary canal*, which had previously become thin by ulceration—ulceration of the appendix vermiformis, simple ulcer of the stomach (malignant ulcer rarely or never perforates because of the infiltration around), typhoid ulcer of the ileum, etc. (see Perforative Peritonitis).

Acute peritonitis has to be *Diagnosed* from four diseases : (1) Acute intestinal obstruction, in which the constipation is absolute and no flatus is passed ; there is usually no pyrexia, and the constitutional disturbance is usually less. (2) In *colic*, although the pain is also very severe, there is an absence of tenderness, and pressure may give relief. Pyrexia and collapse are absent, and the pulse is normal. (3) In *catarrhal enteritis* there is pain, and there may be vomiting and tenderness on pressure, but in this disease there is profuse diarrhœa. (4) In certain cases of *hysteria*, acute peritonitis may be very accurately simulated, though the temperature and pulse are normal, there is very little collapse, and there are evidences of the hysterical stigmata.

The *Prognosis* is always very serious. As regards etiology, perforative peritonitis, formerly considered the gravest, is probably now the most hopeful if promptly dealt with. Modern surgery has done much for the rescue of such cases, and undoubtedly the most favourable of them is that due to appendicitis. Cases of this disease, if properly managed, should hardly ever be lost. The prognosis in any particular case depends in general terms on the cause and the severity of the collapse, the dyspnœa, and the hiccough.

Treatment.—The treatment of acute peritonitis depends upon whether it is general or local. If *general* the only rational treatment is by operation immediately a diagnosis has been made. A fatal issue is almost invariable in cases not operated upon, since the condition is rarely primary, and a definite local lesion is usually present. Even with operation the prognosis is very grave, but this is largely due to the late stage at which it is usually performed. If for any reason an operation cannot be done recourse must be had to the older methods of treatment. In *local* peritonitis medical treatment is indicated, but even in this condition, if there are signs which make it probable that pus has formed, an exploratory incision should be made. Medical treatment comprises keeping the patient in bed and relieving symptoms. The diet should be fluid, consisting chiefly of milk, to which stimulants may be added according to the condition of the pulse. Local applications may give relief, especially cold in the form of icebags, or ten or twelve leeches to the abdomen. Fomentations, either simple or with tincture of belladonna, relieve the pain. The most valuable drug is opium, for

it relieves the pain, and reduces the peristalsis of the bowel, and so gives local rest. It may often be given in fluid form by the mouth, and can be tolerated in large doses. If vomiting persists it should be administered hypodermically. If there is any doubt as to the advisability of a surgical operation, either immediately or later, opium should be withheld, for by masking the symptoms it may lead to a continuation of medical treatment when operation is called for. It is therefore of use chiefly in local peritonitis, or in general peritonitis where an operation is not permitted. Purgatives are better avoided, but the lower bowel should be opened by means of enemata. The hiccough may be relieved by giving ice to suck, and by opium or chloral.

III. *The patient complains of* **acute abdominal pain** *which is* **attended by collapse,** *and the* PULSE IS RAPID ; *there is* ABSOLUTE CONSTIPATION, *with inability to pass even flatus, and* VOMITING (*at first of food, then of bile, and finally of stercoraceous matter*)—the condition is ACUTE INTESTINAL OBSTRUCTION.

Acute Intestinal Obstruction—*i.e.*, obstruction coming on suddenly, is always a matter of serious importance, and every practitioner should be thoroughly acquainted with its several causes. In actual practice, whenever the three symptoms, **constipation, vomiting,** and **abdominal pain** occur together, one of three conditions should be suspected—acute peritonitis, intestinal obstruction, or colic.

The various causes of acute intestinal obstruction—the chief of which are External Hernia, Internal Strangulation, Intussusception, and Appendicitis—are fully dealt with under Intestinal Disorders (Chapter XI.), Appendicitis being described below (§ 175).

§ **171.** *The patient complains of* **acute abdominal pain,** *with more or less* **collapse** ; *the temperature is probably normal or subnormal, but the symptoms do not quite conform to any of the preceding*—some of the **rarer causes** are probably in operation, such as the following :

IV. **Displacement of a Gravid Uterus** is known by the pain being referred to pelvis, and examination revealing the local mischief. It may occur when jumping from a height, and performing active exercise, especially in early pregnancy (Chapter XIV.).

V. In **Embolism of the Mesenteric Artery,** a cause of embolism, such as endocarditis, is present. It is rarely diagnosed during life. The absence of symptoms pointing to the other causes may lead one to suspect embolism.

VI. **Acute Pancreatitis.**—(1) The pain here is very sudden and severe, usually in the upper part of the left side of the abdomen ; (2) vomiting and constipation are usually present ; and (3) there is usually tympanitic abdominal distension (see also § 181).

VI*a*. **Hæmorrhage into the Pancreas** is attended by : (1) severe and sudden pain in the upper part of the abdomen, and that part soon becomes tender ; (2) vomiting of increasing severity ; (3) symptoms of collapse, with the restlessness and subnormal temperature which accompany collapse when it is due to hæmorrhage (§ 181).

Very few cases of VI. and VI*a*. have been recorded,[1] and the *Diagnosis* of both of

[1] See Middleton-Goldsmith Lecture, *New York Medical Record*, 1889, vol. i.

these conditions from intestinal obstruction or perforation into the peritoneum is usually impossible before laparotomy. In both diseases death from collapse is the usual result. In acute pancreatitis death occurs usually in four days.

VII. *The patient complains of* **acute abdominal pain,** *which has come on* **suddenly** *while apparently in good health* **without** *definite* **collapse** ; *the pulse does* NOT EXCEED 100 ; *there may be* VOMITING *and constipation.* The case is probably one of the three kinds of COLIC, though APPENDICITIS, VISCERAL NEURALGIÆ, and some OTHER AFFECTIONS may start in this way.

§ **172. Colic** is a somewhat vague term applied to spasmodic paroxysmal pain situated in the abdomen. There are three kinds—**intestinal, hepatic,** and **renal colic**—and they have the following features in common : (1) The pain is extremely severe, and sudden in its onset ; (2) not infrequently there is vomiting from the severity of the pain ; (3) the face is pale and " anxious," and in severe cases the pulse is rapid and feeble, though it practically never exceeds 100 ; (4) *the temperature is neither above nor below normal ;* (5) the physical signs in the abdomen are negative, and the pain may even be relieved by pressure.

(*a*) **Intestinal Colic** is due to spasm of the bowel caused by the irritation by masses of hard pieces of undigested food, such as raw apples. The pain of intestinal colic is characteristically twisting, paroxysmal, and limited to the abdomen, principally around the umbilicus, and is relieved by pressure which distinguishes it from peritonitis. The abdomen may be distended with flatus. Sometimes it is followed or accompanied by diarrhœa, or, as in lead colic, by constipation. The pain of colic due to lead-poisoning is accompanied by a slow and hard pulse, with contraction of the abdominal muscles and other symptoms of plumbism, such as a blue line on the gums ; and a history of working amongst lead is obtainable.

(*b*) **In Hepatic Colic** which is due to the passage of a gall-stone into the bile duct, the pain shoots *upwards* to the right shoulder and backwards, never downwards ; a dull pain continues during the intervals between the spasms. After lasting a few hours or a day or two it is followed by jaundice.

(*c*) **Renal Colic** is due to the passage of a calculus along the ureter. The pain radiates *downwards* from the loin to the thigh and the testicle of the same side, which is often retracted. It may last for a day or two. During the attack micturition is frequent ; sometimes there is hæmaturia or strangury. There will probably be a history of gravel in the urine, or attacks of a similar nature.

The *Diagnosis* of intestinal from other forms of colic is given in Table XIII., p. 256.

Prognosis.—The course of an attack of colic is short and severe. If the patient does not recover in the course of one or two days, we

must suspect one of the more serious conditions mentioned on p. 248 The pulse must be carefully watched.

Treatment.—For all forms of colic some of the following measures—hot fomentations, a warm bath, belladonna, opium, or chloroform, as local applications, and hypodermics of morphia (gr. $\frac{1}{6}$, with atropin gr. $\frac{1}{60}$)—may be necessary to alleviate the extreme pain. Large draughts of warm water should be taken. For intestinal colic in particular, a full dose of castor oil, with 20 minims of laudanum, should be given, followed by saline purgatives. If it is due to lead-poisoning start a course of treatment with small doses of potassium iodide and mag. sulph.; and for prevention, scrupulous cleanliness and the systematic drinking of lemonade containing sulphuric acid are advisable. Hepatic and renal colic are treated under gall-stones (§ 241) and renal colic (§ 301).

TABLE XIII.—DIAGNOSIS OF COLIC.

	Character and Distribution of Pain.	*Associated Symptoms.*	*Age and Sex of Patient.*
Intestinal.	Twisting, around umbilicus, paroxysmal; relieved by pressure.	Constipation (or diarrhœa). No jaundice.	Any age or sex. Sometimes evidence or history of plumbism.
Biliary.	In right hypochondrium shooting upwards to right shoulder, constant, but also in paroxysms.	Jaundice soon supervenes. Other hepatic symptoms may be present.	Female sex. At or after middle life.
Renal.	In loin, shooting down to thigh and testicle or ovary of same side.	Crystals or other urinary change, hæmaturia. No jaundice. Sometimes frequent micturition or strangury.	Usually male. Children and adults.

§ **173.** VIII. Among the **rarer causes of abdominal pain** without collapse are various OBSCURE ORGANIC AFFECTIONS of the abdomen, evidenced at first only by pain. Two may be mentioned which came under my notice, PANCREATIC CALCULUS and OBTURATOR HERNIA, in both of which the only symptom for some time was pain coming on SUDDENLY without collapse. In the former the pain was extremely severe, and of a paroxysmal character, situated just below the umbilicus; later on it was associated with fat in the fæces, emaciation, and glycosuria.

DISLOCATED or FLOATING KIDNEY (§ 177), which is a more frequent condition than is usually supposed, may be attended by a constant (chronic) pain, or give rise to severe attacks, hardly distinguishable from intestinal colic.

APPENDICITIS is also a cause of abdominal pain, which may be of sudden onset. Sir William Macewen told me of the case of a young man who was *suddenly* seized with severe abdominal pain in jumping out of a hansom cab. But appendicitis is rarely so acute, and is therefore treated of more fully in § 175.

In SPLENIC EMBOLISM the pain is generally sudden in onset, but is not usually very severe or lasting, and is referred to the splenic region. Its most common cause is acute or chronic endocarditis, evidences of which are present (§ 47).

In most obscure organic affections the pain comes on gradually, and is of a chronic character.

IX. In **Visceral Neuralgiæ** abdominal pain may come on suddenly and acutely, and may be for a long time the only symptom.

1. *Gastralgia*, or gastric neuralgia, is rare, but it is the most typical and best-known visceral neuralgia. The pain is severe, periodic, but usually relieved rather than aggravated by food or by pressure. The skin may, however, be very sensitive to the flick of a handkerchief (§ 206).

2. The gastric crises and neuralgia of the bladder or other viscera in association with *tabes dorsalis*.

3. *Neuralgiæ*, or " colic " of other viscera (*i.e.*, pain in the viscus without functional or organic derangement), have been described by various authors—*e.g.*, ovarian colic, vesical or splenic neuralgia, etc.—though these cases sometimes turn out to be connected with an undiscoverable organic disease, or with *tabes dorsalis*.

4. The neuralgia which accompanies or follows *herpes*.

5. *Angina Pectoris* is in some cases referred more to the abdomen than it is to the chest, but it is recognised by the circulatory disturbances, etc. (§ 41).

6. *Migraine* is certainly met with, alternating with abdominal pain.

§ **174.** By **Chronic Abdominal Pain** I mean that kind of abdominal pain which has come on somewhat gradually, and is running a chronic course. Chronic abdominal pain may be produced by a large number of causes which it would be impossible even to enumerate. It is only possible here to refer to those conditions which do NOT PRESENT signs or symptoms distinctly pointing to some affection of the STOMACH, LIVER, SPLEEN, or other ABDOMINAL VISCUS. Abdominal pain is the leading or only symptom in the following conditions :

I. Appendicitis	§ 175
II. Chronic intestinal obstruction (malignant stricture, simple stricture, pressure by a tumour, paralysis of the bowel, etc.) ..	§ 229
III. Chronic peritonitis	§ 176
IV. Movable kidney	§ 177
V. Intestinal dyspepsia ; VI. Enteroptosis ; VII. Obscure visceral and spinal disease ; VIII. Pancreatic disease	§§ 178-181

The history must be thoroughly investigated, and every organ thoroughly examined. Three features may afford us important clues :

1. The POSITION, character, degree, and constancy of the *pain*, and the presence of *tenderness* must be observed. (i.) If the pain and tenderness be *generalised*, one might suspect Tubercle or Cancer of the Peritoneum. (ii.) If they be situated chiefly in the *lower abdomen*, one might suspect Appendicitis or incipient disease of the Bladder or Uterus. (iii.) If the pain be chiefly in the *upper abdomen*, incipient Gastric or Liver disease. Thorough and REPEATED EXAMINATIONS of the *abdomen*, *rectum*, and *vagina* are nearly always necessary. The *urine* also should be repeatedly examined for gravel, etc., and the *fæces* (§ 215) for gall-stones. If there be general abdominal enlargement, turn to § 182 ; if a localised tumour, turn to § 187.

2. The AGE of the patient, and the history and duration of the illness, should be inquired into. In *children* perhaps the commonest of the obscure causes of chronic abdominal pain are intestinal worms and tuberculosis of the peritoneum ; in the *aged*, incipient cancer of some organ.

3. The STATE OF THE BOWELS, both previously and at the time of examination. In I., II., and III. above there is constipation, while in most of the other causes there is diarrhœa or irregularity of the bowels.

The **Abdominal Pain** *is constant, but liable to* EXACERBATIONS, *especially after exercise; there is* TENDERNESS *in the right iliac region; the* PULSE *is* RAPID, *and the* TEMPERATURE *elevated from time to time; the patient is young.* The disease is probably APPENDICITIS.

§ **175. Appendicitis** (typhlitis, perityphlitis) is a disease now known to be much more common than used to be suspected, yet it is still frequently overlooked. Appendicitis may consist simply of a *catarrhal* inflammation of the vermiform appendix, which may go on to *Ulceration, Peritonitis* (usually localised), or *Perforation.* It is sometimes associated with an impaction in the appendix of intestinal concretions, undigested food, or some other foreign substance, such as a fruit-stone or toothbrush bristle, any of which can, as Mr. Lockwood has shown, start a very acute inflammation.[1] Inflammation may extend to the cæcum (typhlitis) or the surrounding tissues (perityphlitis). Adhesions frequently form round the appendix, due to a localised peritonitis. If the appendicitis in such a case goes on to perforation, a *local peritonitis* or *local abscess* results. This abscess may burst into the general peritoneal cavity, but more often burrows extra-peritoneally in various directions. Thus it may form a perinephric abscess, or may open externally over Poupart's ligament. In other cases septicæmia or pyæmia may occur. Both intra- and extra-peritoneal abscesses may open into the bowel or bladder. Sudden perforation of the appendix, without previous formation of adhesions, or after the breaking down of adhesions, results in *Generalised Peritonitis.*

There are two clinical forms of appendicitis : Acute or recurrent, and chronic appendicitis. (*a*) In CHRONIC APPENDICITIS there may be no symptoms other than pain in the right iliac region increased after any over-exertion. Sometimes there is also alternating diarrhœa and constipation; there may or may not be local signs of swelling or tenderness.

(*b*) RECURRENT APPENDICITIS consists of recurring acute attacks. Here again the course of the disease is essentially a chronic one, with a constant liability to a recurrence of the inflammation. After this has subsided the patient may go on for many months in apparent health. Possibly he may never be troubled again, but in the vast majority of cases a fresh attack of inflammation occurs sooner or later, with localised, and sometimes generalised peritonitis.

Symptoms.—In a typical *attack of appendicitis* there are four symptoms, which, occurring in a young person, point to appendicitis—pain with tenderness, constipation, gastric symptoms, and slight pyrexia. (1) The chief symptom, as above mentioned, and sometimes the only one, is pain with tenderness, usually situated in the right iliac region. The *tenderness is generally fixed,* and is nearly always in this

[1] "The Pathology and Treatment of Appendicitis," by C. B. Lockwood (Macmillan, London, 1901).

situation ; but the *pain has a tendency to radiate*, and it may be referred to the umbilical, or even to the left inguinal region. It is a constant, dull, aching pain, liable to be brought on or *increased by exertion*. The pain generally comes on quite gradually, but sometimes suddenly. Special tenderness is present at "MacBurney's point"—*i.e.*, midway between the umbilicus and the right anterior superior iliac spine. (2) Deep-seated local swelling is sometimes absent ; but there is usually a feeling of resistance, with dulness to percussion, or an indefinite tumour, in the right iliac fossa. (3) Vomiting may be urgent at the onset of an attack ; when it continues for many days the prognosis is unfavourable. Constipation is usually present, so that the case is apt to be mistaken for intestinal obstruction. In other cases the attack is ushered in with diarrhœa. The urine is scanty, and the bladder irritable. (4) The pulse is quickened and the temperature rises soon after the onset of pain, and remains about 100° to 102° F. for a few days (Fig. 106, § 384). The pulse forms the best single indication as to the acuteness of the progress.

Course and Prognosis.—When an acute attack, as above described, sets in, there are three possible events—recovery, local abscess formation, or general peritonitis. (1) In a favourable case the temperature falls about the third day, the swelling disappears, pain and other symptoms subside, and the patient may be well in ten days. In other cases slight fever persists for a few weeks, and there is an indurated swelling left due to adhesions. The patient may go about for months or years with chronic appendicitis, and apart from vague pains, general malaise, and dyspeptic symptoms suffer no inconvenience. At any time, however, he is liable to have a recurrence of the acute symptoms. (2) When the general symptoms show no improvement by the third day, and the local swelling progressively increases, it is probable that an abscess is forming. (3) Perforation, with generalised peritonitis, may occur at any time. The general symptoms in such cases are much more severe, vomiting persists, and the abdomen is distended and motionless by the second or third day.

Treatment.—Rest in bed and light diet are essential. Hot fomentations locally are useful for the pain. Opium in small doses (short of causing drowsiness) is also admissible for the relief of pain after the diagnosis is established. It should not be given for long, as it not only confines the bowels, which is injurious, but masks the symptoms on which surgeons rely as indications for operation. Other hypnotics may be employed. An enema may be given on the third day if the condition be improving.

The *question of operation* requires careful consideration, and a surgeon should be early in touch with the case. Mr. Lockwood's rules (*loc. cit.*) are as follows : During the first few days of an acute appendicitis the patient must be carefully watched, and if symptoms of perforation with collapse or general peritonitis set in, surgical aid must be promptly sought. If there are signs of intestinal obstruc-

tion, but the patient can pass flatus, and the pulse remains under 100, watch the case, but be ready to operate at any moment. When flatus cannot be passed, and the pulse is over 100, or when there are signs of abscess formation, operation is called for at once. In chronic appendicitis the bowels must be opened regularly; if care be taken, the disease may remain quiescent. Yet the question of operation must be entertained even in these cases, as there is a constant danger of relapse and perforative peritonitis.

In addition to **chronic abdominal pain,** *there is a history of* CONSTIPATION, *gradually increasing to* COMPLETE STOPPAGE *of the bowels, with the gradual supervention of* VOMITING. The case is probably one of CHRONIC INTESTINAL OBSTRUCTION.

In **Chronic Intestinal Obstruction** (§ 229) the abdominal pain is more or less generalised and intermittent. The constipation may at first have alternated with diarrhœa, but after a time it is so complete that not even flatus can be passed. Vomiting, at first of food, and later fæculent matter, a rapid pulse, and other constitutional symptoms ensue if the condition is not relieved. The four commonest causes are Malignant Stricture, Simple Stricture, Pressure of a Tumour, and Paralysis of the Lower Bowel.

The **abdominal pain** *is* **chronic** *and* GENERALISED; *it is attended by* CONSTITUTIONAL SYMPTOMS, *and some* ABDOMINAL ENLARGEMENT *or other local signs.* The disease is probably CHRONIC PERITONITIS.

§ **176. Chronic Peritonitis** runs a slow and chronic course, and is usually attended by a certain amount of generalised pain. Some authorities describe a simple or idiopathic chronic peritonitis, but practically the disease is only met with in two forms: (*a*) That due to **tubercle,** and (*b*) that due to **cancer**—two conditions which, by the way, are met with at the opposite extremes of life, and which present a very marked contrast both in their clinical and anatomical features.

In **Chronic Tuberculous Peritonitis** (tuberculosis of the peritoneum, abdominal tuberculosis) the patient is nearly always a child. There is a deposit of tubercle in the peritoneal tissues, and the intestines become *matted together by adhesions.* Sometimes fistulous openings form between different portions of the bowel. (1) Pain and tenderness are present, but are not very marked features, except during one of the subacute or acute exacerbations which arise. (2) Tuberculous peritonitis is contrasted with cancerous peritonitis by its marked tendency to the formation of adhesions without fluid, while in cancerous peritonitis there is effusion of fluid without adhesions. As a consequence of the adhesions and the deposit of tubercle, knots or thickenings can be felt through the abdominal walls, which have a very characteristic doughy or "boggy" feeling. (3) Fluid may be present when the disease occurs in young adults, and in acute cases. As the disease advances there is considerable tympanitic distension. (4) Local tumours may be felt in some cases, the most characteristic being a sausage-shaped band produced by the rolled and infiltrated

omentum running transversely across the abdomen below the edge of the liver. Hard tuberculous masses can often be felt around the umbilicus. Other tumours are produced by matted mesenteric glands or cystic collections of fluid. (5) There are emaciation and hectic fever—*i.e.*, morning temperature normal and an evening rise of 2° or 3° F., as in all active tuberculous processes. (6) Tubercle is generally found in other parts also, especially in the lungs. (7) In one form of this disease in children a tuberculous abscess forms, which points at the umbilicus, and gives rise to a *persistent discharge from the navel* (*fæcal fistula*).

Prognosis and Treatment.—The prognosis is very serious, though not so bad as formerly. Much may be done by treatment on the same lines as for pulmonary tuberculosis, with the administration of sedatives if much pain be present. Laparotomy has been successful, especially in the early stage of the affection and in ascitic cases, but usually good feeding, plenty of fresh air, and inunction with blue ointment are all that is necessary. If the temperature is raised the patient should be kept in bed.

Chronic Cancerous Peritonitis (Cancer of the Peritoneum) is always attended by a great deal of pain, constant, but also in paroxysms. There is a great tendency to the rapid formation of fluid in the abdominal cavity, which is nearly always tinged with blood. It arises only in late middle or advanced life. Its recognition is easy in typical cases on account of the age, acute pain, and ascites (under which heading it is described, § 185).

The **pain** *is of a "dragging" character,* increased by exertion, *accompanied by* dyspeptic *and other* vague symptoms. The disease is possibly Dislocation of the Kidney.

§ **177. Movable Kidney** (also called Dropped, Dislocated, or Floating Kidney, according to the degree of mobility).—This condition is by no means uncommon, and does not usually give rise to symptoms unless the degree of mobility is considerable.[1]

The *Physical Signs* can only be discovered by palpation of the abdomen, with the patient lying down. The method of palpating the kidneys is given in § 292. With the patient in the erect or sitting posture, the kidney comes down more during inspiration than when lying down. After a little practice she will be able to lean forward and relax her muscles, which is an important aid to the observer. The left kidney rarely falls below the umbilicus, but the right one may be displaced into the iliac fossa, and even into the pelvis.

Symptoms.—In a few cases two kinds of pain may be experienced : (*a*) A constant dull, dragging pain in the back, or perhaps only an uneasiness in the loin, radiating down to the groin and inner side of the thigh, relieved by rest ; (*b*) attacks like renal colic, which may be followed by the passage of blood and albumen, and are due to the kinking of the ureter. Such are called "Dietl's crises." Sometimes hydronephrosis results. Neurasthenia is often associated, with mental depression or symptoms of dyspepsia, vertigo, diarrhœa, or constipation.

[1] Dr. Hector Mackenzie, the *Lancet*, vol. i., 1907, p. 1362, and vol. ii., 1907, p. 1140.

Etiology.—The fact that a very much larger percentage of women than of men have movable kidney is attributed by Suckling to the wearing of corsets. A fall or strain will also displace the organ, and that is why it is advisable for those with spare abdominal muscles to wear a belt when at work in the gymnasium. Attendants at refreshment bars, who have to draw beer or draw corks, are often found to suffer from movable kidney. It is said to be extremely common among those who suffer from migraine (owing possibly to the retching, which is a feature of that condition), and it occurs more often in tall than in short people. Rapid loss of fat, or lowering of the intra-abdominal pressure, such as occurs after delivery, are frequent causes.

Treatment.—Bromides and rest will relieve the patient for a time, and any concurrent dyspepsia must be remedied ; but the radical treatment consists either in the wearing of a proper form of belt, or an operation for stitching up and attaching the kidney in position. The abdominal belts usually supplied by instrument makers are not very successful, but Suckling has designed an apparatus[1] for applying additional pressure outside the belt, which he claims does away with the necessity of operation, and is permanently successful. Fattening of the patient is often a successful means of relieving the symptoms.

Among the **rarer causes** *of chronic abdominal pain may be mentioned* Visceral Neuralgiæ (§ 173), Intestinal Dyspepsia, Enteroptosis, Incipient Spinal *or* Visceral Disease, *and* Disease of the Pancreas.

§ **178. Intestinal Dyspepsia** and **Intestinal Catarrh** are conditions which it is sometimes a little difficult to separate from one another and from gastric dyspepsia, and some doubt whether they ought to be described as separate entities. The patient generally complains of obscure and erratic pains in different parts of the abdomen. He also complains of irregular attacks of diarrhœa and constipation, brought on by very slight dietetic errors or exercise. There is generally a good deal of flatus passed per rectum. The fæces are offensive, very often fermenting and decomposing, and *contain a good deal of undigested food.* This latter really constitutes the ground for regarding the disease as a separate one. When there is rectal or intestinal catarrh, there is a certain amount of mucus (see § 215) and specks of blood (not streaks, such as come from piles) in the fæces. The patient also generally complains of a feeling of exhaustion, with little or no exertion, and there may be prostration, nervousness, and other nervous symptoms. In very prolonged cases there is loss of flesh, and the appetite is capricious. The disease in itself is not very serious, though it is inconvenient and intractable. In view of the large number of organisms normally found in the intestinal contents, any continual damage of the intestinal wall, such as intestinal catarrh, is to be deprecated, since the bacilli can then more readily make their way through a damaged wall, and in this way give rise to what used to be called idiopathic peritonitis and other troubles.

Treatment.—The indications are : (1) To prevent the decomposition which goes on in the intestines ; (2) to allay the irritation of the bowels. Dietetic measures are generally called for, and the patient may derive benefit from diet consisting entirely of boiled milk for a time ; and the water which he takes should always be boiled. The bowels should never be allowed to be confined. If there is a tendency to constipation, an occasional dose of mercury and chalk, or castor oil (which can be given in capsules), or salines, is useful. Intestinal antiseptics are very beneficial, such as salol and salicylate of bismuth. Enemata or large antiseptic intestinal douches (3 to 5 pints) are given once a week in chronic cases with excellent results.

§ **179. Enteroptosis** (synonyms : visceroptosis, Glénard's disease, abdominal ptosis, dropping of the viscera) is a condition in which there is a general ptosis, or downward displacement or dropping of one or more of the movable abdominal viscera. Any of the viscera may be dropped in this way, owing to laxity of their ligaments or mesentery—liver, spleen, kidneys, and even intestines (Glénard .

[1] Supplied by Messrs. Salt, of Birmingham.

In many cases *no symptoms* are present. The most constant are: (1) Pain or a sense of weight or dragging in the abdomen, and sometimes in the back, accompanied by a feeling of sinking, or of emptiness or hollowness, is frequently present. From time to time the pain may assume a colicky character. It has been noticed in many cases that there is tenderness at a localised spot a little to the left of the middle line, just above the level of the umbilicus. (2) There is generally nausea, and from time to time vomiting. Symptoms of dyspepsia are usually present. (3) Sometimes there is diarrhœa, but more often constipation, and it is a special feature that aperients seem to cause considerable distress. (4) Great depression, nervousness, a general unfitness for all forms of exertion, and, indeed, all the symptoms of neurasthenia, may ensue, and the patients are apt to drift gradually into hypochondriasis. (5) The examination of the abdomen should be made while the patient is standing erect. The position of the viscera should then be marked, and afterwards an examination should be made with the patient in a recumbent posture. In marked cases the symptoms are considerably aggravated by the erect position, and they may be relieved by lying down, by pressing on the lower abdomen, or by wearing a supporting belt. Undoubtedly in many cases a displacement of the viscera can thus be made out, and when the patient is upright, a normal sized liver, or even a kidney, may be mistaken for a tumour.

The *Prognosis* as regards recovery is very uncertain. On the other hand, the condition is not fatal, and much can be done by judicious treatment if the physician secures his patient's confidence. The lives of these patients are often very miserable.

Treatment.—The indications are: (1) To relieve the nervous symptoms; (2) to relieve the dyspepsia; (3) to support the viscera. Much relief may be derived by wearing a well-fitting, adjustable abdominal belt. Flannel is the best material if the patient's skin tolerates it. The treatment of the neurasthenic symptoms and dyspeptic symptoms respectively is given elsewhere (Chapter X.). In severe cases which resist milder measures the idea of operative procedure should certainly be entertained. Cases have been recorded in which all the symptoms disappear after a simple abdominal incision, and the subsequent compulsory rest. In other cases definite organic lesions were found within the abdomen, which had not been detected during life, and which apparently caused the displacement.

§ 180. Incipient or Obscure Visceral or Spinal Disease.—(*a*) In cases of chronic pain GENERALISED OVER THE ABDOMEN one might suspect cancer of the intestines, of the pancreas, or of the kidney, cancer or tubercle of the suprarenals (*i.e.*, Addison's disease, in which pain over the stomach is a constant sign), or other incipient disorders, rheumatism of the abdominal muscles, enteroptosis, or movable kidney. Children may suffer from recurrent attacks of abdominal pain for which no cause can be found. Such cases, Dr. Guthrie says,[1] should be treated as incipient intussusception—that is to say, avoid purgatives and give digestible foods and small doses of opium.

(*b*) In various spinal affections the pain is frequently referred to the FRONT OF THE ABDOMEN, and among the more obscure causes may be mentioned abdominal aneurysm pressing on the spine, and cancer or caries of the vertebræ. The first of these occurs mostly in male adults, the second in the aged, and the third (Pott's disease) in children. In the latter the child frequently refers to the pain as "stomach-ache," worse after running about. The girdle pain of chronic and acute myelitis should also be borne in mind.

(*c*) If the patient complain of PAIN SITUATED CHIEFLY IN THE LOWER ABDOMEN, one might suspect appendicitis (*vide supra*), cancer or other disorders of the bladder, tubercle or cancer of the prostate or testes, peri- and para-metritis (in which there is a good deal of pain shooting down the legs), extra-uterine pregnancy, pyosalpinx, dysmenorrhœa and all its causes, uterine neuralgia, and obturator hernia. Hæmorrhoids are sometimes attended by pain in the abdomen (which disappears upon the cure of these), and so also are new growths and various ulcers of the lower bowel;

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[1] *Treatment*, May 12, 1898.

and, finally, among the unsuspected causes I have seen pelvic hydatid in a boy of ten.

(*d*) PAIN SITUATED CHIEFLY IN THE UPPER ABDOMEN may be due to various affections of the liver, stomach, and spleen. Among the painful affections of the *liver*, perhaps passive congestion, perihepatitis, and cancer are the commonest; hydatid is one of the obscure conditions, though it is rarely painful. Abscess above or below or within the liver should be suspected in those who have resided in tropical countries. Among the painful affections of the *stomach* may be mentioned gastric (or duodenal) ulcer, gastritis (acute or chronic), cancer of the stomach—which in its most usual form, scirrhus of the pylorus, is commonly very obscure in its early stages—and gastralgia. Painful affections of the *spleen* are not common, the chief being infarction, but the capsule is sometimes the seat of a painful inflammation.

§ **181. Diseases of the Pancreas** are fortunately rare, for they are always very obscure, and are often unrecognisable during the life of the patient.

As far as our present means of investigation go, the *Symptoms* to which they give rise are: (1) *Abdominal pain, deep-seated in the epigastrium*, radiating to the left shoulder, and round the left loin; (2) nausea, and vomiting of glairy mucus, anorexia, and acid eructations; (3) great debility, rapid emaciation, and mental depression; (4) undigested fat and muscle fibre in the fæces;[1] (5) glycosuria; (6) frequently jaundice; (7) a tendency to hæmorrhage; (8) the pancreatic reaction in the urine. Dr. P. S. Cammidge[2] has described a reaction in the urine, which he believes to be diagnostic of pancreatic disease. Certain crystals are obtained from the urine, which are said to differ in cases of pancreatitis from those obtained in cases of pancreatic carcinoma. (9) When salol is administered by the mouth for twenty-four hours, it does not appear in the urine as carbolic acid. (10) Sahli's test: the administration of glutoids of iodoform, hardened in formalin, is not followed by the appearance of iodoform in the urine. As to *Physical Signs*, a tumour may be felt only when the disease is very advanced.

The diseases of the pancreas which have been recognised, chiefly after death, are as follows:

I. HÆMORRHAGE WITHIN THE PANCREAS, a rare condition, which, if of any extent, causes death in twenty-four hours, or less (§ 171).

II. PANCREATIC CYSTS, due to obstruction or obliteration of the duct by biliary or pancreatic calculi, or cicatricial contraction. An injury to the abdomen is the chief cause. Fatty diarrhœa does not occur in these cases. The fluid withdrawn by aspiration will emulsify fat, and convert starch into sugar.[3]

III. PANCREATIC CALCULI are small concretions consisting chiefly of carbonate of lime.

IV. ACUTE PANCREATITIS is, according to Dr. R. H. Fitz,[4] met with in three forms: (1) *Acute Hæmorrhagic Pancreatitis*, which sets in suddenly with agonising pain, and results in death in one to four days (§ 171). (2) *Acute Suppurative Pancreatitis* begins suddenly with pain and irregular pyrexia, and may lead to death in three or four hours, but Fitz's cases more often became chronic, and lasted some months. There may be several small or one large abscess. (3) *Gangrenous Pancreatitis*, in which necrosis of the organ occurs, and it may be passed as a slough by the bowel. Two of Fitz's cases recovered.

V. CHRONIC PANCREATITIS is a fibrosis of the organ which mostly runs a latent

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[1] The presence of fat and muscle fibre in the fæces has not the same pathognomonic value formerly attributed to it. Their occurrence depends on the amount of gland tissue which is diseased.

[2] Robson and Cammidge, "Diseases of Pancreas," 1908.

[3] A case of retroperitoneal rupture of a pancreatic cyst occurring in a young man about twenty-five years of age was admitted in the Paddington Infirmary with all the symptoms of acute peritonitis. Laparotomy was performed by Sir Frederick Treves, but nothing was found until after death, forty-eight hours later. The origin of the cyst was not even then discovered, but the cellular tissue behind the peritoneum was infiltrated with the usual pultaceous material.

[4] Middleton-Goldsmith Lecture, *New York Medical Record*, 1889, vol. i.

course, but has received considerable attention of late years because it is frequently associated with diabetes, especially in those cases where atrophy of the gland ensues. It may also, by affecting the bile-duct, produce jaundice, with dilatation of the gall-bladder, and thus resemble gall-stones and cancer of the head of the pancreas.

Pancreatic Diabetes.—The association of glycosuria with pancreatic calculus was first pointed out by Cowley in 1788. But it was Lancereaux, in 1877, who maintained there was a special form of diabetes dependent on grave alterations in the pancreas (Pancreatic Diabetes), characterised by polyuria, excessive thirst and appetite, rapid loss of flesh, and glycosuria. Pancreatic diabetes may, however, occur with lesions of the pancreas other than chronic pancreatitis ; and grave alterations of the organ may exist without diabetes, the occurrence of which probably depends on destruction of the cells of Langhans (Opie).

VI. Cancer of the Pancreas may be primary or secondary, and is a rare condition. It is said to occur in about 6 per cent. of all cancers (Segre). The symptoms are : (1) Pain in the epigastrium, which at first occurs in paroxysms, then becomes constant, and runs a chronic course. (2) Symptoms of gastric disorder may be present for months before any other symptom. (3) Jaundice, intense and persistent from the pressure on the bile-duct, is usually present, and sometimes pain like biliary colic accompanies this. (4) The other symptoms are those above described. (5) Later on a tumour is found in the epigastrium or in the umbilical region, with little or no mobility, deep-seated, and hard to define. (6) Œdema of the legs, from pressure on the inferior vena cava, may occur.

The *Diagnosis* of cancer and other tumours of the pancreas is always difficult. A tumour of the liver, pylorus, or transverse colon, is more mobile. Much indican in the urine points to an intestinal rather than to a pancreatic tumour. No great stress can be laid on the presence of fat in the fæces, or on glycosuria, but abundant undigested muscle fibre found in the fæces is more characteristic of pancreatic disease.

Prognosis.—In cancer of the pancreas death usually occurs within four weeks after the onset of jaundice, or six weeks after ascites sets in. Emaciation and debility may not come on till late in the disease. The complications are : (i.) Symptoms due to pressure on the neighbouring organs—intestine, stomach, or portal vein ; (ii.) sudden hæmorrhage into the alimentary tract or the peritoneal cavity ; (iii.) pulmonary embolism. Sudden death occurs in the last two.

Treatment.—This is mainly symptomatic. The administration of pancreatin or similar preparations may aid the digestion. Opening and draining the gall-bladder has been successful in cases of pancreatitis accompanied by jaundice.

GENERALISED ABDOMINAL ENLARGEMENT

§ **182. Classification.**—Generalised abdominal enlargement occurs under four conditions :

I. Solid abdominal tumours	§ 188
II. Gas in the intestines (tympanites), or occasionally in the peritoneum	§ 183
III. Fluid in the peritoneum (ascites)	§ 185
IV. A cystic collection of fluid in the abdomen	§ 186

The **Routine Procedure,** as previously described (§ 167), should be by Inspection, Palpation, Percussion, Mensuration.

If a **hard tumour** can be felt in any part, turn first to § 188.

If the abdomen is quite **soft to palpation** and **resonant** all over, turn first to § 183.

If the abdomen is **dull to percussion** in the flanks, and presents the fluctuation test, turn first to § 185.

If the abdomen is **resonant in the flanks** and **dull in front,** turn first to § 186.

The abdomen is **uniformly enlarged** ; *it is* SOFT *and yielding to palpation ; and percussion, systematically conducted over the whole area, gives a* RESONANT *note.* The swelling is probably due to TYMPANITES.

§ **183. Tympanites** is the term employed for a flatulent distension of the stomach and intestines by gas. It should be remembered that flatulent distension may accompany and render obscure a *small quantity of fluid in the peritoneum.*

The *Causes* of tympanitic enlargement are as follows :

I. Atonic and other forms of DYSPEPSIA are the most frequent causes of flatulent abdominal distension. It is usually intermittent, and is generally greatest after meals (§ 204).

II. In ATONY OF THE COLON the bowels are constipated, and the patient, an adult, is liable to " colicky " pains ; but there are no constitutional symptoms or emaciation as in the next cause.

III. In TUBERCULOUS PERITONITIS there is a tendency to the formation of intestinal adhesions and *flatulent distension.* In tuberculous peritonitis, moreover, the distended abdomen has a doughy feel and here and there a patch of dulness on percussion, which is quite characteristic. It is accompanied by emaciation and an evening rise of temperature.

IV. " PHANTOM TUMOUR " may assume the shape of a generalised more or less resonant enlargement, but it more often resembles a localised tumour (§ 188).

V. In OBSTRUCTION OF THE BOWELS there is considerable abdominal distension, accompanied by pain, vomiting, and other general constitutional disturbance (§§ 228 and 229).

Gas in the Peritoneum gives much the same signs as tympanites, only there is extreme distension, and hyper-resonance all over to such a degree that the normal dulness of the liver and spleen is obscured. It is met with only when perforation of some part of the alimentary canal occurs. The patient is collapsed, and presents all the symptoms associated with perforation (§ 169). A few hours after the occurrence of the perforation a delusive lull occurs in the collapse and other symptoms, only to be succeeded by a fatal exacerbation. Perforation of gastric ulcer is the commonest cause, and one of the diagnostic features of this condition is the loss of the normal area of liver dulness.

There is **uniform abdominal enlargement,** *which is soft and yielding to palpation and* DULL TO PERCUSSION *in parts ; the* FLUCTUATION SIGN *is present.* There is FLUID WITHIN THE ABDOMEN.

§ **184.** When there is **Fluid in the Peritoneum,** either free or encysted, the belly is soft to palpation, dull to percussion in parts (either in the flanks or in front), and the measurements show the abdomen to be uniformly enlarged.

When the fluid is in any quantity, two special signs can be elicited. (1) *Fluctuation test.*—When a large amount of fluid is present, a wave of *fluctuation* may be *seen* to travel *across the surface* when we tap or "flip" one side. This can only be satisfactorily elicited when the abdomen is full and tense. (2) *Percussion test.*—A percussion wave can be transmitted from one hand to the other *through the fluid* by the law that fluids transmit pressure or a blow equally in all directions. Place the left hand over one side of the dull portion, and tap sharply with the fingers of the right hand over the opposite side ; an impulse will be felt by the left hand if fluid be present. Neither of these signs can be elicited in a gaseous enlargement or a solid tumour.

Fallacies of these Tests for Fluid.—In *obese persons* considerable difficulty arises in the detection of fluid, because the fluctuation test can in them be elicited whether fluid be present or not. In applying the "percussion test" for fluid in obese subjects, an assistant should place the edge of his hand vertically on the umbilicus. This will prevent the wave or impulse from travelling across the surface of the omental and subcutaneous fat instead of through the fluid.

But the fluid may be either (*a*) FREE in the peritoneal cavity, when it is termed ascites ; or (*b*) enclosed in a CYST, such, for instance, as an ovarian cyst.

(*a*) If FREE in the peritoneal cavity it will obey the law of all fluids, and *shift with the position of the patient.* Thus in ascites (§ 185) when the patient is on his back you will find both flanks are dull to percussion, and the umbilical region is resonant ; then, if the patient turns on one side you will find that the uppermost flank which was before dull is now resonant, and the umbilical region, if there is much fluid, is dull. Much may be learned from the character of the fluid withdrawn by a trocar. Ascitic fluid is straw-coloured, with much albumen. Hæmorrhagic fluid usually means cancer.

(*b*) If the fluid is ENCYSTED—*e.g.*, ovarian cyst, we can still elicit the fluctuation and the percussion tests just referred to, but the level of the dulness will not alter with the position of the patient (§ 186).

There is a **generalised uniform enlargement** *of the abdomen, which gives all the* SIGNS OF FLUID, *and the fluid* ALTERS ITS LEVEL *with the position of the patient.* The condition is ASCITES.

§ **185. Ascites** is a term applied to an effusion of non-inflammatory fluid within the peritoneum (dropsy of the peritoneum). The physical signs of fluid have just now been described above. It is sometimes difficult to detect a very small quantity of fluid in the peritoneum, but

its existence is rendered probable (i.) by the dulness on percussion of the umbilical region with the patient on his hands and knees, and (ii.) by finding that when the patient turns to on one side the flank which was dull is now resonant.

Ascites may have to be *Diagnosed* from any of the cystic conditions mentioned below (§ 186), but certainly the most frequent and important source of difficulty is *ovarian cyst*. In ascites (i.) the flanks bulge, (ii.) the front is flat and resonant, and (iii.) both flanks are dull, but if the patient turns on his side the upper flank becomes resonant—three features which are the exact reverse of those found with ovarian and other cystic tumours (see also Table on p. 271). Occasionally peritoneal adhesions (especially cancerous) may confine the fluid to one part of the abdomen, and then the fluid does not shift with the position of the patient. A greatly distended urinary bladder may simulate ascites, but the passage of a catheter readily excludes this fallacy.

The other *Symptoms* which accompany ascites belong to two categories : (1) Those due to pressure within the abdomen—*e.g.*, œdema of the feet and legs, from pressure on the vena cava and its branches ; later on dilatation of the surface veins of the anterior abdominal wall may occur from the same cause ; albuminuria from pressure on the renal veins, and dyspnœa from mechanical impediment in the circulation. (2) There are evidences of the condition which has caused the ascites, and of all the causes by far the commonest is alcoholic cirrhosis of the liver. The temperature is generally normal, except in chronic peritonitis.

The *Causes of Ascites* are five in number. In reference to the diagnosis of these causes, if there be any œdema of the ankles, it is important to ascertain whether this œdema or the ascites came first. For instance, when Portal Obstruction is in operation, the dropsy of the feet will have started subsequently to the ascites ; in Heart or Lung disease it will have preceded the ascites ; whereas in Renal Disease they would have started about the same time. Ascites with well-marked Jaundice in an old person is extremely likely to mean Cancer of the Liver or peritoneum. Ascites with sallowness of the skin in a middle-aged person is most probably Alcoholic Cirrhosis of the liver.

I. **Portal Obstruction** is the commonest cause of well-marked ascites. This is recognised in two ways : (*a*) By a history or presence of the *symptoms* of portal obstruction (of which ascites is only one) ; and (*b*) the presence or a history of one of the *causes* of portal obstruction.

(*a*) The Symptoms of portal obstruction, in the order in which they usually appear, are as follows : (1) A liability to attacks of gastric and intestinal catarrh, as evidenced by pain in the stomach, irritable dyspepsia, alternating diarrhœa and constipation, and the vomiting of mucus streaked with blood, especially in the early morning before

breakfast. (2) Hæmorrhoids. (3) Hæmorrhage, sometimes in very large quantity, from the stomach and the bowels. (4) Congestion, and therefore enlargement of the spleen. (5) ASCITES is one of the later results. (6) Enlargement of the veins of the abdominal wall from the establishment of a collateral circulation. (7) Œdema of the legs also appears subsequently to the ascites, and is due to pressure on the large veins in the abdominal cavity by the ascitic fluid. (8) Albumen in the urine may arise in the same way, or from concurrent disease of the kidney; in the former case the albuminuria may disappear after paracentesis.

(*b*) The CAUSES of portal obstruction may be grouped into (*α*) diseases within the liver, or (*β*) diseases outside it.

(*α*) *Diseases within the Liver.—Cirrhosis* of the liver is by far the commonest of all the causes, and this is nearly always due to alcoholism, there being a history of this and alcoholic dyspepsia. Simple ascites without marked jaundice or other obvious symptoms is presumptive of cirrhosis. *Cancer* produces portal obstruction usually by the pressure of the enlarged glands in the fissure, or by masses protruding outside the liver. *Perihepatitis* sometimes produces ascites by puckering of the capsule. Ascites only very rarely accompanies *hepatic congestion*, and never fatty liver, hydatid, or abscess.

(*β*) The causes of portal obstruction *outside the liver* are: (1) *Cancer* of the stomach, duodenum, or pancreas, and various other tumours pressing on the vein. (2) Enlargement of the *glands* in the fissure of the liver (cancerous, tuberculous, or syphilitic). (3) *Thrombosis* of the portal vein is rare, and the symptoms are very acute.

II. In **Heart Disease,** either primary (*e.g.*, mitral disease and cardiac dilatation) or secondary to lung mischief, the ascites is generally part of the dropsy affecting the cellular tissues and other serous cavities of the body. Here dropsy of the *feet will have preceded the abdominal dropsy*, and there will be previous history of palpitation, dyspnœa, and perhaps cough. An examination of the heart will also reveal the nature of the disease.

III. In **Kidney Disease** ascites may be part of a General Dropsy affecting the face, limbs, peritoneum, pleuræ, and pericardium. The fact that the dropsy started in all of these situations about the same time reveals this cause. Albuminuria is frequently enough a consequence of the pressure of the ascitic fluid, but the presence of epithelial casts almost certainly indicates that the renal disease was primary. It usually takes the form of acute or chronic parenchymatous nephritis, rarely waxy or granular kidney.

IV. **Chronic Peritonitis** is another cause of fluid in the peritoneum. An idiopathic form of chronic peritonitis is sometimes described, but it is practically never met with apart from a deposit of tubercle (in the YOUNG) or of cancer (in the AGED), § 176. In the tuberculous form

adhesions rather than fluid are met with; in the cancerous it is *vice versâ*.

V. A small amount of effusion into the peritoneum is found in severe **anæmia,** especially pernicious anæmia, and some other blood disorders; but it is never very great.

VI. Chylous ascites, or the collection of chyle in the peritoneal cavity, occurs as the result of obstruction of the thoracic duct. This is very rare, and due chiefly to the adult *Filaria sanguinis hominis*.

The *Prognosis and Treatment of Ascites* is very largely that of the morbid condition with which it is causally related, and the reader should turn to cardiac disease, renal disease, or chronic peritonitis according to the case. Again, the *Prognosis of Ascites due to portal obstruction* depends very much on the nature of the intra- or extra-hepatic lesion which has produced it, as given above and in Chapter XII. The degree of the obstruction is measured by the amount of ascites and other symptoms present, and still better by the amount and frequency of the hæmorrhage that has taken place from the stomach or intestines. Life may be prolonged for many years, even when a considerable amount of ascites has accrued, provided it has come on slowly, and time has thus been afforded for the gradual establishment of the collateral circulation through the surface veins of the abdomen and other collateral channels. It is in this sense that repeated tappings are good, for in this way time is gained for the establishment of collateral circulation. In cases of alcoholic cirrhosis the habit must be abandoned, otherwise the patient cannot live longer than six to twelve months, for ascites indicates an advanced condition of cirrhosis.

The *Treatment of Ascites*, like its prognosis, must depend upon its cause (*q.v.*). The treatment of *ascites due to portal obstruction*, and to some extent that of other forms, is as follows: (1) Hydragogue purgatives are certainly called for, and mag. sulph. and the other salines are the best. Elaterium seems particularly valuable if given in sufficient quantities to produce three or four watery stools a day. (2) Diuretics are recommended by some, but in my experience there is no form of dropsy in which they are of so little use as in ascites, at any rate until the pressure has been relieved by tapping. Copaiba resin and cubebs are sometimes useful, and I have in one or two cases given pil. digitalis co. with some benefit after repeated tapping. (3) Tonics are useful combined with the preceding, such as a mixture containing pot. bitart., fer. tart., and digitalis. (4) Paracentesis is generally called for sooner or later. Some physicians say it should be put off until it is called for by the urgency of dyspnœa. In cancer this is certainly a good rule, but in cirrhosis of the liver it is best to operate at once in all cases where there is much fluid, unrelieved by medicine. It is often found that medicines which were useless before are efficacious after the operation, because the kidneys are relieved from pressure. Sometimes

complete recovery takes place after repeated paracentesis, because time is thus afforded for the establishment of the collateral circulation as above mentioned. It is best to use a small trocar with the tube conducted to the pail, so that the peritoneum may gradually empty itself. With a large one leakage may remain, or peritonitis may ensue. In 1896 a surgical method of promoting the collateral circulation by the artificial production of peritoneal adhesions in cases of alcoholic cirrhosis was introduced by Dr. D. Drummond and Mr. J. R. Morison,[1] which has been attended by a measure of success.

There is a **generalised abdominal enlargement** *which gives all the* SIGNS OF FLUID (§ 184) ; *but the fluid does* NOT ALTER ITS LEVEL *with the position of the patient.* There is ENCYSTED FLUID (probably ovarian) IN THE ABDOMEN.

By far the commonest of such cystic tumours is an OVARIAN CYST. Other and less common cystic abdominal tumours are HYDRAMNIOS, CYSTIC FIBROMA of the uterus, HYDRO- and PYO-NEPHROSIS, PANCREATIC CYST, a large HYDATID, a cyst of the GALL-BLADDER, and an ENCYSTED ASCITES.

§ **186.** I. **Ovarian Cyst**[2] is centrally situated, and grows from below upwards. The enlargement is fairly uniform, and it gives all the signs of fluid (§ 184). But the level does not alter with the position of the patient ; and whereas the umbilical region is dull on percussion, the flanks are resonant. On palpation it is tense and elastic, and in malignant ovarian cysts nodules can be felt in the walls. The diagnostic features between ascites and ovarian cysts are given in Table XIV.

TABLE XIV.

	Ascites.	*Ovarian Cyst.*
Inspection.	Flanks bulge, front flat.	Flanks flat, front bulges.
Percussion.	Flanks dull, front resonant. On turning, upper flank becomes resonant.	Flanks resonant, front dull. No alteration of dulness on turning.
Measurement.	Umbilicus to xiphoid greater than umbilicus to pubes. Circumference at umbilicus greater than slightly below. Navel to iliac spine same both sides.	Umbilicus to xiphoid less than umbilicus to pubes. Circumference at umbilicus less than slightly below. Navel to iliac spine greater one side than another.

The features associated with it are (1) a history of it having grown upwards from the pelvis, and (2) these tumours (unlike ascites) may

[1] *Brit. Med. Journ.*, vol. ii., 1896. See also a paper by Rolleston and Turner, the *Lancet*, December 16, 1899.

[2] Parovarian cysts are rare. They present much the same symptoms as ovarian cysts.

be of very rapid growth, and reach quite a large size in three or four months. (3) There have usually been menstrual irregularities, though by no means always. There may have been no general symptoms of any kind, but generally some pain and local discomfort have been complained of. Often when the cyst contains pus there is little or no fever. When there is a history of attacks of pain, it generally indicates adhesions, an important matter from an operator's point of view. An examination of the uterus usually reveals nothing. A malignant cystic ovarian growth is indicated by (1) the presence of nodules in the walls; and (2) the age of the patient and a history of emaciation, and severe pain.

Diagnosis.—The characters of the fluid withdrawn by aspiration from the several abdominal cysts are given in a later chapter.

In the *earlier stages* the diagnosis of an ovarian tumour is sometimes difficult. It is an elastic, movable, and globular swelling; the uterus is not enlarged, and it can be defined as quite separate from the tumour. In this stage it may have to be diagnosed from *hydro-* or *pyo-salpinx*. *Para-* and *peri-metric* exudation and *pelvic hæmatocele* would be very firmly fixed in the pelvic cavity and accompanied by constitutional symptoms. In *extra-uterine fœtation* there would be morning sickness, a patulous os uteri, and other symptoms of pregnancy, with an empty uterus. These are dealt with in Chapter XIV.

In the *later stages* ovarian cysts have to be diagnosed from all the conditions mentioned below.

II. Pregnancy with hydramnios and a thin uterine wall is sometimes very difficult to diagnose from an ovarian cyst, for both develop very rapidly. Experienced clinicians have been known to fail in the differentiation. The symptoms of pregnancy (see § 328), the exactly central position of the tumour, and, as a last resort, the careful use of uterine sound, may aid us in the diagnosis. *Hydatid mole* presents similar difficulties, but it is fortunately more rare.

III. Large cystic fibroid of the uterus, especially of the subperitoneal variety, may produce the signs of a fluid tumour. It is recognised by (1) its connection with the uterus, which is enlarged; and (2) its slow growth, which may extend over many years; and (3) menorrhagia in some cases.

IV. A large hydatid cyst of the spleen or liver, a hydro- or pyo-nephrosis, a dilated gall-bladder, a large pancreatic or omental cyst, or a large peri-typhlitic abscess, may on rare occasions produce the appearance of a general fluid enlargement of the abdomen, and may require to be diagnosed from ovarian cyst; but they are nearly always *asymmetrical*. They grow from, and their percussion dulness is continuous with, the organs whence they rise; they are referred to among Abdominal Tumours (§ 187).

V. Encysted ascites is not common. It may result from previous peritonitis, of which there will probably be a history. More frequently, perhaps, it results from tubercle or cancer of the peritoneum (§ 176). In very rare cases congenital deficiency or adhesions may exist. In all of these there is a want of symmetry in the enlargement and in the fluid, an absence of the associated symptoms of ovarian tumour, and a history or other evidences of the cause in operation.

The *Prognosis* of ovarian tumour is always serious, though in the non-malignant form it may be quiescent for some years. If not treated, a cyst may go on (1) to rupture and fatal peritonitis; (2) it may become

inflamed ; (3) the pedicle may become twisted ; (4) hæmorrhage may take place into its cavity.

The *Treatment* is entirely surgical, and the earlier it is removed the better, and before the attacks of pain indicating inflammatory adhesions take place. Tapping is a temporary measure only, it certainly increases the risks for future operation, and is only justifiable for diagnostic purposes.

ABDOMINAL TUMOURS.

§ **187. Method of Procedure.**—We now turn to the second group of abdominal enlargements—namely, those in which the enlargement has originated in, or is localised to, one part—*i.e.*, Abdominal Tumours. It is only by repeated and careful examination that mistakes can be avoided in the diagnosis of abdominal tumours. The same methods are adopted here as in general enlargement (§ 167), which should be consulted. (1) *Inspection should never be omitted* in the recumbent, and sometimes in the erect, posture ; (2) *Percussion*, to define the boundaries and nature of the tumour, and its continuity with some organ ; (3) *Palpation*, with a flat hand previously warmed and with the abdominal muscles thoroughly relaxed by a suitable posture ; and (4) careful *Measurement* made and recorded both for comparison one part with another, and to see what progress the growth makes.

Fallacies of Abdominal Tumours.—(1) *Fat in the omentum* (obesity) may offer a serious obstacle to the examination of abdominal enlargements or tumours. The only way in these circumstances to arrive approximately at a correct decision is to place the hand flat upon the belly and then dip the fingers suddenly and forcibly inwards. The stomach and intestines should be empty.

(2) The presence of *fluid* within the abdomen, as well as a solid tumour, may prevent our discovering or examining it thoroughly. The difficulty may be obviated to some extent by suddenly flexing the fingers as in the case of obesity.

(3) *Fæcal accumulations* may simulate malignant and other tumours, though they can generally be indented by the fingers. They are always situated in some part of the large bowel. In doubtful circumstances a course of castor oil or other hydragogue purgative is desirable. But they may exist for many weeks in spite of purgatives.

(4) A " *phantom tumour* " is a swelling (usually tympanitic, sometimes dull), produced by irregular muscular contraction, and it is wonderful how precisely it may simulate a solid tumour. It is apt to appear and disappear suddenly, hence the name. The condition is met with for the most part in young hysterical women, and is quite beyond the control of the patient. It is a frequent cause of error in diagnosis. It is generally due to spasmodic contraction of one or both recti muscles. Spasms of the diaphragm may produce a generalised abdo-

minal enlargement by pushing the viscera down. The patient should be placed in a position of perfect ease for the relaxation of all the muscles of the body, with the knees drawn up and the neck slightly bent. Sometimes nothing but the administration of an anæsthetic to complete narcosis will enable us to establish the diagnosis, and this must be done in cases of importance.

Having excluded these fallacies, and being satisfied as to the existence of an abdominal tumour, there are five points to which our attention should be directed :

1. The first and most important question is the *locality of the tumour*, what region is it situated in, or where did it start ?

2. To ascertain with *which organ it is connected*, consider what organs are located in the region occupied by the tumour, and then see if it be structurally continuous by palpation and percussion with one of these.

3. If it *moves with the breathing* of the patient we know that it must be connected with the diaphragm, or some organ depressed by it during respiration, such as the spleen, liver, stomach, intestines, or omentum. If fixed, it is a tumour of the kidney (unless it be dislocated), aorta, lymphatic glands, or some other organ unaffected by respiration, or bound down by adhesions.

4. Inquire for a *history of any disease* or functional disturbance of the abdominal organs—*e.g.*, in case of the kidney, whether the urine contains, or has contained, blood or pus, although the tumour may appear to be far from these organs ; or perhaps there has been jaundice pointing to hepatic mischief.

5. The diagnosis of the *nature of the tumour* depends very largely upon its history and the age and sex of the patient. Tense cystic tumours are extremely difficult to differentiate from solid growths, but we can try to obtain the percussion and fluctuation tests (§ 184). In obscure cases the use of a long thin exploratory trocar is justifiable ; and then the presence of the opalescent fluid of hydatid, the blood-stained fluid of malignant disease, the straw-coloured fluid of encysted ascites, a fluid which will digest starch and emulsify fat (pancreatic cyst), or the absence of any kind of fluid, will materially help us to arrive at the precise nature of any given tumour. There is also another question which very frequently presents itself for consideration—viz., is the tumour benign or malignant ? The general symptoms of malignant disease (cancer) are discussed in § 415 ; but the age of the patient, and the rapid course and lethal tendencies of the disease, are the chief means of differentiating it.

§ **188.** *If there is a visible or palpable* **tumour** *in the abdomen, ascertain which* REGION *the tumour chiefly* OCCUPIES *or* ORIGINATED *in, and refer to that region in the following summary. Having identified* ITS ORIGIN

in this way, reference must be made to the diseases of the organ affected to ascertain the NATURE *of the tumour.*

I. RIGHT HYPOCHONDRIUM.—The commonest tumours in this position are tumours of the *liver*, especially cancer and enlargement of the organ. The features which HEPATIC TUMOURS present in common, in addition to their position, are: (1) They are not covered in front by resonant bowel, and their dulness is continuous with that of the liver; (2) they move with respiration; and (3) there are ascites, jaundice, and other evidences of liver derangement. It must not be forgotten that hepatic tumours may be simulated when the liver is pushed down by emphysema, or by pleuritic and pericardial effusions; or that it may be puckered by contraction of the capsule, and so simulate a tumour or enlargement (Diagnosis of Hepatic Enlargements, § 235); Riedel's lobe (see below) is another fallacy. Dilatation of the GALL-BLADDER (*e.g.*, by gall-stones) is recognisable as a tense rounded swelling below the ninth costal cartilage. There is only occasionally a history of biliary colic, but always a history of "chills" (biliary fever), see § 241. Tumours in this region may also be connected with the *duodenum* or *right kidney* (see II. and IV. below).

Riedel's Lobe[1] (lingiform or floating lobe of the liver).—In certain cases, usually associated with gall-stones retained within the gall-bladder, a tongue-shaped process projects downwards from the right lobe of the liver, or the lobus quadratus. It may reach as far as the iliac crest, or even to the iliac fossa. In hardly any of the cases in which it has been observed (Glénard[2] collected eighty), has the condition been correctly diagnosed until operation or an autopsy was performed. It has most often been mistaken for floating kidney, and has also been taken for distended gall-bladder, hydatid cyst, new growth, and omental tumour. It is sometimes tender, its shape more or less that of a pear. Under chloroform its connection with the liver might possibly be made out.

Suprarenal Tumours become manifest in the right or left hypochondrium, and are difficult to distinguish from tumours of the liver, gall-bladder, and spleen respectively. Mayo Robson[3] summarises the symptoms thus: (i.) Pain radiating from the tumour across the abdomen and to the back; (ii.) pain complained of at the shoulder tip; (iii.) emaciation, with nervous depression, and digestive disturbance; (iv.) a tumour felt beneath the costal margin (right or left), at first movable with respiration, but soon fixed; and it can be felt posteriorly in the costo-vertebral angle; (v.) absence of urinary and gall-bladder symptoms. More recently other symptoms have been described in relation to suprarenal tumours. In children precocious general and sexual development may occur,[4] or precocious obesity and hirsutes.[5] When the tumour is sarcomatous, there is a special tendency to secondary affection of the bones, particularly those of the skull.[6]

II. In the EPIGASTRIC REGION tumours may be connected with the liver (*vide supra*); but the first tumour which would occur to one's mind would be CANCER OF THE STOMACH—*i.e.*, a hard irregular swelling

[1] *Berliner Klinische Wochenschrift*, 1888, Nos. 29 and 30.
[2] "Les Ptoses Viscérales," Paris, 1899.
[3] *Brit. Med. Journ.*, 1899, vol. ii., p. 1100.
[4] Bulloch and Sequeira, Trans. Path. Soc., April, 1905.
[5] Guthrie, Trans. Clin. Soc., 1907, vol. xl., p. 175.
[6] Hutchison, *Quarterly Journal of Medicine*, 1907, vol. i., No. 1.

attended by vomiting, " coffee-ground " in character. The commonest form of malignant disease of the stomach, however, is scirrhus of the pylorus, and in this copious vomiting at long intervals and other gastric symptoms appear long before any swelling can be detected (§ 208). *Tumours of the duodenum* may sometimes be distinguished from those of the stomach by their immobility during a deep respiration.

Pancreatic cysts may cause a fluctuating swelling in the epigastrium, but their detection is extremely difficult. There may be a history of pain, and symptoms of pancreatic disease (see § 181). Cysts of the *small omental sac* present a similar swelling. *Pulsation in the epigastrium* is a symptom of that frequent condition, dilated right ventricle, and it must not be mistaken for abdominal aneurysm (*vide infra*).

III. In the Left Hypochondrium tumours of the spleen originate, and sometimes they attain to an enormous size. These are fully discussed in § 260. They move with respiration, and they make their way forward in *front* of the colon. The tumour can generally be moved forwards by getting the hand behind it, a procedure which distinguishes them from tumours of the left kidney. They resemble tumours of the left lobe of the liver, but these latter cannot be displaced downwards by the hand. Other tumours in this position may be connected with the *stomach*, *pancreas*, *liver*, *kidney*, and *sigmoid flexure*.

IV. The Lumbar Region may be the starting place for Renal Tumours, which are characterised by four features: (i.) Their fixity during respiration. (ii.) Dulness in one flank, and, unless both kidneys are involved, resonance in the other. (iii.) They are *always resonant in front*, because as they make their way forward they push the colon in front of them ; and (iv.) there is no resonant part between the dulness of a renal tumour and the spine, as there would be in the case of a splenic tumour. They generally retain their rounded and reniform shape. They are distinguished from hepatic tumours by the dulness in the flank not being continuous with that of the liver, and by the presence or history of blood, pus, or other urinary changes. The commoner forms of renal tumours are hydro- and pyo-nephrosis, renal sarcoma (commonest tumour in children), and perinephric abscess. *Pyo-* or *Hydro-nephrosis* are cystic tumours, containing urine *with* or *without* pus respectively (see § 315). Hydronephrosis may be almost painless, not tender, and unattended by any subjective or constitutional symptoms ; pyo-nephrosis is always tender, and attended by hectic fever and malaise. Other tumours starting in the lumbar regions may be connected with the *ascending* and *descending colon*.

Movable Kidney is one of the most frequent of abdominal tumours. It may be found in any part of the cavity below the liver. Its mobility, rounded or reniform shape are characteristic, but not always easily detected. There is a characteristic pain of a dull, aching, or dragging character in the back, increased by exertion (see § 177).

V. The Left Iliac Region may be the seat of a tumour caused by

Cancer of the Sigmoid Flexure, and this is the most frequent position in the bowel for cancerous growth. Cancer and other *tumours of the large intestines* are distinguished generally by their free mobility (unless fixed by adhesions). They are, when cancerous (far the commonest neoplasm of the intestines), usually multiple, and attended by more or less ascites, and by some irregularity of the bowels, generally chronic diarrhœa. The commonest starting-point for primary cancer of the bowel is, as just mentioned, the sigmoid flexure ; but before a cancerous swelling can be detected in the left iliac region the patient will have been troubled with recurrent diarrhœa and sciatica, sometimes melæna. These symptoms are followed in course of time by œdema of the leg. In the condition known as *colloid cancer* of the peritoneum all the intestines become matted together, and although fluctuation may be detected, there is little or no fluid in the peritoneal cavity. Cancer of the *small intestines* gives rise to hard, irregular, nodular, usually multiple tumours, and, in addition to the signs just mentioned, there are the advanced age of the patient and cachexia. Constipation, going on sometimes to obstruction, may also be present. The prognosis of cancer is given in Chapter XVI. But so-called " colloid cancer " of the peritoneum is a remarkable exception in regard to its duration, and it may go on for years before death occurs. The treatment, which is not very hopeful, is referred to under " Emaciation."

VI. The Right Iliac Region is the position in which Appendicitis is usually manifested ; it is fully described under " Abdominal Pain " (§ 175). *Intussusception* of the bowel, which occurs mostly in childhood, gives rise to a soft, sausage-shaped swelling generally situated in this region (§ 228). *Pelvic cellulitis* may form a firm swelling in either iliac region. Its other features are (i.) vaginal examination reveals a tender swelling in the corresponding fornix, pushing the uterus to the opposite side ; (ii.) there is a history of acute pain and fever at the onset of the condition, frequently following childbirth or abortion. *Cancer of the cæcum*,[1] contrary to what we might expect, often constitutes a *movable* tumour in the iliac region, and is very apt to be mistaken for masses of fæces. Epithelioma of the cæcum may be attended by suppuration, so giving rise to abscess in this region with pyrexia. The history of such cases may run a long course, and except in the age of the patient, resemble chronic appendicitis. *Psoas abscess* points in this region ; if originating from dorsal caries it presents below, if from lumbar caries above Poupart's ligament.

VII. The Umbilical Region is the starting place of tumours connected with the pancreas, duodenum, mesenteric glands, and aorta, all of which are *immobile during respiration ;* though a tumour in this position is far more often connected with the stomach, liver, or large

[1] Clin. Soc. Trans., November 24, 1899.

bowel, which *move with respiration*. Enlargement of the *mesenteric glands* may be sometimes detected in spare subjects by grasping the two sides of the abdomen either between the two hands or the finger and thumb of one hand. When large enough to form a tumour they are fixed and matted together.

Aneurysm of the Abdominal Aorta is a pulsatile and expansile swelling also immobile during respiration. In thin subjects a thrill may be felt, and a murmur heard. In auscultating the abdominal aorta we must be careful not to produce a murmur by pressure of the stethoscope. It is attended always by a severe fixed neuralgic pain in the spine, and sooner or later breathlessness and cardiac signs. It is these latter symptoms which distinguish true aneurysm from "pulsatile aorta" (see below), and from a swelling in front of the vessel to which the pulsation has been communicated. An endeavour should be made to grasp the swelling on each side, so as to observe the expansile nature of the tumour.

Pulsating Abdominal Aorta (throbbing in the belly).—Dyspeptic subjects and nervous females are often troubled with marked pulsation of the abdominal aorta, which is sometimes obvious both to the patient and the doctor. There is in this affection great local discomfort, and even pain, with marked pulsation, obvious to both inspection and palpation. The diagnosis from aneurysm rests partly on the fact that the pulsation is not limited to any part of the aorta, and partly that such rapid and violent action of the heart is not common in aneurysm.

VIII. The HYPOGASTRIC REGION is the situation whence BLADDER, UTERINE, and OVARIAN TUMOURS grow. *Ovarian tumours* (which are nearly always cystic) are usually characterised in the *early stages* by their free mobility, unless they are malignant, and their rapid growth (§ 186). *Tumours of the bladder* are usually rendered sufficiently obvious by changes in the urine. *Tumours of the uterus* are similarly revealed by uterine symptoms, excepting perhaps some subperitoneal fibroids. These may reach a large size without any symptoms at all; their origin and relations are readily detected by bimanual examination. *Pregnancy* causes a symmetrical enlargement, starting from the hypogastric region about the third month of gestation (§ 328). Among the rarer tumours in this region pelvic hydatid and pelvic hæmatocele may be mentioned.

The NATURE, PROGNOSIS, and TREATMENT of these various abdominal tumours are discussed under the organ with which they are connected.

§ **189. Flattening or Recession of the Abdomen** is not a sign of any great importance. "Ventre plat, enfant il y a," is a French expression signifying that the abdominal wall slightly recedes during the first two or three months of pregnancy. It is also met with in abstinence from food, and in wasting disorders, such as cancer and tubercle. It may be present also in intestinal, hepatic, and renal colic, and it may occur as a consequence of excessive purging or vomiting. A hollow or "boat-shaped" abdomen is said to be characteristic of meningitis in infants.

CHAPTER X

THE STOMACH

Two features cannot fail to strike the student in this department of medicine. The first is that we are very largely dependent upon subjective symptoms in the investigation of disorders of the stomach, a large proportion of the disorders of this organ being functional. Until the use of the "test-meal" (§ 199), and methods for estimating the motor-power of the stomach (§ 198), were adopted, we had to rely almost entirely upon the patient's sensations before and after meals to know how the stomach had been discharging its functions. The other feature relates to the important and widespread effects which derangements of the stomach produce in the general economy. The nutrition, of course, fails; but, apart from this, sufferers from gastric disorders are always liable to depression, which may sometimes be extreme. Prostration is apt to occur in all acute abdominal diseases; but in chronic disorders of the stomach the functions of the nervous system may be so profoundly disturbed by neurasthenic and other symptoms that the physician may overlook the primary cause of the mischief—namely, malassimilation of food.

PART A. SYMPTOMATOLOGY.

The symptoms which reveal disorders of the stomach may be **local** (viz., epigastric pain or discomfort, nausea or vomiting, hæmatemesis, dryness or bad taste in the mouth, flatulence, heartburn, water-brash, thirst, altered appetite); or **general** and **remote** (viz., cardiac symptoms, various nervous derangements, skin symptoms, and emaciation).

Among the **Local Symptoms** of gastric disorder, PAIN OR DISCOMFORT AFTER FOOD, and NAUSEA OR VOMITING, are perhaps the most constant and important—*i.e.*, the cardinal symptoms. HÆMATEMESIS is less frequent, but more serious. The other local symptoms are also of much value for diagnostic purposes.

§ **190. Gastric Pain,** or discomfort, in diseases of the stomach, is a most important *local* feature. Although it is not in every case suffi-

ciently constant in its characters to enable us to establish the diagnosis, nevertheless it merits the closest study. In some cases it is altogether absent (even when simple ulcer or malignant disease exists), but when present, the features which should be noted are its *position*, its *character*, its *degree*, its *constancy*, and above all, its *relation to the taking of food.*

Its *Position* is usually over the epigastrium, but pain is very frequently complained of between the shoulders, and very severe pain in the back may also occur. In its *character* it varies considerably. Sometimes it is like a dull weight or a feeling of distension, such as occurs in atonic dyspepsia and chronic gastritis ; or it may be of a burning character and such is the pain of acid dyspepsia ; or it may resemble abdominal cramp, as in spasm of the pylorus, or in some cases of gastralgia. Sharp or lancinating pain of a continuous character usually attends ulcer or cancer of the stomach.

Its *Relation to Food* is by far the most important feature of the pain in gastric diseases : (*a*) *It comes on at once* and lasts a variable time in atonic dyspepsia, in acute gastritis and in ulcer (simple or malignant). In simple ulcer the pain is at once relieved by vomiting—a very characteristic feature. (*b*) When pain *comes on an hour or more after food*, it is due to excessive acidity, either from hypersecretion or fermentation (organic acids). In hypersecretion, pain is relieved by taking food, but not in fermentation. (*c*) Pain *coming on without time relation to food* is characteristic of gastralgia. If pressure *over the seat of pain* relieves it, the condition is probably functional, not organic.

Fallacies.—Pain of the acute type met with in gastralgia may be mistaken for *biliary colic*, but in that condition the pain is greater on the right side, and is sometimes followed by jaundice. In *hepatic* disorders, pain is more often limited to the right hypochondrium. The spine should always be examined for *caries*, especially when stomach pain is complained of by children. The pain in such cases is referred to the terminations of the intercostal nerves. The gastric crises of *tabes dorsalis* may be mistaken for simple gastritis. Pain in the *chest* (§ 23) must not be mistaken for stomach pain. *True angina pectoris* might be mistaken for that type of dyspepsia where the stomach is distended with gas and hampers the heart's action. Darting or lancinating pain may be due to *growths* involving the nerves near the stomach. In acute *pancreatitis* there is extreme pain of sudden onset in the left hypochondrium, and the case usually terminates fatally in a few days. Other pancreatic diseases are also attended by pain.

§ **191. Nausea** or **Vomiting** is, after pain, the most frequent and most definite symptom of stomach disorders, though it arises, also, in many other conditions. Its causes may be grouped under three

headings : (*a*) Local Causes ; (*b*) Nervous Causes ; and (*c*) Toxic Causes. Water-brash (*vide infra*) is sometimes spoken of by the laity as " vomiting," but is not true vomiting. Regurgitation from a dilated œsophagus is another fallacy.[1] The mechanical discomfort of prolonged coughing may induce vomiting. Phthisical patients may come complaining only of the vomiting, and the physician may be led in consequence to treat the stomach instead of the lungs.

(*a*) Local Causes, producing vomiting, include : (1) *Errors of diet*, such as shell-fish, tinned food, excess of alcoholic and other irritating foods. Under these circumstances the vomiting of the peccant material occurs soon after ingestion. (2) *Irritant and corrosive poisons* and *emetics* also speedily give rise to vomiting. The diagnosis of this cause is aided by (i.) an examination of the vomit, which should *always be preserved ;* it may smell of phosphorus (which is luminous in the dark), or of carbolic, or other acids. (ii.) An examination of the mouth for any corrosive action. (iii.) The occurrence later of the toxic effects peculiar to the several poisons ; and (iv.) a history of poisoning obtained from the patient or his friends. (3) *Fermentation* of the contents of the stomach, such as that met with in dilatation, when the vomiting may occur at very considerable intervals, sometimes of a day or two ; the vomited matter also is frothy, and contains sarcinæ and yeast (Fig. 62, p. 292). (4) *Diseases* such as acute gastritis, cancer, and simple ulcer are usually accompanied by vomiting. In chronic gastritis the vomiting is of mucus, and occurs in the early morning.

(5) Persistent vomiting and marasmus in young infants are the two chief symptoms of **Congenital Hypertrophic Stenosis.** The symptoms begin about the end of the second week of life—(i.) forcible vomiting, which cannot be stopped ; (ii.) progressive emaciation ; (iii.) constipation ; and later (iv.) visible peristalsis of the stomach. (v.) A small hard nodule (the hypertrophied pylorus) may be made out under the upper part of the right rectus. Careful feeding and lavage will sometimes effect a cure, and operation (pyloroplasty, Loreta's operation, gastro-enterostomy) has been successful, but the mortality is very high.

(*b*) Vomiting of Nervous Origin may be classified under two groups—(*α*) that due to cerebro-spinal irritation, and (*β*) that due to visceral and sympathetic irritation.

(*α*) That due to Cerebro-spinal Irritation. 1. In *Hysterical Vomiting* the vomiting may follow any or every kind of food, no matter what its quantity or quality may be ; or perhaps digestible articles like milk will cause vomiting, while indigestible things like cheese may be retained. Sometimes this vomiting resembles a simple

[1] How closely regurgitation from the œsophagus, especially when it is dilated, may simulate vomit from the stomach is evidenced by three cases narrated by Dr. J. S. Bristowe (" Clin. Lects. and Essays on Dis. of the Nervous Syst.," pp. 42 *et seq.*). The chief differential features are the ease and promptness with which food is returned from the œsophagus in cases of dilatation and spasm, the *absence of an acid reaction* in the matters so returned, and the absence of signs or symptoms definitely referable to the stomach.

regurgitation, as compared with the urgent vomiting of organic disease, the symptoms of which are wanting.

2. In *Migraine* and *Bilious Headache* the patient perhaps awakens with a headache, and vomits only bile (merely an indication that the vomiting is urgent, or that the stomach is empty); the headache being relieved by the sickness (§ 605).

3. Another important cause of vomiting is *Cerebral Disease—e.g.*, tumour, early meningitis, abscess, Mènière's disease. This is recognised by: (i.) The vomiting occurs without relation to food; (ii.) there is no nausea; (iii.) the vomiting may be excited by simple change of posture; (iv.) the presence of other cerebral symptoms, such as vertigo and perhaps optic neuritis (Chapter XIX.). Vomiting may also attend the gastric "crises" of *locomotor ataxy*. It occurs at intervals, and is usually severe. It is recognised by the absence of the knee-jerk and the presence of other symptoms of the disease.

(*β*) Reflex vomiting due to Sympathetic or Visceral Irritation may be met with in a great many abdominal disorders, such as peritonitis, pancreatitis, intestinal, biliary, or renal colic; in all stages of intestinal obstruction, and in strangulated hernia. It occurs also with pregnancy, uterine and ovarian disorders. If at the end of an operation the surgeon puts in stitches while the patient is coming out of the anæsthetic, vomiting is at once excited every time the needle is put in. This is especially noticeable with children.

(*c*) Toxic Causes are uræmia, Bright's disease, and jaundice. Some of the acute specific fevers are accompanied by vomiting, especially at their advent. The vomiting of Addison's disease and pernicious anæmia might perhaps come under this heading.

The *Treatment* of vomiting must be directed to its cause, but there are certain measures which can be applied to relieve the symptom. The patient should be kept absolutely at rest in the horizontal position, and without food, or only given milk in small quantities at a time, and iced water. Milk diluted with barley-water, whey, or peptonised milk are given where ordinary milk is not retained. Among the remedies which may be employed are effervescing mixtures, alkalies, hydrocyanic acid, bismuth, drop doses of vinum ipecacuanha, opium, and acetanilid (especially in the vomiting after anæsthetics), seidlitz powder (if the vomiting be due to constipation) or calomel. Bromides and hydrocyanic acid are useful for nervous vomiting; a mustard-leaf applied to the epigastrium may also be useful. For *Sea-sickness*, chlorobrom, bromides, chloral, morphia, and recently chloretone are recommended.

Cyclical or Recurrent Vomiting is an occasional condition occurring in children. Usually two or three attacks occur in the course of a year, lasting from a few days to two weeks, and coming out without assignable cause, and even with the most careful dieting. An attack

comes on suddenly with drowsiness, constipation, and perhaps pyrexia ; all food is vomited ; the breath smells sweet from the presence of acetone, and acetone and diacetic acid are found in the urine (*vide* § 287). Drowsiness and restlessness increase, and cases have been mistaken for meningitis and intestinal obstruction. The condition is probably toxæmic in origin. Copious enemata, with normal saline, and by the mouth frequent small doses of alkaline, carbonates and citrates may be tried. It is important to open the bowels freely.

§ **192. Hæmatemesis** (Vomiting of Blood).—Bleeding from the stomach, unless in slight quantity, is usually accompanied by nausea and vomiting. In the first place, it is important to decide whether the blood really comes from the stomach.

Sources of Fallacy.—(1) Blood from the *lungs* may be mistaken for blood from the stomach (see Hæmoptysis, § 73). (2) *Epistaxis*, the blood running down the gullet and being vomited, is a common fallacy in children, in whom the blood is apt to be swallowed. It is recognised by making the patient blow his nose. In epistaxis there are no abdominal symptoms. (3) Blood from the *fauces* or *gums*, especially when the gums are spongy, or when pyorrhœa alveolaris exists, may give rise to a sanguineous vomiting or expectoration, the cause of which is very apt to be overlooked, if unsuspected, even by competent observers[1] (§ 145) ; but the blood is mixed with saliva, and is rarely very large in amount. On the other hand, *hæmorrhage from the stomach* is (i.) preceded by a feeling of faintness and nausea, and (ii.) followed by melæna (tarry stools). (iii.) Blood from the stomach is mixed with food, and mostly brown, though it may be red if the quantity is large (*e.g.*, in ulcer) or if food has been brought up before the blood. (iv.) There is an absence of previous history or local signs of pulmonary disease, and there may be a previous history of disease or derangement of the stomach or liver.

The *Causes of Hæmatemesis* may be roughly divided into (*a*) those which produce a slight or protracted hæmorrhage, and (*b*) those which give rise to a large quantity at one time.

(*a*) **Slight or Protracted Hæmorrhages** occur chiefly in Chronic Gastritis and Cancer. A temporary irritation or congestion of the stomach produced by irritating articles in the food or by urgent vomiting, may be attended by *streaks* of blood in the vomit.

I. Chronic Gastritis, or gastric catarrh, is known by (i.) vomiting in the morning—often viscid mucus streaked with blood—or at other times. (ii.) It may be accompanied by, and due to, disease of the liver (portal obstruction), or advanced cardiac disease, and is found especially in alcoholic subjects. Its other causes are mentioned in § 209.

[1] Cases of this kind, mistaken at first for organic disease of the stomach and the lungs, have been recorded (see Report Roy. Med. Chir. Soc., *Lancet*, June 16, 1900).

II. Cancer of the Stomach is recognised by : (1) The patient is usually beyond middle age ; (2) pain is complained of—severe, constant, and generally worse after food ; (3) the blood vomited is rarely copious, but typically "*coffee-ground*" in character (due to the digestion of the blood, which is constantly oozing into the stomach), and may continue for weeks ; (4) the hæmatemesis is very rarely followed by melæna, because the blood is scanty, and because there is often obstruction of the pylorus ; (5) progressive cachexia is marked, and an abdominal tumour, or evidence of cancer elsewhere, *may* be found (see also § 208).

(*b*) A **Large Hæmorrhage** at one time may occur in Simple Ulcer of the Stomach or Duodenum, Liver Diseases, other causes of Portal Obstruction, Aneurysm of the Aorta, Vicarious Menstruation, Gastrostaxis, Morbid States of the Blood, or Chemical Irritants.

III. Simple Ulcer of the Stomach.—This is known by : (1) The hæmatemesis is copious ; therefore the blood is bright red, after first being a little black, and melæna usually follows ; (2) characteristic pain occurs directly after food, and is *relieved* by vomiting ; (3) it is found chiefly in young women, (4) who are the subjects of anæmia, but not often great emaciation ; (5) a history of previous attacks of bleeding is often present (§ 207).[1]

III*a*. Ulcer of the Duodenum is often difficult to distinguish from gastric ulcer. It occurs mostly in men.

IV. Liver Disease (by causing portal obstruction), especially cirrhosis (§ 250). The hæmatemesis may be slight, but it is more often very copious—the most copious met with.

V. Other Causes of Portal Obstruction (see § 233)—*e.g.*, tumour pressing on the portal vein. This, as with cirrhosis, is known by the other symptoms of such disease—*e.g.*, (1) the accompanying and rapidly increasing ascites, and (2) diarrhœa.

VI. Aneurysm of the Aorta, or of one of its branches, leaking into the bowel, or œsophagus.—This is known by (1) possibly a previous history of aneurysmal symptoms (§ 53) ; (2) the blood is copious ; (3) sudden death is the usual result. This is the usual course, but in certain other cases there is a small recurrent leakage from the aneurysm for a few days or weeks preceding death.

VII. Vicarious Menstruation.—It is impossible to be certain in the diagnosis of this condition. Its leading features are that it occurs periodically, and in women with amenorrhœa.

VIII. Gastrostaxis.—Under this title are included cases of severe hæmatemesis, occurring usually in young anæmic women, due to capillary oozing. Such cases were formerly thought to be due to

[1] Cases have been recorded of profuse hæmatemesis resembling that of simple ulcer, occurring in older patients, which disappeared under antisyphilitic treatment. The condition was apparently a syphilitic ulcer of the stomach.—Dalgleish, *Lancet*, 1898, vol. ii., p. 410.

gastric ulceration, but more frequent operation and post-mortem examinations have shown that no ulcer is present.

IX. Morbid Conditions of the Blood, such as yellow fever, malignant forms of the specific fevers, purpura, leukæmia, and hæmophilia.

X. Chemical Irritants (*e.g.*, mineral acids), or mechanical injuries from articles which have been swallowed.

In the *Differentiation* of the causes of hæmatemesis (1) examine the stomach ; (2) examine the liver, especially for Cirrhosis, which is perhaps the commonest cause of hæmatemesis, simple or malignant ulcer being the next ; (3) ascertain the approximate quantity of vomited blood, and then review the case.

Prognosis.—Hæmatemesis is usually a serious symptom, but its gravity depends upon the cause. In portal congestion, hæmatemesis not infrequently serves as a safety-valve to relieve the abdominal congestion, and in a sense is beneficial. As regards the lesion, aneurysm is the most grave of the causal conditions ; then, in order, cancer, morbid blood states, cirrhosis, and simple ulcer. The amount of hæmorrhage is a less valuable guide to prognosis, although where the amount is copious the patient will remain debilitated for a considerable time.

The *Treatment* of hæmatemesis must also have reference to the cause. (*a*) When small in quantity, it calls for but little immediate treatment. (*b*) When in larger amount, the patient must not be moved from the place where the bleeding occurred, and should be kept absolutely at rest in the horizontal positon. Nothing should be given by the mouth except iced water for some time ; feed by rectum. Morphia hypodermically is the best hæmostatic, and also relieves the pain. If bleeding continues, give astringents by the mouth, such as alum (gr. v.) and dilute sulphuric acid (℥ xx.), or ergot. Adrenalin (1 in 1,000), ½-drachm doses every two or three hours, is a recent and valuable remedy. In profuse hæmorrhages saline transfusion may be necessary. If the bleeding is due to vicarious menstruation, try to restore the natural discharge by giving iron and aloes, and by hot sitz- and foot-baths.

§ **193.** The **other Local Symptoms** of gastric disorder are of considerable diagnostic value.

1. Bad Taste in the Mouth is very often complained of in gastric disorders, and is always most noticeable in the morning. Dryness of the Lips is another very constant manifestation, and will often give an acute observer the first clue to the existence of gastric disorder.

2. Thirst is often associated with dyspepsia ; it is specially apt to occur with dilatation of the stomach, inflammatory stomach lesions, and in all cases where there is persistent vomiting.

3. Flatulence is a distension of the stomach or intestines by gas, which may be brought up by the mouth or passed by rectum. This gas may be due to repeated swallowing of saliva and air, as in chronic gastritis,[1] or to decomposition of food. Among its causes are excessive ingestion of vegetables, sugars, and starches, chronic dyspepsia, or chronic gastritis, and all conditions attended with dilatation of the stomach.

4. "Heartburn" and Acid Eructations are usually met with together. Heartburn is a burning sensation passing up from the epigastrium to the pharynx, and sometimes mouthfuls of acid fluid are brought up at the same time. It is due to hyperacidity and partial regurgitation of the gastric contents into the lower end of the œsophagus.

Causes.—Hyperacidity, or "acid risings," may be of two kinds. (*a*) *Organic acids* are met with in diseases where there is *deficient* gastric secretion—some forms of atonic dyspepsia, chronic gastritis, cancer, and dilatation of the stomach. HCl is a germicide, and when from any cause it is absent, bacteria flourish; fermentation ensues within *a few hours* after food, and is accompanied by pain in the epigastrium. The three principal types of acid fermentation are: butyric, lactic, and acetic.

(*b*) Hyperchlorhydria, or *excessive secretion of HCl.* This condition is met with in one form of acute dyspepsia, and chronic glandular gastritis. The name is often misapplied to the acidity due to organic acids arising from fermentation. Here, the pain or "gnawing" generally occurs *before* meals, and is temporarily relieved by food (see also § 205).

5. "Water-brash" (Pyrosis) is the name given to a clear alkaline fluid expelled from the mouth in gushes, most often in the morning. Sometimes it is expelled without any kind of straining, but more often it is attended by retching. It is probably a reflex hypersecretion of saliva due to irritation in the stomach, swallowed during the night. It is met with in many dyspeptic conditions, and is a fairly constant symptom in *chronic gastritis.*

6. Anorexia (Loss of Appetite) is not always an indication of stomach disease, as it is present in many general constitutional disturbances. Its chief clinical importance lies in its presence in the earliest stage of *gastric cancer.* In *atonic dyspepsia* there is sometimes no appeite before a meal; but the first few mouthfuls of food induce secretion of gastric juice, and so excite appetite. Hysterical Anorexia is known by: (i.) The appetite is perverted; for instance, the patient will only eat some unreasonable article—*e.g.*, a penny bun

[1] The semi-voluntary swallowing or gulping down of air is met with in lunatics, and in some hysterical or neurotic individuals without gastric derangement. It is diagnosed from dyspepsia by the absence of all other symptoms of that condition.

bought at a particular shop.[1] Such patients may push matters to extremes, almost to the point of death. The condition is really a form of hysterical insanity. (ii.) It is only met with in the female sex, in whom also (iii.) the hysterical stigmata are generally present (Chapter XIX.).

Increased Appetite is far more often met with, as Shakespere pointed out, in gastric disorders. It is found in some cases of chronic dyspepsia, chronic gastritis, and dilated stomach, in pregnancy, and during convalescence. A false appetite which is satisfied with the first few mouthfuls of food is sometimes met with in subacute and chronic gastritis, owing to the irritated condition of the mucous membrane. Boulimia or ravenous appetite is seen in diabetes, in neuroses of the stomach, after acute gastritis, in wasting disorders such as mesenteric gland disease, in phthisis, intestinal worms, and Graves' disease.

§ **194. General** or **Remote Symptoms** are very constant accompaniments of all gastric diseases.

1. General Malaise and a sense of ill-health and incapacity for work are among the earliest and most constant accompaniments of all derangements of the digestion, whether functional or organic. The dark rim beneath the eyes, and the sallow, "earthy" complexion, so frequently associated with town-dwellers, is quite as often due to dyspepsia, just as this latter is often due to defective teeth or to the insufficient use of them. Emaciation is not so frequently associated with gastric disorder as might be supposed, though in very chronic cases there is sure to be some loss of flesh. Early and marked emaciation is, however, one of the surest indications of cancer of stomach.

2. The Cardiac Symptoms met with in dyspepsia are palpitation, pain in the region of the heart (pseudo-angina); dyspnœa and asthmatic paroxysms;[2] syncope and vertigo; intermission of the cardiac rhythm; and cough, due to pharyngeal catarrh or reflex irritation. Collectively, these symptoms may, as previously mentioned, give rise to the impression that the case is one of cardiac valvular disease, although the heart may be structurally healthy.

3. Functional Disturbance of Nervous System.—*Headache and depression of spirits* are invariably met with in all forms of dyspepsia. A sense of general ill-health and irritability of temper out of all proportion to the local mischief attends most gastric disorders, and, where stomach symptoms are not prominent, may lead the physician away from the true cause.[3] Any or all of the symptoms of *neuras-*

[1] An instance of this kind has happened in my experience, and the patient, thin as a skeleton, was really on the point of death when I first saw her.

[2] True asthma is sometimes occasioned by an attack of indigestion.

[3] Gautier and others have shown that certain toxins are being constantly generated within the body, and especially in the digestion and metabolism of the food. They produce no evil effect in health when not in excess, partly because

thenia (Chapter XIX.) may undoubtedly result from gastric disorder, and this constitues one variety of what the author has described as Toxic Neurasthenia.[1]

4. DIARRHŒA may accompany stomach disease when the gastric contents are of an irritating nature; CONSTIPATION is usually found with simple ulcer, cancer, and chronic gastritis. But a more usual condition is an IRREGULARITY of the bowels, accompanied by borborygmi (rumbling in the bowels).

5. The URINE invariably exhibits signs which reveal the disturbances in the metabolism of the body. The commonest of these, perhaps, is an excess of URATES, as shown by the pinkish sediment when the urine cools. In other cases PHOSPHATES form the deposit; and in certain cases OXALATES are found (compare § 314). In these circumstances dyspepsia must be regarded as a predisposing cause of renal and vesical calculus.

6. SKIN SYMPTOMS.—General *pruritus* may accompany many forms of gastric derangement. *Flushing* of the face after meals is met with in many gastric disorders, especially when they occur in the female sex. The face may be swollen so that the case appears like one of acute Bright's disease; but the sudden onset, without much constitutional disturbance, and early disappearance on curing the indigestion, distinguish it from that disease. The occurrence of general *urticaria* in certain individuals after eating indigestible articles is very common. It may also attend the different forms of gastric disorder.

PART B. PHYSICAL EXAMINATION.

Disorders of the stomach are investigated by Inspection, Palpation, Percussion, and Examination of matters vomited, or withdrawn from the stomach by a tube.

§ **195. Inspection.** (1) The *Teeth* in all cases must be closely examined. Among my out-patients the two commonest causes of indigestion are certainly defective teeth and bolting the food. Disorders of the teeth are referred to in § 143.

(2) The *Tongue* and its diseases have been already described, and § 147 should be specially consulted. At one time the tongue was thought to indicate the state of the stomach, but this is by no means

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they are excreted by the urine, fæces, and sweat, partly because they are being constantly destroyed (chiefly, perhaps, by the liver). But when in excess they produce profound disturbance of the general economy, and especially of the nervous system. Their composition, as far as we know, is analogous to the vegetable alkaloids (morphine, atropine, etc.), and they are therefore known as *animal alkaloids*. There are two kinds—(1) *Ptomaines*, which are produced by the fermentative disintegration of dead albuminous substances (*e.g.*, during digestion); and (2) *Leucomaines*, which are produced by the activity of *living* nitrogenous substances (see also Sir Lauder Brunton in the *Practitioner* for October and November, 1880).

[1] "Clinical Lectures on Neurasthenia," Glaisher, London.

always the case, and it is a far more certain indication of the patient's general condition. But even in this, allowance has to be made for certain variations—namely : (i.) Individual variations, since a coated tongue is normal to some, even in health, and a clean tongue in others may be associated with disease ; (ii.) certain diets—*e.g.*, milk—produce a coated tongue ; and (iii.) certain habits—*e.g.*, smoking and " tippling "—also coat the tongue.

(3) Inspection of the epigastric region may reveal a tumour, or the peristaltic movements of a dilated stomach. Aortic pulsation may be transmitted by a pyloric tumour, although no bulging is visible.

§ **196. Palpation.**—To palpate the stomach successfully requires considerable experience. The patient's shoulders should be supported, and he should be instructed to open his mouth, to draw up his knees, and to " let his breath go."[1] Talking to him is useful to distract his attention. The hand should be warmed, and it should always be laid quite *flat* upon the abdominal wall. Then only can we detect the presence of a tumour, tenderness, or other abnormality.

Gastric Succussion or *Splashing* is made out by placing one hand on each side of the stomach, and suddenly pressing inwards the finger-tips of each hand alternately. Listening over the stomach with a binaural stethoscope during this procedure materially aids in discovering this sign. Splashing can be *normally* elicited during the process of digestion—*i.e.*, during the first hour or two after a meal, especially if much fluid has been taken. But if succussion can be elicited after that time, it suggests that there is atony of the stomach, either with or without dilatation.

§ **197. Percussion** of the stomach is not very satisfactory or precise. The only diseases in which the area of stomach resonance has to be defined are DILATATION (§ 210), and GASTROPTOSIS (§ 211).

PERCUSSION OF THE STOMACH.—The normal stomach is depicted on p. 363, and its situation in Fig. 61, the cardiac orifice being under the seventh costal cartilage, about an inch to the left of the sternum. The pylorus is just to the right of the sternum, and 2 inches below it. The lesser curvature corresponds closely to a line drawn round the tip of the xiphoid cartilage and along its left side. The fundus of the stomach is its highest point, and is just behind the heart apex at the fifth rib. The position of the greater curvature (lower border) of the stomach varies according to the degree of distension ; it ought not to come lower than midway between the umbilicus and xiphoid. Its position is hard to define, owing to the proximity of the transverse colon. Only the lower border and part of the anterior wall of the stomach are normally in contact with the abdominal parietes. The percussion note over the stomach is tympanitic, but has rather a lower pitched tone than that over the transverse colon, which is, of course, also resonant.

The rough outline of the stomach resonance can be made out by percussion in the usual way without any elaborate precautions, but the following method is

[1] Some say it is better to have the legs extended loosely, and some advise examination in a hot bath to relax the muscles. Finally, anæsthesia with chloroform or ether may be necessary in very obscure cases.

more accurate : The stomach being empty, let the abdomen be stripped, with the patient standing. Percuss lightly from above downwards, and mark the change of note, the stomach being usually the most resonant of the abdominal viscera. After this give the patient a large drink of water. The note over the lower border of the stomach is now dull, and the lower level of the dull note so produced can be marked. Finally, the area of the stomach can be percussed out, with the patient in the recumbent posture, when the lower boundary will be found at a different level.

Ausculto-percussion is sometimes employed to define more accurately the boundaries of the stomach. Place the stethoscope in the angle between the xiphoid cartilage and the left costal margin, and elicit the normal stomach note by percussing lightly around it. Then percuss from the periphery towards the stethoscope till a note is heard similar to that first elicited. When there is fluid in the stomach, the percussion note varies with the position of the fluid, and then it is necessary to percuss the boundaries, first with the patient lying on his back, then on his right, and finally on his left side.

Fallacies.—(1) Apparent *enlargement* of the stomach may arise in contracted cirrhotic liver, or fibrosis of the lung. (2) Apparent *diminution* may occur when the liver is enlarged or pleuritic effusion is present. (3) *Dislocation* of the stomach downwards may simulate dilatation. In such cases the lesser curvature may sometimes be seen or felt below its normal position.

§ 198. Motor Insufficiency of the Stomach (Gastric Atony or Myasthenia) leads to Dilatation (§ 210). A man can live and maintain weight without the secretory and resorptive functions of the stomach being quite perfect ; but serious auto-toxic effects and malnutrition result from a *retention of food* within the stomach. Moreover, motor insufficiency is always attended sooner or later by disturbance of the secretory and resorptive powers.

After a normal meal, consisting of 50 grammes of bread, 200 grammes of beefsteak, and a glass of water, no solid portions should be found in the stomach in six or seven hours. There is, however, considerable individual variation. Recently X rays have been found to give reliable information of the motor functions of both the stomach and intestine. Two ounces of bismuth carbonate are given with breakfast, mixed with arrowroot, milk, or minced meat. This produces a definite shadow when examined with the fluorescent screen, and so its passage down the alimentary canal can be observed. Thus motor weakness, dilatation, or obstruction may be detected.[1] Carbohydrates have thus been shown to leave the stomach in three hours, whilst proteids and fats are still present in small amount after six. The average time for the bismuth breakfast to reach the cæcum is four and a half hours, the splenic flexure nine hours, and the sigmoid flexure twenty-six hours.

§ 199. Examination of Stomach Contents.[2]—First as to the Chemistry of Digestion, from a clinical standpoint, and the practical information to be derived from clinical examination of the stomach contents. *Four processes* normally take place in the stomach : (1) The conversion of starch into sugar, begun in the mouth, is carried on a stage farther ; (2) proteids are changed into peptones ; (3) fat globules are set free from their envelopes ; (4) milk is curdled. Delay in digestion may be caused by (1) deficient peristalsis of the stomach walls, (2) deficient quality or quantity of the gastric juice, (3) the consumption of indigestible articles, or (4) the dilution of the gastric juice by drinking too much fluid at meal-time.

The gastric juice contains HCl, water, pepsin, rennet, mineral salts, and a *little* mucus. Pepsin and rennet exist in the secretory cells only as zymogens, which,

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[1] Hertz, Proceedings of Royal Society of Medicine, February, 1908.

[2] It is not possible here to give more than a brief outline of this important subject, and the excellent treatises of Hemmeter and others on Diseases of the Stomach may be consulted with advantage.

in the presence of the HCl, become active ferments or enzymes. In the healthy state, as the result of digestion, about 30 c.c. of fluid should be obtained from the stomach one hour or so after a test-meal (*vide infra*), straw-coloured, without much odour, without organic acid, and with about 0·2 per cent. of free HCl.

As regards *hydrochloric acid*, much depends on the time of examination. *Hyperchlorhydria* is merely a convenient term for excessive secretion of HCl. It has come to be somewhat loosely used for " excessive acidity," and thus to be confused with the acidity of fermentation (due to organic acids). On the other hand, after a meal, a negative result on testing for HCl would indicate the absence of peptic activity, as an acid is required to convert the inactive proenzyme or pepsinogen into pepsin. An excess of HCl is distinctive of gastric ulcer, as compared with gastralgia ; for in the latter the HCl is normal or diminished. HCl is also diminished in all catarrhal conditions of the mucous membrane, in great anæmia, and neurasthenia. When there is a difficulty in diagnosing malignant disease, the absence of free HCl is a point in favour of cancer.

Lactic acid is not normally present in the gastric juice after digestion has proceeded for one hour, but traces may be found, due to the ingestion of lactic acid in certain foods, or to fermentation in the mouth. A decided reaction with Ueffelmann's test (*infra*) is found with cancer of the stomach, but a negative reaction does not have equal value in proving the absence of the disease.

Butyric and *acetic* acids prove the presence of fermentation, and are found where HCl is deficient, or the food is delayed in the stomach, as in dilatation of the stomach, or a narrowing of the pylorus.

The secretion of *pepsin*, according to most authors, is not interfered with, unless there be destruction of the glandular elements of the stomach. The presence of pepsin is also indicative of the presence of HCl (which is necessary to convert pepsinogen into pepsin), and of the activity of the glands. If only pepsinogen is found, the glands are active, but HCl is deficient. The presence of the latter (pepsinogen) is an important feature in diagnosing between chronic gastritis, where the glands are destroyed, and dyspepsia, or any neurosis of the stomach, where the glands are not destroyed.

Renninogen and Rennin are diminished or absent in the later stages of gastritis and cancer. The amounts of renninogen and rennin present appear to be directly proportional to the quantities of pepsinogen and pepsin.

Summary.—The two most important questions, therefore, to determine in an analysis of stomach contents are : (1) The presence and amount of HCl ; and (2) the presence and amount of pepsin and pepsinogen. The latter question is specially important, because—

(*a*) If *the ferments present are chiefly pepsin and rennin*, the gastric glands are active, and HCl is not deficient, since this is required to convert pepsinogen into pepsin.

(*b*) If *the ferments present are chiefly pepsinogen and renninogen*, the gastric glands are active, but the HCl is deficient.

(*c*) If *neither are present*, the gastric glands are destroyed or inactive.

Examination of Gastric Contents after a Test-Meal is a useful method of investigation in cases of severe and intractable indigestion or dilated stomach, or when cancer is suspected. It consists of three steps : (*a*) The administration of the test-meal ; (*b*) the withdrawing of the gastric contents by means of a stomach-tube ; and (*c*) the microscopical and chemical examination of the material withdrawn.

(*a*) The **test-meals** suggested are of many kinds, but the following answers : A pint of weak tea (infused only two or three minutes), with the addition of not more than 1 ounce of milk, and sugar if desired, or a pint of thin arrowroot, made with water and about 2 ounces of milk, sugar being added to taste. With these a round of thin buttered toast should be taken. The test-meal should be given in the morning before any food is taken, and drawn off an hour and a half later.[1]

[1] Dr. W. H. Willcox, *Lancet*, June 10, 1905, and July 25, 1908.

(*b*) **Method of Passing the Tube.**—Use a rubber tube nearly 2 yards long, with large "eyes." It should be thoroughly cleaned, and moistened with warm water before being passed into the pharynx. The patient should sit with the head erect, the chin being projected forward, and the mouth open. The tube is passed into the pharynx, while the patient is instructed to swallow, and the tube is pushed down into the stomach. Then bring the end down to a lower level than the stomach, and the contents should siphon out into a receptacle. If the gastric contents do not flow at once, the patient should strain, as if trying to vomit; or draw out the tube a little, lest the end becomes folded upon itself, or the "eye" stopped by a fold of mucous membrane. It may be necessary to start the flow by suction with a syringe. It is convenient to tie a thread round the tube, in order to mark a position 16 inches from its tip. By this means we know the amount of tube which has been passed into the œsophagus, because when the thread is opposite the incisors, the tip ought to have reached the stomach. A flexible tube curling up in the œsophagus is a contingency which may thus be obviated. Its use is *contraindicated* in angina, heart failure, fevers and other acute diseases, recent hæmorrhages, great arterio-sclerosis, aneurysms, and a high degree of emphysema and bronchitis.

(*c*) **Examination of Stomach Contents** after the test-meal.

General Examination.—Appearance, acidity to litmus paper, smell, consistency, and presence of slimy mucus, bile, or blood, should first be observed.

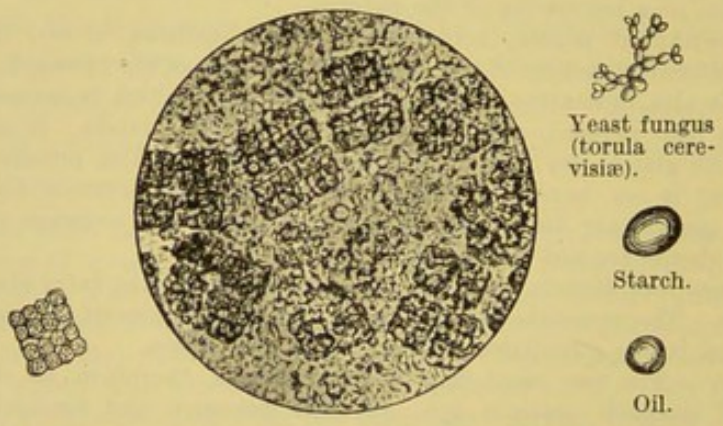

Fig. 62.—Vomited Matter. Sarcinæ Ventriculi. × 350. These are fungoid vegetations which indicate that food residues are retained in the stomach and fermentation has taken place. Yeast fungus has a similar significance.

Microscopically (Fig. 62) we can detect fat globules, starch cells, vegetable and muscle fibres, and sometimes fatty crystals, leucin and tyrosin, cells of the mucous membrane, torulæ cerevisiæ, or sarcinæ, and pus cells. Epithelial cells are present in excess in carcinoma. The bacillus of Boas may sometimes be seen on examination under the high power.

The stomach contents should be filtered, and the filtrate used subsequently.

Chemically we have to answer eight questions:

(*a*) *Are the contents acid?* can be roughly detected by the use of carefully neutralised litmus paper.

(*b*) *Method of estimating total acidity*—*i.e.*, acidity due to hydrochloric acid, organic acids, carbon dioxide, and such acids as react acid to phenolphthalein. Titrate 10 c.c. filtered gastric contents with decinormal solution of caustic soda (prepared free from carbon dioxide), using 1 per cent. solution of phenolphthalein as indicator. Add a drop or two of the indicator to the gastric contents, dilute with two or three times its volume of distilled water, and then run in the decinormal caustic soda solution from a burette till further addition produces a faint purple-red tint. If 5·5 c.c. of decinormal caustic soda are required for 10 c.c. of gastric contents, the acidity may be calculated *in terms of HCl* by multiplying the

5·5 c.c. of the soda solution used by 0·00364 × 10. The percentage of HCl actually present is therefore 10 × 5·5 × 0·00364 = 0·2 per cent.

(*c*) *Is free hydrochloric acid present? Tests:* (i.) A 1 per cent. solution of dimethyl-amido-azo-benzene in alcohol is used. A drop of gastric contents is placed on a white tile, and a drop of the reagent added. A deep pink-red colour is given by free HCl. (ii.) Gunzberg's test: Two or three grains of phloroglucin are mixed in a small evaporating dish with 1 or 2 grains of vanillin, about 1 c.c. of alcohol is added, and then about 1 c.c. of gastric contents. Evaporate on a water-bath, when a bright cherry-red colour indicates free HCl. If much organic acid is present, a slight reaction may occur in test (i.), which, though more convenient, is therefore not quite so reliable as test (ii.), which is never given by organic acids. Free hydrochloric is normally present. It is always present in gastric ulcer and hyperchlorhydria, hardly ever in carcinoma.

(*d*) *What is the amount of active hydrochloric acid?* The "active" hydrochloric acid includes (i.) free HCl; (ii.) the HCl which is combined with proteid and nitrogenous organic bases. It does not, of course, include the inorganic chlorides like NaCl. *Estimation.*—Two equal volumes of the filtered gastric contents (10 c.c.) are taken. (i.) One portion is made alkaline with sodium carbonate solution and evaporated to dryness on a water-bath in an evaporating dish. The residue is ignited over a small Bunsen flame until it is well charred and all the organic matter is decomposed. (ii.) The other portion of the gastric contents is placed in a porcelain evaporating dish (4½ inches in diameter), and evaporated to dryness on the water-bath, as in (i.). In each case the dish is cooled, and about 60 c.c. of water, 6 c.c. of pure nitric acid, 3 c.c. of 10 per cent. iron alum solution, and 10 c.c. of decinormal silver nitrate solution, added. Decinormal ammonium sulphocyanide solution is run in until a permanent brownish-red tint appears. The active HCl in 10 c.c. of gastric contents is equal to the difference of the amounts of ammonium sulphocyanide solution used in (i.) and (ii.). *Example.*—If in (i.) 3 c.c. of $\frac{N}{10}$ sulphocyanide solution are used, and in (ii.) 8 c.c. of $\frac{N}{10}$ sulphocyanide solution are used, then the active HCl in 10 c.c. of gastric contents is 5 c.c. of $\frac{N}{10}$ HCl. Therefore the percentage is 10 × 5 × 0·00365 = 0·18 per cent. This is the most important estimation, and the amount normally is about 0·15 per cent. *In carcinoma it is usually much below* 0·1 *per cent.; in simple ulcer it is usually considerably above* 0·15 *per cent.* It is the most important estimation in the analyses of gastric contents.

(*e*) *Is lactic acid present?* A quantitative test should be made, a weak solution of Ueffelmann's reagent (made by mixing a little 5 per cent. solution of carbolic acid with a few drops of liquor ferri perchloridi) being added to the filtered gastric contents. The development of a distinct yellow colour indicates lactic acid.

(*f*) *What is the amount of organic acids?* This is given with sufficient accuracy by the difference between the total acidity (expressed as HCl), and the active HCl as estimated by the method described above. They are in excess in conditions of much fermentation and gastric carcinoma.

(*g*) *Is mucin present in the filtrate?* To the filtrate add an equal volume of distilled water, and then a 2 per cent. solution of acetic acid, drop by drop. A white precipitate indicates the presence of mucin. It is always present in carcinoma, and sometimes in simple gastritis; usually absent in gastric ulcer.

(*h*) *What is the ferment activity?* The pepsin and pepsinogen may be roughly gauged by the carmine-fibrin test; the rennin and renninogen may be estimated quantitatively. *The Carmine-Fibrin Test.*[1]—Three test-tubes are taken containing respectively (1) 4 c.c. of gastric contents; (2) 2 c.c. of gastric contents and 2 c.c. of 0·4 per cent. HCl; and (3) 2 c.c. of gastric contents and 2 c.c. of water. A few shreds of washed carmine fibrin are placed in each, and the test-tubes are incubated

[1] The remainder of this section is largely composed of a quotation from the *Lancet*, 1908, vol. ii., p. 220, by Dr. Willcox, to whom the author is greatly indebted for valuable help in its preparation.

at 37° C. The test-tubes are shaken every few minutes, and the depth of red colour present indicates approximately the quantity of pepsin present. *Estimation of Rennin and Renninogen.*—The following method is exceedingly simple and well adapted to clinical purposes: Narrow test-tubes, 5 inches by $\frac{3}{8}$ inch, are taken. Into each 5 c.c. of fresh unboiled milk are added. They are placed in a water-bath at 40° C. Into the tubes are placed seriatim gradually increasing quantities of the filtered gastric contents. These are run in from a pipette, which is graduated in $\frac{1}{100}$ths of a cubic centimetre. Thus are added 0·01, 0·05, 0·1, 0·15, 0·2, 0·25, 0·3, 0·35, 0·4, 0·5, 0·6, 0·7, 0·8, etc., of gastric contents. The liquids are mixed by gently inverting each tube. The tubes are left thirty minutes in the bath, and then it is found that above one point in the series the contents of the tube are solid, and do not flow out on inversion, while below this point the contents are liquid, and flow out on inversion of the tube. The minimum quantity of gastric contents to cause complete clotting, so that the contents of the test-tube do not flow out on inversion, gives an accurate measure of the rennin activity. In the normal gastric contents of adults about 0·2 c.c. is required; in children a greater amount is normally required. If X = the minimum quantity of gastric contents to cause complete clotting, then the quantity of rennin present in the gastric contents = $\frac{0{\cdot}2}{X}$ of the normal amount; *e.g.*, if 0·05 c.c. of gastric contents was the minimum amount to cause complete clotting, the quantity of rennin present = $\frac{0{\cdot}2}{0{\cdot}05}$ = four times the normal. In *gastric carcinoma* usually much more is required —*e.g.*, 0·5 c.c., or more. Possibly no complete clotting will occur at all. In *gastric ulcer* and *hyperchlorhydria* the rennin activity is usually higher than normal, and never below normal. Often 0·05 c.c. or less causes complete clotting. In cases where the acidity of the gastric contents is low, and also the ferment activity, most of the ferment is frequently present as renninogen, and not as rennin. In these cases the above method of analysis should be adopted for the estimation of the rennin, and a separate estimation made for the rennin plus renninogen. The renninogen can be estimated by taking the gastric contents, adding an equal volume of 0·4 per cent. HCl, which converts the renninogen to rennin. The same procedure is adopted as above—viz., the addition of the mixture of HCl and gastric contents in increasing quantities to the milk in the test-tubes respectively, and the minimum amount of the mixture which will cause complete clotting, so that the contents do not flow out on inversion, is noted. In the normal gastric contents of adults about 0·1 c.c., when mixed with 0·1 c.c. of 0·4 per cent. HCl, is the minimum quantity which will clot completely 5 c.c. of milk at 40° C. in half an hour; with children a greater amount is necessary. If X = the minimum quantity of the gastric contents which, when mixed with 0·4 per cent. HCl, will cause clotting (*i.e.*, half the amount of the mixture taken), then the quantity of rennin plus renninogen present = $\frac{0{\cdot}1}{X}$ of normal; *e.g.*, if in a given case 0·1 c.c. of the mixture of gastric contents and 0·4 per cent. HCl was the minimum amount to cause complete clotting, then, since 0·05 c.c. = the quantity of gastric contents in 0·1 c.c. of mixture, the quantity of rennin plus renninogen present = $\frac{0{\cdot}1}{0{\cdot}05}$ = 2 times normal. Sometimes where the rennin is below normal the renninogen plus rennin are found to be normal. This would contraindicate a condition such as gastric carcinoma. The importance of the estimation of the rennin is accentuated by the fact that the amount present is usually directly proportionate to that of the pepsin.

PART C. DISEASES OF THE STOMACH, THEIR DIFFERENTIATION, PROGNOSIS, AND TREATMENT.

§ **200.** The **Routine Investigation** of the disorders of the stomach consist of three steps :

FIRST : We must identify the patient's LEADING SYMPTOMS as being referable to gastric disorder (see Part A).

SECONDLY : Inquire as to the HISTORY, and especially whether the illness came on *acutely* and recently, or whether, as is more usual, it came on insidiously, and has run a *chronic* course. Much depends on the skill and method with which the history is elicited. Inquire particularly as to pain or discomfort and its relation to meals, and as to the other symptoms mentioned in Part A.

THIRDLY : Proceed to the PHYSICAL EXAMINATION, and ascertain whether there be any localised tenderness and pain, and whether any tumour or other abnormality be present.

Classification of disorders of the stomach.

A. Acute Diseases of the Stomach.

I. Acute dyspepsia (bilious attack) : *without tenderness* ..	§ 201
II. Acute gastritis : *with tenderness*	§ 202

B. Chronic Diseases of the Stomach.

(*a*) WITHOUT TENDERNESS ON PRESSURE : and pain less marked.

I. Chronic atonic dyspepsia	§ 204
I. Chronic acid or irritable dyspepsia	§ 205
III. Gastralgia (gastric neuralgia)	§ 206

(*b*) WITH TENDERNESS ON PRESSURE : pain a marked feature.

IV. Simple ulcer of the stomach	§ 207
V. Cancer of the stomach	§ 208
VI. Chronic gastritis	§ 209
(*c*) DILATATION OF THE STOMACH	§ 210

This classification, based on the presence or absence of tenderness, is not very satisfactory, and each group will be found to contain many exceptions. It is, however, the least unsatisfactory of those clinical classifications possible at the present time. The division of Chronic Dyspepsia into Atonic and Acid is also very unsatisfactory (see footnote, p. 297).

If the patient's symptoms have come on gradually, and lasted a considerable time, turn to **Chronic Disorders** of the Stomach (§ 203).

If, on the other hand, his symptoms have commenced somewhat suddenly and recently, the case is probably one of the two **Acute Disorders** of the Stomach : I. ACUTE DYSPEPSIA ; or, II. ACUTE GASTRITIS.

I. *The patient—whose temperature is normal—complains of* NAUSEA, GASTRIC DISCOMFORT, *headache, and depression, which have come on suddenly ; and there is no marked epigastric tenderness.* The disease is probably ACUTE DYSPEPSIA.

§ **201. Acute Dyspepsia** (" Bilious Attack ") consists of a sudden disturbance of the digestion in a previously healthy person, such as occurs in association with surfeit, high living or other errors in diet.

The *Symptoms*, which come on suddenly, are : (1) Pain, or a feeling of oppression or distension in the epigastrium, occasionally accompanied by some tenderness on pressure, though the tenderness is never very marked. (2) Nausea and vomiting very generally ensue (but not always). (3) Headache, depression, anorexia, coated tongue, constipation, scanty urine loaded with lithates. (4) The illness is sometimes preceded and accompanied by drowsiness, and not infrequently there is a history of previous similar attacks.

The *Diagnosis* is not difficult, the only condition resembling it being acute gastritis, in which the constitutional symptoms are more apparent, the duration of the illness considerably longer, and the *tenderness much more marked*. Irritant poisoning comes on much more suddenly with very urgent vomiting (§ 191).

Etiology.—(1) Too large a meal, especially after previous fatigue. (2) Errors in diet, such as excess of alcohol (which retards digestion), ice, and many other articles which vary with the idiosyncrasy of the individual.

Prognosis and Treatment.—Acute dyspepsia of the kind here referred to usually passes off in two or three days. (1) If pain be present, assist vomiting by mild emetics, such as copious draughts of salt and water, tickling the fauces, etc. Violent emetics aggravate the condition. (2) Three grains of calomel, and milk diet for a day or two, generally relieve the condition. (3) Bismuth and tonics may be given during convalescence.

II. *The patient complains of considerable* PAIN *or discomfort, and* TENDERNESS IN THE EPIGASTRIUM, *with nausea or vomiting, all of which have come on somewhat suddenly*. The disease is probably ACUTE GASTRITIS.

§ **202. Acute or Sub-acute Gastritis** is relatively a much more serious disorder than the foregoing. It consists of a sudden derangement of digestion due to inflammation of the stomach. This condition is not so much a catarrhal inflammation of the mucous membrane (excepting in cases of irritant poisoning) as of the glands of the stomach.

Symptoms.—(1) Pain, intense and burning, or a feeling of distension in the epigastrium, coming on directly after food, and accompanied by tenderness on pressure. (2) Vomiting, not always immediately after a meal, of undigested food, sometimes with streaks of blood. (3) Malaise, anorexia, slight pyrexia, headache, depression, and other constitutional symptoms may be present, attended sometimes by great prostration, thirst, furred and coated tongue. (4) Diarrhœa may ensue after a day or two.

The *Diagnosis* may have to be made from acute dyspepsia (§ 201), and from other causes of vomiting (§ 191).

Recovery generally takes place in about three to six days, the affection

rarely lasting longer than eight or ten days. It may go on to chronic gastritis. Death rarely takes place, excepting from irritant poisoning or in cases of membranous gastritis.[1]

Etiology.—(1) In the majority of cases simple acute gastritis is caused by errors in diet, or by decomposing meat—*e.g.*, tinned food containing ptomaines ;[2] an excessive quantity of normal food will cause it. (2) Irritant poisons (*e.g.*, arsenic, antimony, phosphorus, etc.). In long continued vomiting, without apparent cause, poisoning should be suspected, and the vomited matters examined. (3) In some cases, gout and other constitutional conditions predispose to or determine an attack.[3] Heart, lung, and liver disease are predisposing causes.

Treatment.—The indications are : (1) To remove any irritant that may be present from the stomach. This can be done by promoting vomiting, which is specially indicated if the epigastric pain continues, employing the means mentioned in § 201. It may be desirable to give a purgative, such as 3 grains of calomel, and a seidlitz powder in the morning. Hot fomentations or a mustard leaf to the epigastrium may relieve the pain. (2) The second indication is rest to the stomach, which is gained by twelve or twenty-four hours' abstinence from food, followed by milk in small quantities. Later on, bismuth combined with opium is the best treatment. The milk diet should be supplemented only very gradually.

CHRONIC DISORDERS OF THE STOMACH.

§ **203.** *The patient, whose temperature is normal, complains of* "**Chronic Indigestion,**"—i.e., *pain or discomfort in some way connected with his food, which has probably come on gradually, and may have lasted a long time.* There are SIX DISORDERS, from any one of which he may be suffering, and there may be *Dilatation of the Stomach* in addition.[4]

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[1] One case of this rare condition which recovered is recorded by Dr. Grunbaum in the *Lancet*, August 2, 1902.

[2] The products of the decomposition of nitrogenous food-stuffs, especially when enclosed in hermetically sealed tins, occasionally give rise to the formation of toxic substances. The effects are very severe. Symptoms of acute irritant poisoning come on within a short time after the meal. The collapse is extreme, and death may take place within a few hours.

[3] I remember being called to see a medical man, about forty-five years of age, of markedly gouty diathesis, who had previously had gouty manifestations. The symptoms were thought at first to resemble those of enteric fever, the temperature being 103° to 105° F. for several days ; but a brisk purge of calomel, salicylates, alkalies, and milk diet, produced immediate improvement.

[4] As I have already remarked, this classification is a very unsatisfactory one. As more scientific methods of investigation, such as those foreshadowed in §§ 198 and 199, come to be employed, we shall be able to classify cases which we now vaguely describe as " HRONIC DYSPEPSIA " into the following groups—groups which tell us wherein the chief error of digestion lies :

I. DISORDERS OF SECRETION.

(*a*) *Irritative States.*—(1) Hyperacidity (hyperchylia), hyperchlorhydria, excessive formation of HCl. (2) Supersecretion (gastrosuccorrhœa.

(*a*) **Functional** diseases of the stomach **without tenderness.**
I. Atonic Dyspepsia.
II. Acid or Irritable Dyspepsia.
III. Gastralgia.
(*b*) **Organic** diseases of the stomach **with marked local tenderness** and pain.
IV. Simple Ulcer of Stomach.
V. Cancer of Stomach.
VI. Chronic Gastritis.
(*c*) There are also many other disorders **unconnected with the stomach** which may give rise to symptoms of chronic indigestion, among which the following may be mentioned : Phthisis (of which dyspepsia is often the earliest symptom), Appendicitis, Abdominal Tumour, Cardiac or Hepatic Disease, Renal or Uterine Disease, various Nervous Disorders, and Pancreatic Disease (rare).

I. *The patient complains of* CHRONIC INDIGESTION, *and the epigastric pain or discomfort comes on* SOON AFTER A MEAL. The disease is probably ATONIC DYSPEPSIA.

§ **204. Chronic Dyspepsia** may be defined as deranged digestion without gross or inflammatory changes in the mucous membrane of the stomach. It may be, and often is, attended by Atony or Dilatation, § 210. It occurs in two generally accepted types.

I. ATONIC DYSPEPSIA (the commoner form) is chronic indigestion due to diminished digestive power of the stomach. It is probably due to a deficiency of the acid in the gastric juice ; the pepsin is said by most to remain normal in amount. In this disease the food may, in process of time, undergo butyric acid fermentation, and then it is difficult to distinguish this form of dyspepsia from II.

II. ACID or irritable DYSPEPSIA (§ 205 ; Synonym, hyperchlorhydria)

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a continuous flow of gastric juice : doubtful if this exists apart from dilatation.
(*b*) *Depressive States.*—(1) Subacidity (hypochylia). (2) Inacidity (achylia gastrica).

II. DISORDERS OF MOTILITY, or Peristalsis.
(*a*) *Irritative States.*—(1) Cramp of the cardia, pylorus, entire stomach. (2) " Peristaltic unrest " of Kaussmaul. (3) Nervous eructations. (4) Nervous vomiting.
(*b*) *Depressive States.*—(1) Insufficiency of the cardia or the pylorus. (2) Gastroplegia—atony or insufficiency of the entire gastric muscle, leading to dilated stomach.

III. SENSORY DISORDERS.
(*a*) *Irritative States.*—(1) Hyperæsthesia. (2) Gastralgia. (3) Bulimia and polyphagia.
(*b*) *Depressive States.*—(1) Anorexia. 2. Acoria (? gastric anæsthesia).

IV. GASTRIC NEURASTHENIA, and GASTROPTOSIS.

These various conditions are, of course, met with most frequently in combination, just as paralysis and anæsthesia are met with in disease of the spinal cord. Thus atony and dilatation inevitably lead to disordered secretion. But it is of the greatest use for purposes of prognosis and treatment to know which particular element in the digestive process is at fault. The subject, however, is extremely complex. For instance, the gastric contents, in cases of Subacidity, may be highly acid from the presence of fatty acids ,the products of decomposition. Those desirous of pursuing this subject should consult Hemmeter's " Diseases of the Stomach " (Blakiston, Philadelphia), or the works of Mathieu (" Traité des Maladies de l'Estomac et de l'Intestin," Paris, 1901), Ewald, Reisshmann, Einhorn and Rosenheim.

is a chronic indigestion due to hypersecretion of acid in the gastric juice.

I. **Atonic Dyspepsia** is the commoner form of chronic dyspepsia. The *Symptoms* are: (1) Pain or distress, usually in the epigastrium, coming on immediately or *very shortly* after food. The pain may be in the back or shoot up to the shoulders; or there may be no definite pain, only a feeling of weight or distension. It is unaccompanied by tenderness on pressure, a feature which distinguishes it from gastritis and other organic conditions. The pain is often relieved by eructations of wind. (2) Nausea and vomiting are not frequent. (3) The appetite is usually diminished, but sometimes it is increased; and the tongue is flabby and indented by the teeth. (4) There are languor, depression, general discomfort; and there may be palpitation, dyspnœa, and other cardiac symptoms. Thirst is not usual unless there be dilatation, and pyrexia is absent. Urates in excess are constantly present in the urine.

Etiology.—(1) Errors of diet. (2) Overwork, mental anxiety, and other nervous derangements: reading during meals is said to give rise to it. (3) Imperfect mastication in previous years. (4) Convalescence from acute diseases, anæmia, and debility from any cause (*e.g.*, phthisis), predispose. Dyspepsia is often the earliest symptom met with in phthisis. (5) Various abdominal disorders — *e.g.*, pancreatic or renal disease, appendicitis, and abdominal tumour, may for some time be evidenced only by symptoms resembling atonic dyspepsia.

Diagnosis.—The chief condition from which it has to be distinguished is *chronic gastritis*, in which there is usually tenderness on pressure; and, while stimulating articles of food (pickles, condiments, etc.), relieve the pain of atonic dyspepsia, they tend to aggravate chronic gastritis (see also Table XV.). Atonic dyspepsia may have to be differentiated from *gastric ulcer* in the young, or *cancer of the stomach* in the middle-aged and old (*q.v.*). The differentiation from *acid dyspepsia* is given under that disease (§ 205), but it must be remembered that in atonic dyspepsia butyric acid fermentation is apt to take place, and it is then almost indistinguishable from acid dyspepsia, except by an examination of the stomach contents.

Prognosis.—It is never fatal, but often makes the life of the patient very wretched, and unfits him for the duties of life. If met with early, treatment may be very efficacious; but, if untreated, it may go on to chronic gastritis and dilatation of the stomach, and lead to general malnutrition (§ 194).

Treatment.—The indications are: (1) To remove the dietetic errors (see § 212). In anæmic cases, soups and stimulating proteid foods are better than farinaceous ones. (2) To stimulate the secretory and motor power of the stomach. This may be done by the use of alkalies and

alkaline carbonates shortly before meals, combined with nux vomica, bitters, and carminatives (F. 66). Some cases do best by taking their meals dry, so that the gastric juice may be undiluted. (3) Some find aid in pepsin, pancreatin, peptenzyme, taka-diastase, or other artificial digestive. (4) Various symptoms require treatment. For the *flatulence* 20 grains of sodium bicarbonate in a cupful of hot water gives great relief. Peppermint, sp. chloroformi, rhubarb, cinnamon, ginger, cardamoms, pepper, charcoal, or F. 50, are all useful. *Acid eructations* (which are generally due to butyric acid fermentation) may be counteracted by antiseptics (carbolic acid or creosote, etc.), or acids after meals. *Pain* disappears when the gastric juice is increased ; otherwise bismuth, hydrocyanic acid, and opium (with caution)[1] may be called for. For *breathlessness*, *palpitation*, and other *cardiac symptoms*, sal volatile, saline purgatives, and alkalies may be given. (5) Tonics are useful in the later stages, especially strychnine. For further detail as to treatment see Chronic Gastritis (§ 209).

TABLE XV.

	CHRONIC DYSPEPSIA.	CHRONIC GASTRITIS.
Tenderness	Absent.	Present.
Vomiting	Not frequent, but relieves pain.	Frequent, especially in the morning, of mucus ; no relief.
Thirst ..	Varies ; not common.	Usually marked.
Fever ..	Absent.	Sometimes slight fever.
Causes ..	1. Dietetic errors. 2. General weakness of system (anæmia after fevers, etc.) ; or nervous exhaustion, leading to *deficient secretion* of gastric juice.	1. Dietetic errors, especially alcoholic excesses. 2. Sequel to Heart or Liver Disease.
Course ..	Liable to come on in attacks, lasting a few days or weeks at a time ; brought on by slight causes.	Does not come and go, but progressively advances, and goes on to dilatation of the stomach.

II. *The patient complains of* CHRONIC INDIGESTION, *but the discomfort does* NOT COME ON DIRECTLY *after a meal, and may be relieved by food.* The disease is probably ACID DYSPEPSIA.

§ **205. Acid Dyspepsia** (Irritable Dyspepsia, Hyperchlorhydria, compare § 199) may be defined as chronic indigestion due to the hypersecretion of hydrochloric acid in the stomach ; pepsin, according to

[1] De Quincey started his habit of "opium eating" for an intractable form of chronic dyspepsia.

most authorities, remaining constant in amount. Opinions are divided as to whether this is or is not a pure neurosis. Some regard it as a *chronic glandular gastritis*, set by up local irritation of injudicious food, alcohol, or the decomposition of retained food residues.

The *Symptoms* which distinguish this from Atonic Dyspepsia, which it resembles in other respects, are : (1) Pain, severe, gnawing, intense, burning, *coming on one or two hours after food ;* unattended by tenderness on pressure ; and usually relieved by taking food. (2) Vomiting may occur, or acid eructations, which may be so acrid as to make the throat sore ; thirst, and generally an increased appetite. (3) *The presence of HCl in an empty stomach*, say, before breakfast, is the crucial test of hypersecretion.

Etiology.—(1) It is usually met with in young adults, or men in the prime of life with strong constitutions. (2) Some maintain that this is in reality a neurosis (*vide supra*). (3) It may arise from excess in alcohol, or highly spiced foods, or simply overloading of the stomach.

Diagnosis.—Acid may be distinguished from *Atonic Dyspepsia* by the above symptoms. For the diagnosis from *Chronic Gastritis*, see § 209, and Table XV., p. 300. *Cancer* is detected by the " coffee-ground " appearance of the vomited matters, and the cachexia of the patient. *Gastralgia* may simulate acid dyspepsia ; but in the latter the pain is relieved by alkalies, while in gastralgia it is not so relieved. The examination of the stomach contents shows that in hyperchlorhydria the proteids are more completely digested than in gastralgia.

The *Treatment* is mainly dietetic (see § 212). A diet of proteids relieves the condition, but if persisted in too long, this further stimulates the secretion of HCl. Antacids, such as large doses of sodium bicarbonate or creta preparata, may be given one to two hours after meals. Lozenges constantly sucked, which induce a considerable amount of alkaline salivary secretion, are useful, especially the bismuth, magnesia, and chalk lozenge (B.P.). As a temporary measure, the acidity of the stomach may be diluted by a copious draught of hot water, which relieves the pain and acid eructations. A course of galvanism (see § 210), combined with the proteid diet given below, is very efficacious.

In severe cases of Acid Dyspepsia the following diet may be tried for a few days and relaxed gradually afterwards. The *meals* to consist of meat cakes (as much as desired), and one or two slices of toast. Meat cakes are prepared by scraping the fibres either of meat, fish, or poultry, with a blunt knife, leaving behind all the gristle and sinews. Add a little salt, press into cakes, and fry lightly. Only 4 ounces of *fluid* to be drunk with each of the meals. As much fluid as desired may be drunk, but not nearer to a meal than one hour before or two hours afterwards. Osler recommends strictly meat diet[1]: 3½ ounces of meat, minced fine, taken raw, with two slices of stale bread and 1 ounce of butter, with one glass of Apollinaris water, thrice daily.

[1] Herschell (*Brit. Med. Journ.*, 1898, vol. ii., p. 1323) holds that cases of *Hyperchlorhydria* get worse on the administration of a largely or entirely proteid diet,

III. *The patient complains of sharp paroxysmal* PAIN, *having* NO DEFINITE RELATION *to the taking of food, and careful investigation reveals no structural disorder of the stomach.* The case is probably one of GASTRALGIA.

§ **206. Gastralgia** is a gastric neuralgia, sometimes attended by a hyperæsthesia of the mucous membrane of the stomach, but always without structural changes or alteration of secretion.

Symptoms.—(1) The pain is of a sharp or burning character in the epigastrium, usually relieved by pressure. There is generally no tenderness, but if present, it is usually more marked with a light than a heavy touch, thus differing from organic disease. Sometimes it is unilateral. The pain may begin immediately after food, but may come on either when the stomach is empty or when it is full. The *irregularity of its advent* is one of its most characteristic features. Sometimes it comes on with the first mouthful of food ; sometimes food relieves it ; sometimes it occurs in attacks unrelated to food. Dieting gives no relief, for it may be worse after a milk diet than after raw apples. (2) Vomiting and other symptoms are rare. (3) It generally occurs in neurotic people, who have had neuralgia elsewhere. (4) It may accompany gastric ulcer, or follow this and other diseases of the stomach.

Etiology.—(1) Gastralgia may come on at any age, and in either sex. (2) Some constitutional state, such as hysteria, neurasthenia, anæmia, ague, alcoholism, or gout, is usually present at the same time. (3) In tabes dorsalis, gastralgia is the most frequent form of crisis (*crise gastrique*).

Diagnosis.—Those cases of gastralgia in which food relieves the pain have to be diagnosed from *Hyperchlorhydria.* The diagnosis in such cases is effected, first, by administering alkalies an hour or so after meals ; they relieve the pain of hyperchlorhydria, but not that of gastralgia. Secondly, acid eructations are a prominent feature of hyperchlorhydria, but not of gastralgia. Thirdly, excess of hydrochloric acid is found on examining the stomach contents in hyperchlorhydria. *Ulcer of the stomach* has a more limited area of tenderness on pressure ; the pain comes on immediately after food, and is relieved by vomiting. Increased HCl is found on examination of the vomited matter. *Cancer of the stomach* is very difficult to diagnose from gastralgia before tumour or hæmatemesis supervenes, but in this disease the pain is usually more constant. *Biliary colic* is usually associated with jaundice.

Treatment.—(1) Treatment directed to the constitutional condition generally relieves the gastric trouble sooner or later. (2) Warmth to the epigastrium, opium (with caution), nitrate of silver internally ($\frac{1}{6}$ grain), and arsenic in small and frequent doses. For the vomiting give hydrocyanic acid, bromides, and liq. arsenicalis (♏1 in a drachm of water every half an hour).

GROUP B. If the patient complains of **Chronic Indigestion,** attended by pain and marked **tenderness on pressure,** we are justified in suspecting the presence of organic disease of the stomach—viz., IV. SIMPLE ULCER ; V. CANCER ; or VI. CHRONIC GASTRITIS.

IV. *The patient is an anæmic young woman, and complains of severe* PAIN, PRODUCED BY FOOD *and* RELIEVED BY VOMITING, *the vomit sometimes containing a large quantity of blood.* The disease is SIMPLE ULCER OF THE STOMACH.

§ **207. Simple** (*i.e.*, non-malignant) **Ulcer** of the stomach, is so called in distinction from cancerous ulceration. The ulcer is usually single,

by the encouragement thus given to the hypersecretion of acids. He therefore suggests the substitution of a carbohydrate diet partially dextrinised by taka-diastase, and the neutralisation of the hyperacidity by large doses of alkalies. Weak solutions of tannin are also good.

and generally situated on the posterior wall, near the pylorus on the lesser curvature. In this disease there are, in addition to symptoms of chronic dyspepsia, three very characteristic features :

(1) *Pain* of an intense boring character usually limited to one spot, (2) *aggravated by food*, and accompanied by tenderness. A small, very tender area, pressure on which even by the bed-clothes cannot be borne, is sometimes present, and is very characteristic. It is usually situated in the epigastrium. (3) The pain is *relieved by vomiting*, which comes on very shortly after food. The vomited matter contains an excess of hydrochloric acid. (4) Hæmatemesis, which may be profuse, comes on suddenly from time to time. (5) The appetite is usually normal or increased, but the patient avoids food because of the pain it produces. There is generally constipation. In some cases all symptoms may be in abeyance, the disease being quite *latent*, until hæmorrhage or perforation suddenly occurs.

The *Diagnosis* is not difficult if pain, an area of tenderness, and hæmatemesis be present. The last, which was thought to be the most characteristic symptom, is now known to be very profuse in gastrostaxis (§ 192). When one or other is absent, the disease has to be diagnosed from *gastralgia* (§ 206) ; from *cancer* and *chronic gastritis*, see Table XVI., below, or from the other causes of Hæmatemesis (§ 192).

TABLE XVI.

	SIMPLE ULCER.	MALIGNANT DISEASE.	CHRONIC GASTRITIS.
Vomiting	Frequent ; directly after food ; relieves pain.	Infrequent ; but very large quantity every few days.	Morning vomiting of mucus.
Hæmatemesis	Occasional but profuse ; therefore bright red.	A continuous oozing ; therefore "coffee-ground" in character.	Rare ; and only streaks, unless in the venous congestion due to heart disease.
Tumour	None.	Present, though may not be palpable ; secondary deposits may be recognisable in liver, peritoneum, glands, etc., later on.	None.
Age ..	Young women, twenty to thirty.	Usually men over forty.	Any age.
Course ..	Indefinite ; relapses occur.	Fatal in one to two years.	Indefinite ; may go on to Dilatation.

Etiology.—Sixty per cent. of Welch's cases[1] occurred in females. The usual age in the former was twenty to thirty; in the latter, forty to fifty. It is very apt to occur in association with anæmia and anæmic states generally (Chapter XVI.), and some cases have been traced to embolism from heart disease. Some say it is more common among the poorer classes; it certainly is very often met with among domestic servants.

Prognosis.—On the whole, the mortality is about 10 per cent. The prognosis is usually favourable if the condition is treated early, but there is a great tendency to relapse. If untreated, perforation into the peritoneal cavity may cause death (see § 169). When a more favourable course is followed, the resulting cicatrisation may lead to distortion or stricture of the stomach or pylorus. Adhesions to surrounding viscera, subphrenic abscesses, or abscess in other situations may result. Death occasionally results from hæmorrhage. The amount of the bleeding is no measure of the size or depth of the ulcer.

Treatment.—In all but the mildest cases the patient must rest in bed. If there has been recent hæmatemesis or intractable vomiting, no food should be allowed by the mouth, but ice may be given to suck. Alimentation must, in such cases, be solely *per rectum*. A suitable enema is as follows: Pancreatic solution, 1 drachm; bicarbonate of soda, 10 grains; yolk of one egg; beef-tea, ½ ounce; and milk, up to 4 ounces. (Other enemata are found in Fig. 74.) These should be given hourly. The bowel must be washed out with saline at least once a day, and as much saline as possible should be retained, for by this means the thirst is relieved. After from three to ten days, a very gradual return must be made to ordinary diet, beginning with peptonised or citrated milk, then custard and bread and milk, then bread and butter, eggs, fish, and chicken, and finally butcher's meat. The treatment extends over about three months, most of which should be spent in bed. In less severe cases dieting may start with milk only, either peptonised or citrated, and the food gradually increased.

The treatment of the hæmorrhage is fully given under Hæmatemesis (§ 192). In the intervals between the acute attacks alkaline carbonates, bismuth, small doses of tannin and many other of the remedies used in gastritis (*q.v.*) has been recommended. For the pain, give hydrocyanic acid, opium, bismuth, alkalies, or cocaine. In very chronic cases nitrate of silver may be tried. To regulate the bowels, the best form of aperient, if enemata fail, is a drachm of Carlsbad salts in 3 or 4 ounces of water (120° F.) taken every fifteen minutes in four doses up to half an hour before breakfast.

The indications for operation in gastric ulcer are (i.) perforation;

[1] Quoted by Osler, "Prin. and Prac. of Med." (second edition), p. 394.

(ii.) for frequently recurring obstinate cases ; and (iii.) for hæmatemesis. For perforation, immediate operation is imperative. For (ii.) and (iii.) surgical measures should not be lightly employed, since recurring and severe hæmatemesis may be associated with ulceration (see Gastrostaxis, p. 284). Moreover, recurrence is by no means uncommon, after excision of the ulcer or gastroenterostomy has been performed. If, in spite of adequate treatment, such as is detailed above, the pain, vomiting, or bleeding prove intractable, operation must be considered.

Duodenal Ulcer occurs mostly in males ; Osler refers to statistics where the condition occurs in 171 males and 39 females. The symptoms may be very obscure. Sometimes there is only a fixed abdominal pain. It may be diagnosed clinically by three symptoms occurring in a person of the male sex : (1) Sudden intestinal hæmorrhage, as evidenced by melæna, preceded or accompanied by hæmatemesis ; (2) pain two to three hours after food in the right hypochondrium ; (3) severe attacks of gastric pain from time to time, usually with hæmorrhage. Local tenderness and rigidity in the right lumbar and hypochondriac regions is sometimes present. As in gastric ulcer, the first symptoms may be those of perforation. The treatment resembles that for gastric ulcer.

V. *The patient, who is in middle or advanced life, presents more* CACHEXIA *than could be accounted for by dyspepsia, and vomits from time to time* "COFFEE-GROUND" MATERIAL. There is probably MALIGNANT DISEASE OF THE STOMACH.

§ **208. Cancer of the Stomach.**—The stomach is a frequent site for primary cancer ; it has been found in as many as 1 per cent. of all post-mortems. The word "cancer" is associated in our minds with a tumour, but in two-thirds (two-fifths Hemmeter) of the cases of cancer of the stomach there is no tumour, but a scirrhus infiltration of the pylorus, which produces obstruction of that orifice and leads to Dilatation (§ 210). The clinical history, which rarely extends beyond one or two years, may be described in three stages. In the first stage we find the symptoms of chronic gastritis (§ 209) combined with marked cachexia. In the second stage, combined with these are acute pain (generally), vomiting, and hæmatemesis of a very characteristic kind. In the third stage, beside the preceding, we get either dilatation of the stomach, or tumour, or both. In many cases, however, there are no symptoms referable to the stomach, and the diagnosis is only made in the deadhouse.

Symptoms.—(1) *Loss of appetite*, soon followed by *cachexia*, occur early, and are very marked ; and these symptoms in a patient of 40 or upwards should always make us suspect the condition. The sallowness of the skin may almost pardonably be mistaken for Addison's disease, or even jaundice. (2) The *pain* is situated in the epigastric region or back, radiates in different directions, and is usually accompanied by tenderness. It is continuous, sometimes increased by food, but sometimes independent of the taking of food. (3) *Vomiting* is a fairly constant sign. Generally it takes place some time after the

ingestion of food, the interval depending upon the position of the lesion; thus, if at the cardiac end the interval is short; if at the pylorus it may be hours after taking food. Sometimes the vomiting occurs every two or three days. An examination of the vomited matter shows diminution or absence of hydrochloric acid and the presence of lactic acid. (4) *Hæmatemesis* is generally present sooner or later. The bleeding is small in quantity, but occurs frequently, and therefore the blood is partly digested, and gives rise to a characteristic brown appearance, as of *coffee-grounds*. (5) *Dilatation* of the stomach is sure to ensue if the pylorus is involved (§ 210). Sarcinæ (Fig. 62) and other evidences of decomposition may be present, and sometimes cancer cells. (6) *Tumour* is much less rarely met with than one would expect. Transmitted aortic pulsation, and a little fulness or rigidity of the upper end of the right rectus, may be present without a palpable tumour. When cancer is deposited in the pylorus it may cause adhesions which prevent the tumour from coming forward. The great majority of gastric tumours come forward to the left of the middle line. It is usually stated that whereas hepatic tumours move, gastric tumours do not generally move with respiration; but this feature, as Hemmeter points out (*loc. cit.*), has many exceptions. One of greater importance is their alternate appearance and disappearance. At first they are extremely mobile, but later on they become fixed owing to adhesions. This is also the reason why perforation is rare. Distension of the stomach by copious draughts of water may help us in the physical examination.

Etiology.—(1) Cancer of the stomach is more frequent in men. (2) It is rarely met with under forty, although I have seen one case of twenty-eight, another of thirty, and several between thirty and forty years.[1] (3) Hereditary influence often exists. (4) Simple ulcer and chronic gastritis appear to predispose.

Diagnosis.—Anorexia and cachexia are the only constant symptoms. When the typical vomiting is absent, the real nature of the case may be readily overlooked.

The chemical examination of a test-meal is of great value. According to Willcox, in involvement of the *cardiac* end, the total acidity is low, and free hydrochloric acid is never present. Active hydrochloric acid is present in very small amount—about 0·02 per cent. Organic acid is generally present, also traces of mucin, but little or no albuminose or peptone. In involvement of the *pyloric* end the total acidity is subnormal, but still may be considerable (from 0·05 to 0·1 per cent.). Free hydrochloric acid is absent; active hydrochloric acid may be present in fair amount, but scarcely ever exceeds 0·1 per cent. Organic acids are present generally. Mucin, albumin, and large amounts of peptone are found.

If emaciation be rapid, and gastric symptoms resist treatment, cancer should be strongly suspected. (1) *Dyspepsia* and *chronic gastritis* have

[1] My experience of this disease at the Paddington Infirmary was perhaps unusually large. The case, aged twenty-eight, is recorded in the *Clinical Journal*, about 1888 or 1889.

pain directly related to food; for these, and (2) *Simple ulcer* of the stomach, see Table XVI., p. 303. (3) *Simple* pyloric stricture (*vide* Dilatation). (4) *Tumour of the pylorus* or stomach has to be diagnosed from tumour in the neighbouring regions (§ 188). (5) *Addison's disease* and other cachectic conditions (Chapter XVI.). *Pernicious anæmia* is sometimes strongly suggested by the colour of the patient, but in this disease there is not a corresponding amount of emaciation, and the blood-picture is characteristic.

The *Prognosis* is very grave. The duration is rarely longer than six to eighteen months after the first definite symptoms appear. Death is the invariable result unless surgical measures are adopted. The symptoms upon which one relies most in the diagnosis in these cases, anorexia and emaciation, have always appeared to me to be those which also best measure the longevity of the patient. Death generally takes place by inanition, but almost as often it occurs suddenly by the involvement of important structures, and it would be unwise to assume that because the patient does not waste he will not die soon.

Treatment.—The indications are to support the strength and relieve the symptoms. The former may be accomplished by easily digestible or predigested food (§ 212). For the latter consult § 210, Dilatation. For the flatulence and pain, creosote and opium, or morphia hypodermically. Condurango, 30 grains four times a day, was said to be a specific in cancer of the stomach, and papain has also been recommended. Pylorectomy and gastroenterostomy are now successfully performed. X rays sometimes diminish the pain, and possibly retard the growth of the neoplasm.

VI. *In addition to other symptoms of* CHRONIC INDIGESTION, *the patient who has been, perhaps, the subject of chronic alcoholism, or cardio-pulmonary disease*—VOMITS MUCUS IN THE MORNING, *sometimes streaked with blood.* The disease is probably CHRONIC GASTRITIS.

§ **209. Chronic Gastritis** may be defined as a form of chronic indigestion due to parenchymatous inflammation (*i.e.*, chiefly of the glands) of the stomach.

Symptoms.—(1) Pain coming on shortly after food, usually of a dull character, and attended by tenderness on pressure. (2) Mucous vomiting in the morning, or, indeed mucus found in the stomach contents at any time, is a very characteristic feature of chronic gastritis. Streaks of blood are occasionally present. (3) Thirst is also a prominent feature. (4) A slight degree of pyrexia is sometimes present. (5) The appetite is usually good, but the first few mouthfuls of food satisfy. (6) Flatulence, and other symptoms, as in *atonic dyspepsia* (§ 204). (7) General symptoms are invariably present—depression, nervousness, anæmia, loss of flesh, sallowness, and other symptoms referable to the causes of the condition (see below). Chronic gastritis may constitute

an early phase of cancer—a fact which it is well to remember; the loss of appetite is then very marked.

Diagnosis.—(1) *Atonic dyspepsia*, which has no tenderness on pressure, and no mucous vomiting in the morning; and (2) *cancer*, is differentiated in Table XVI., p. 303.

Etiology.—(1) Persistent dietetic errors, especially *alcoholic excesses*. (2) Venous congestion, arising either from cirrhosis of the liver, or from heart disease. (3) It may be a sequence of repeated attacks of acute gastritis. (4) Constitutional debility, such as that in Bright's disease, gout, etc., may predispose; and so also may (5) Local causes, such as cancer, ulcer, stricture of the pylorus.

The *Prognosis* depends a good deal on the cause and the duration of the symptoms. The case is more grave when due to irremovable venous obstruction. If the disease remain long untreated, the stomach becomes dilated, the walls fibrous, and the glands impaired or destroyed. There are three stages: First, simple *congestion*, in which the pepsin is normal in amount but the hydrochloric acid is diminished, and lactic and fatty acids are found. The second stage is one of *mucous catarrh*, in which there is a large secretion of mucus, hydrochloric acid is almost completely absent, and very little pepsin is present. In the third stage there is *atrophy* of the mucous membrane. In this stage both hydrochloric acid and pepsin are absent.

Treatment.—(1) Here again a correct diet is the most important indication (§ 212). Give small quantities of *dry* food at long intervals (six or more hours). Alcohol and condiments should be stopped, and smoking must be interdicted. (2) The medicinal indications in the first stage and in mild cases are (i.) to promote the flow of gastric juice and stimulate the stomach power by bitters, gentian, quassia, nux vomica, capsicum, and carminatives; (ii.) stimulate the secretion of the stomach by alkalies and bitters given before meals. (3) Symptomatic treatment: For the pain, bismuth, magnesium carbonate, and opium; for fermentation and acidity, alkalies, two or three hours after a meal. Mucous vomiting is relieved by draughts of hot water, with alkalies, before breakfast. If the appetite is absent, give carminatives and bitters; if it is too keen, give bismuth and magnesium carbonate. In the latter condition bitters are harmful, as they excite the nerve-endings in the stomach. In the later stages the indications are (i.) to replace the absent gastric secretion, which is done by giving pepsin, hydrochloric acid, and predigested foods; (ii.) to prevent fermentation, give alkalies and antiseptics along with meals, such as creosote, carbolic acid, and sulphocarbolate of sodium.

(*c*) *The patient presents all the symptoms of* CHRONIC INDIGESTION, *and on physical examination there is* SPLASHING, *or the* AREA OF THE STOMACH RESONANCE *is increased, or there are* FOOD RESIDUES

before breakfast. The disease is probably GASTRIC ATONY or DILATATION.

§ **210. Gastric Atony and Dilatation of the Stomach** are conditions which may accompany or succeed many of the preceding disorders. Gastric Atony, the importance of which has been previously referred to (§ 198) is insufficiency of the power of the stomach to empty itself, independently of pyloric obstruction.

(*a*) GASTRIC ATONY (Motor Insufficiency) may, it appears, exist in three stages or degrees. (α) In simple *loss of tonicity* the stomach is able to empty itself, but there is delay, and splashing can be elicited during the period of digestion, which is prolonged. Many of these cases are latent, and exhibit no symptoms for a considerable time. (β) *Stagnation myasthenia gastrica*, where the stomach cannot empty itself before the next meal, though it does so during the night. (γ) *Retention myasthenia gastrica*, or true dilatation, in which the stomach cannot empty itself during the night, and at all times contains food residues, even when examined by the tube before breakfast. The symptoms of gastric atony are (1) prolonged lassitude after meals, with other symptoms of delayed digestion and atonic or irritable dyspepsia (§ 204); (2) "splashing" several hours after a meal (§ 197); and (3) on percussion or ausculto-percussion some hours after a meal there will be an enlarged area of resonance, particularly to the left of the middle line. This test may be aided by the patient taking a draught of some aerated water, or a solution of sodium bicarbonate, 53 grains, followed by tartaric acid, 45 grains, in solution (which generates at the body temperature just 1 litre of CO_2), or by inflation of the stomach by a suitable apparatus. (4) The methods mentioned in § 198 are tedious, but useful in difficult cases.[1]

(*b*) GASTRIC DILATATION may be a consequence of gastric atony, or due to pyloric obstruction. Its symptoms are (1) the same as those of gastric atony in a more marked degree; and (2) definite food residues found in the stomach before breakfast, without which one would not be justified in believing that a condition of permanent dilatation existed. In all cases of suspected dilatation the stomach should be examined by the tube in the early morning, after a long fast. This also gives an important clue to the substances in which digestion is defective. (3) Visible peristaltic movements in the epigastric region may sometimes be seen when the dilatation is due to pyloric obstruction. (4) One of the most characteristic symptoms of dilated stomach due to pyloric obstruction is the vomiting, at intervals of two or three days or more, of large quantities of acid frothy material, containing sarcinæ (Fig. 62), on which a scum forms on standing. Vomiting may be altogether absent, but if it is present and has these characteristics we may be

[1] See also an important discussion on Gastric Atony at the Brit. Med. Assoc., 1902, the *Lancet*, August 2, 1902.

satisfied that there is dilatation. (5) The remaining symptoms vary with the *cause*, of which there will be a history, or evidence at the time (*infra*). (6) Autotoxic symptoms invariably ensue—marked lassitude, and various other functional nerve symptoms ; sometimes urticaria and other eruptions. Tetany is one of the sequelæ in severe cases.

Etiology.—Dilatation of the stomach may be a consequence of one of two conditions—ATONY OF THE MUSCULAR TISSUE (*a* and *g* below), OR PYLORIC OBSTRUCTION.

(*a*) Gastric Atony may occur after prolonged overfeeding, " bolting " the food in early life, alcoholism, chronic dyspepsia (and its causes), or chronic gastritis. Rheumatism, enteric, influenza, and other acute infections have also been mentioned ; and there is no doubt that states of general debility and anæmia, such as are associated with phthisis (especially when combined with excessive feeding) and neurasthenic conditions, markedly predispose.

(*b*) Obstruction due to a growth of scirrhus cancer at the pylorus is one of the commonest causes, and it may produce the most pronounced dilatation (§ 208).

(*c*) Pyloric obstruction may also occur from the cicatrisation of a simple ulcer of the stomach. The age and sex of the patient and her previous history are characteristic (§ 207).

(*d*) Pyloric obstruction may be due to pressure from without—*e.g.*, enlarged glands in the fissure of the liver, etc.

(*e*) Pyloric obstruction due to a band of adhesion is rare, and difficult to diagnose. It can only be recognised by the exclusion of other causes, and the history of inflammation of the peritoneum.

(*f*) Congenital hypertrophic stenosis, see § 191.

(*g*) Acute dilatation of the stomach is a rare variety that is often difficult to recognise. It may come on more or less suddenly in early life, or in states of general weakness, with symptoms of collapse, resembling intestinal obstruction. It is a serious condition.

The *Diagnosis* of a markedly dilated stomach is not difficult ; the chief question is as to its cause. But the diagnosis of simple atony or myasthenia is always problematical unless the stomach tube or chemical tests be employed.

Prognosis.—It is always a troublesome malady, especially in cases of incurable stricture of the pylorus. Even in atonic dilatation the cure is very tedious, but the prognosis is ultimately good if the disease be diagnosed early, and the cause removable. Malignant stricture is the commonest cause of pyloric obstruction, and unless dealt with surgically is fatal.

Treatment.—The indications are : (1) To keep the stomach as empty as possible. This may be done by diet No. II., p. 312, or by washing out the stomach.[1] It should be done last thing every night. According to

[1] Method, see § 199. Sometimes Turck's double tube is used, the efferent being wider than the afferent tube, to prevent overdistension.

Dr. Herschell, it is best to use plain water. Give concentrated or predigested foods with very little fluid. Never give carbohydrates and animal foods together. Carlsbad salts carry off much of the residue lying in the stomach when taken every half-hour in the early morning until purging ensues (F. 46 or 51). (2) Give tone to the muscular wall by electricity. (3) Promote digestion (*vide* Chronic Dyspepsia). (4) To prevent fermentation, the symptoms of which are very troublesome, carbolic acid (1 to 3 minims), thymol (5 grains) or sodium sulphocarbolate (20 grains), given preferably in a tumbler of water between meals. After lavage, creosote or calomel ($\frac{1}{6}$ grain t. d.) may be given with advantage. Surgical treatment may be needed in cases due to pyloric obstruction, and pylorectomy or gastroenterostomy have been successfully performed.[1]

Electricity is of great use in dilated stomach, not only for giving tone to the muscular wall, but also for promoting digestion and general nutrition. Faradism may be used, preferably with Einhorn's intragastric electrode, but the author has obtained very good results by means of galvanism applied externally.

§ 210*a*. **Neurasthenic Dyspepsia** (synonym : Gastric Neurasthenia[2]) is probably a form of gastric atony. We have seen that the nervous system may be seriously deranged as a consequence of gastric disorder, and the opinion is rapidly gaining ground that the motor, and probably the secretory, powers of the stomach may fail as a consequence of functional nervous disorder. The symptoms do not differ materially from Chronic Atonic Dyspepsia due to other causes, as far as our present knowledge goes. Such cases must be recognised by the circumstances under which they occur. Electricity is especially useful in their treatment.

§ 211. **Gastroptosis** (Dropping of the Stomach ; Enteroptosis), a condition in which the stomach has dropped from its position, has attracted some attention lately. The symptoms and signs are apt to be confused with Gastric Dilatation, indeed, it is only to be distinguished by X rays, after ʒi of bismuth, or inflation. An aggravated state of neurasthenia is usually associated with the condition.

Dietaries and Invalid Foods.

§ 212. Less food is required in old age than in youth, and with a sedentary life than with an active or outdoor one. For a person in health three meals a day are usually sufficient; but when a man is unable, from illness, to take more than a very small quantity at a time, he may require to take more frequent meals. Dietetic errors are a fruitful source of dyspepsia and gastritis. Too frequent meals, habitual overfeeding, and irregularity of the meals will in time derange any stomach. Deficiency of food, and long restriction to the same kind of food, induce dyspepsia by affording no stimulus to excite the secretions ; and in this connection it is well to remember that a frequent cause of failure on

[1] See Clin. Soc. Trans., 1899-1900. See also an excellent account of the surgery of dilatation of the stomach by Leonard A. Bidwell, *West London Medical Journal*, April, 1900.

[2] The terms "gastric neurasthenia" and "dyspeptic neurasthenia" should be reserved for Neurasthenia of Gastric origin, as explained in the author's Clinical Lectures on Neurasthenia.

the part of the physician to cure dyspepsia is his disregard of this latter fact. It is a good rule to start treatment by cutting down the amount rather than by entirely prohibiting the use of certain articles of diet. Too frequent a use of condiments, spices, and tea, and of alcohol especially, lead to chronic gastritis; while dyspepsia is induced by imperfect mastication, bolting of meals, too much fluid with meals, hard mental or physical work immediately after eating, too cold or too hot food, or food which is badly prepared. Excess of tobacco-smoking is certainly a cause of dyspepsia. Greasy and fried foods are bad in dyspepsia, because the gastric juice cannot penetrate the coating of fat. "Well-made" pastry and other so-called rich foods are a source of dyspepsia only when taken with much proteid food. Hyperchlorhydria is induced by constant proteid overfeeding.

Without appropriate dietetic rules our best efforts may fail, especially in the treatment of gastro-intestinal disorders, and other diseases which depend on the proper elaboration and assimilation of food. A few specimen dietaries will therefore be given, culled from various authors, or my own experience. These will serve as a basis for any number of other dietaries.

I. The following table is given as a guide to aid in the drawing up of a diet for mild cases of **atonic dyspepsia** or **chronic gastritis**: *Breakfast.*—Boiled sole, whiting, or flounder; or a slice of fat fried bacon or a soft-boiled egg; a slice of dry toast with a little butter, or of brown bread (not new) and butter. *Beverage.*—One cup of cocoa or of milk and water, sipped after eating. *Luncheon.*—Chicken or game, with bread, and a little tender, well-boiled vegetable, such as spinach, vegetable marrow, or young French beans. *Beverage.*—Half a tumbler of water sipped *after* eating. *Afternoon Tea.*—A cup of cocoa or of weak tea with milk, and a slice of brown bread and butter. *Dinner* (two courses only).—Fish of the kinds allowed for breakfast, with a little mashed potatoes or potato chip. For sweets and dessert, a plain biscuit will suffice. Or a slice of any tender meat, such as saddle or loin of mutton, or the thick part of an underdone chop; a little mashed or grated potato; a spoonful of any plain milk pudding, to which may be added some stewed fruit. *Beverage.*—Half a tumbler of water, with from one to two tablespoonfuls of spirit if desired.

Condiments and stimulants are good in atonic dyspepsia, but must be avoided in chronic gastritis, as tending to cause further irritation of the mucous membrane. The patient should abstain from salted and cured meats,[1] tinned foods, sweets, pastry, raw vegetables, cheese.

II. The "**Salisbury**" **diet** consists essentially of the administration of nitrogenous food only, the meals being taken almost without fluid, but a quantity of hot water being taken between meals. There are several principles involved in this treatment. In the first place, it is obviously a marked change from a person's ordinary diet, and the principle of "relativity" is introduced. Secondly, the solid food administered is in a highly concentrated form, and gives the stomach a considerable rest from its functions while the diet is administered. Thirdly, there is the elimination of the farinaceous and bulky substances which readily decompose and produce flatulence and kindred troubles. Fourthly, by reason of the dryness and small bulk of the food, a dilated or atonic stomach is enabled to resume its normal dimensions, much in the same way as bleeding will relieve a distended heart. The details of the diet are comparatively simple. One pound

[1] Niemeyer, however, reports the cases of a few patients who voluntarily at times restricted themselves to a diet of salted and preserved meats whenever dyspeptic symptoms arose. Probably the success in these cases was due to the fact that such meats are not readily decomposed.

(1 lb.) of lean butcher's meat, chopped or scraped very fine, and so as to rid it of its white fibrous tissue, and lightly cooked, is taken per diem, divided into four or more meals. Occasionally a little well-toasted or twice baked (Zweibach) bread is allowed also. For a change, ½ pound of fish may be substituted for an equal quantity of meat. The meals are taken quite dry, or 2 ounces of fluid only ; but two hours later ½ to 2 pints of hot water is sipped.

III. **Diet for Chronic Rheumatism** (after Burnett[1]).—All rich, sweet, and complicated dishes should be avoided, and also all cured meats, dried fish, pies and pastry, or sweets. *Breakfast.*—Fat bacon or fat ham, brown bread and butter, with a cup of cocoa and milk. *Luncheon.*—Fresh fish with melted butter, or chicken, tripe, or calf's head, with mashed potato and well-boiled green vegetables. *Afternoon Tea.*—A cup of tea or of cocoa with milk, and a slice of thin bread and butter. *Dinner.*—If fish has been taken at luncheon, any plain meat once cooked may be taken at dinner, with a little mashed or grated potato and some well-boiled green vegetables ; any plain pudding, or cream.

IV. **Diet for Obesity** (§ 18).—*Breakfast.*—Fish, bacon, beef, or mutton (6 ounces) ; one breakfastcupful of tea or coffee without milk or sugar, and one small hard biscuit, or one ounce of dry toast. *Dinner.*—Fresh white fish, beef, mutton, lamb, game, or poultry (6 ounces) ; green vegetables ; one slice of dry toast ; cooked fruit without sugar. *Tea.*—A cup of tea without milk or sugar ; a biscuit or a rusk ; 2 or 3 ounces of cooked fruit. *Supper.*—Meat or fish (about 3 ounces) with toast. If desired, a glass or two of sherry or claret may be taken.

V. **Dietary for Diabetes Mellitus** (Sir William Roberts).

Allow—Butcher's meat, poultry, game, and fish ; cheese, eggs, butter, fat and oil ; broths, soups, and jellies made without meal and sugar ; cabbage, endive, spinach, broccoli, Brussels sprouts, lettuce, spring onions, water-cress, mustard-and-cress, celery. For bread is substituted—bran-cake, gluten bread (and meal), almond meal, rusks, and biscuits ; also " torrified " or charred bread ; dry sherry, claret, light bitter ale, brandy, and whisky in small quantities ; tea, coffee (without sugar), chocolate (made with gluten meal), soda-water, bitartrate of potash water.

Forbid—All saccharine and farinaceous foods, bread, potatoes, rice, tapioca, sago, arrowroot, macaroni, etc. ; turnips, carrots, parsnips, beans, and peas ; crabs and lobsters. Liver contains much sugar-forming substances, therefore oysters, cockles, and mussels, which contain relatively large livers, are forbidden. All sweet fruits, as apples, pears, plums, gooseberries, currants, grapes, oranges, etc. ; port, and all sweet wines ; sweet ales and porter ; rum and sweetened gin.

VI. **Diet in Chronic Bright's Disease** (Sir Andrew Clark, modified).—*Breakfast.*—A plate of oatmeal, whole wheaten meal, or hominy porridge, with cream or good milk ; bread or toast and butter ; cocoa, tea, or coffee, with plenty of milk added. Or a slice of well-cooked bacon, fish, or fat ham, may take the place of porridge. *Luncheon.*—A little fish, with some melted butter, mashed potato, and green vegetable, biscuit or bread and butter. Or a basin of vegetable soup, a bit of cheese, bread, butter, and salad. Or a milk pudding, with stewed fruit and cream, bread and butter. *Afternoon Tea.*—A cup of tea with milk, a slice of thin bread and butter, or rusk. *Dinner.*—Soup, purée of potato, chicken or rabbit, mashed potato, green vegetables, plain or milk pudding, with stewed fruit. Or boiled fish, butter sauce, a plain entrée with vegetables, milk pudding or shape, stewed fruit or blanc-mange, biscuit or bread and butter, a glass of plain or aerated water. Or fish, soup, game, or poultry, mashed potato, green vegetables, macaroni cheese. *Dessert.*—Ripe fruit. *Beverage.*—A glass of plain or aerated water. *The last thing at night.*—A glass of milk and soda-water.

VII. **Regimen for Lithæmia**, early stages of Cirrhosis, or a tendency to Congestion of the Liver.—(1) Average daily exercise equivalent to six to ten miles walking in the open air. (2) The patient may eat bread, fresh vegetables (well boiled), farinaceous puddings, eggs, cornflour, oatmeal, fish, poultry, butcher's meat in moderation. (3) Everything to be prepared in the plainest manner possible.

[1] " Foods and Dietaries," by R. W. Burnett, M.D. ; London, 1892.

The patient should avoid all rich, sweet, and greasy dishes, butter, fats, and alcohol in any form.

VIII. **Predigested Foods** are indicated in dilatation of the stomach, cancer, and advanced cases of chronic gastritis. Benger's *Liquor Pancreaticus* is the usual ferment employed, because the pancreas contains both a proteolytic and a diastatic ferment. *Taka-diastase* is a valuable aid in the digestion of farinaceous foods. The patient takes it with his food at the commencement of the meal.

1. *Peptonised Milk.*—A pint of milk is diluted with a quarter of a pint of water and heated to a temperature of about 140° F. Two teaspoonfuls of Liq. Pancreaticus, with 20 grains of sod. bicarb., are mixed with it. The mixture is poured into a covered jug, and the jug is placed in a warm situation, in order to keep up the heat. At the end of an hour or an hour and a half the product is raised to the boiling-point. It can then be used like ordinary milk. Peptonising powders are now to be obtained.

2. *Peptonised Beef-Tea.*—Half a pound of finely minced lean beef is mixed with a pint of water and 20 grains of sod. bicarb. This is simmered for an hour. When it has cooled down to a lukewarm temperature, a tablespoonful of the Liq. Pancreaticus is added. The mixture is then set aside for three hours, and occasionally stirred. At the end of this time the liquid portions are decanted and boiled for a few seconds. (3) *Other foods* can be similarly prepared.

4. *Peptonised Nutrient Enemata.*—The enema may be prepared in the usual way with a mixture of milk and gruel, or milk, gruel, and beef-tea. A dessertspoonful of Liq. Pancreaticus is added to it just before administration. Another formula is given in F. 74.

IX. **Tapioca Soup with Cream.**—Take a pint of white stock and pour into a stewpan. When it comes to the boil, stir in gradually 1 ounce of prepared tapioca. Let it simmer slowly by the side of the fire until the tapioca is quite clear. Put the yolks of two eggs into a basin, with two tablespoonfuls of cream. Stir with a wooden spoon, and pour through a strainer into another basin. When the stock is cooled, add it by degrees to the mixture, stirring well all the while, so that the eggs may not curdle. Pour it back into the stewpan, and warm before serving. Add pepper and salt to taste.

X. **Beef-Tea.**—Cut up a pound of lean beef into pieces the size of dice; put it into a covered jar with 2 pints of cold water and a pinch of salt. Let it warm gradually, and simmer for a couple of hours, care being taken that *it does not boil.*

XI. **Improved Beef-Tea.**—Three-quarters of a pound of steak, scraped or passed through a mincing machine, and pounded; ¾ pint of cold water; one piece of sugar, one pinch of salt, one teaspoonful of tapioca; simmered in a "Gourmet Boila" for three hours.

XII. **Artificial Proteid Foods.**—Beef-tea and other meat preparations do not contain the nutritive constituents of meat, except in small quantities, but contain quantities of extractives which may derange the digestion and impair the action of kidneys. *Peptonised albumin* (or peptonised meat) is better, but it is doubtful if the organism in certain states of prostration can reconstruct peptone into albumin, and the taste of peptone is very bitter and nasty. The *albumoses* are intermediate between albumin and peptone. They are freely soluble, tasteless, and readily absorbed and reconstructed into albumin, produce no disturbance of the digestive organs, and do not irritate the kidneys. *Somatose* is a meat preparation of which the albumin is mainly converted into albumose, and Stevenson and Luff[1] have drawn attention to its great value as a nutriment, stimulant, and restorative in debilitated conditions, even when the presence of albuminuria shows the kidneys are deranged. It is a yellow powder, freely soluble and tasteless. *Plasmon* is another artificial proteid food. It is prepared from milk, and contains casein in a soluble form. It is a nutriment of some value.

XIII. **Milk, Egg, and Brandy.**—Scald some new milk, but do not let it boil. Put it into a jug, and the jug into a dish of boiling water. When the surface looks

[1] The *Lancet*, September 30, 1899, p. 885.

filmy, it is sufficiently done, and should be put away in a cool place in the same vessel. When quite cold, beat up a fresh egg with a fork in a tumbler, with a lump of sugar ; beat quite to a froth, add a dessertspoonful of brandy and fill up the tumbler with scalded milk.

XIV. **Chicken Panada.**—Take the flesh from the breast of a freshly roasted chicken ; soak the crumb of a French roll or a few rusks in hot milk, and put this into a clean stewpan, with the meat from the chicken reduced to a smooth pulp by chopping it and pounding it in a mortar ; add a little chicken broth or plain water, and stir the panada over the fire for a few minutes.

XV. **Whey.**—Into a warm milk put sufficient quantity of rennet to cause curdling, and strain off the liquid, which is then ready for use.

XVI. **White Wine Whey** (especially good for infants with summer diarrhœa).—Half a pint of milk is boiled : as soon as it boils, add 2½ fluid ounces of good sherry ; allow the mixture to boil for a few minutes, then leave in a cool place in a basin. When the curd falls to the bottom, carefully pour off the whey, or strain through muslin. In grave conditions, with vomiting, give a teaspoonful every ten minutes ; in inflammatory diarrhœa give a tablespoonful every hour.

§ 212*a*. **Artificial Feeding of Infants.**—General Directions.—Feed the child regularly ; if necessary, wake it for that purpose. Use a boat-shaped bottle, with a rubber teat on the end. Feed slowly, holding the bottle on the slope until the milk in it is finished. Keep the bottle strictly clean by scalding it both before and after it is used. Mix a fresh portion for every meal. Do not overfeed ; 2 pints of the mixture in twenty-four hours is enough for a child under six months. No starchy food should be given to an infant under six months, for the pancreatic secretion is not established till then. On no account keep a baby at the breast after it is nine months old—about six months is long enough.

Under One Month.—Feed every two hours from five in the morning to eleven at night. Start with ¼ ounce of milk to ½ ounce of water, and gradually increase to 1½ ounces. Sugar should be added in the proportion of 1 drachm of milk sugar to 4 ounces of the prepared milk. A small teaspoonful of cream may be given with each feed. When the warm mixture has cooled down, a teaspoonful of lime-water may be added.

From One to Three Months.—Feed every two and a half hours, with quantities gradually increasing up to 1½ ounces of milk to 2 ounces of water.

From Three to Six Months.—Feed every three hours with 2 ounces of milk to 2 ounces of water, gradually increasing strength to 4 ounces of milk with 3 ounces of water.

From Six to Nine Months.—Feed with five meals a day. Milk, 6½ to 7 ounces, with water, 2 ounces. With two of the meals add a tablespoonful of some " infants' " food. A little bread and milk, or porridge, or pudding once or twice a day.

From Nine to Twelve Months.—The bottle may be gradually left off. Morning and evening, 6 ounces of bread and milk, sweetened. *Lunch.*—Milk and water, bread and butter. *Dinner.*—Two ounces of farinaceous milk and egg pudding on alternate days ; a little broth or beef-tea with bread on other days, or meat gravy.

From Twelve to Eighteen Months.—Morning and evening, about 6 ounces of bread and milk, sweetened, and bread and butter. *Lunch.*—Half a pint of milk, bread and butter. *Dinner.*—Bread, vegetables, milk pudding, and milk and water. On alternate days give gravy or broth, with bread-crumb, and milk pudding. *Tea.*—Bread and butter, and milk. Half an egg may be given once a day.

From Eighteen Months to Two Years.—In addition to the last-named diet, give minced meat or fish on alternate days, with finely chopped greens and potatoes. At teatime, cocoa. Mutton and bacon fat, finely chopped, and raw meat juice, are to be recommended for delicate children.

A useful addition to methods of infant feeding is the use of sodium citrate, 2 grains to the ounce of milk.[1] In this way undiluted milk may be used, so that no cream or sugar need be added.

[1] Dr. Frederick Langmead, *Clinical Journal*, July 15, 1908.

CHAPTER XI

THE INTESTINAL CANAL

The physiological importance of the intestinal canal is evidenced by the fact that its length is between 25 and 30 feet, along the whole of which absorption may take place; yet the first feature of intestinal disorders which strikes the student is their inaccessibility to examination. Of late years grounds have been adduced for believing that bacilli or their toxins make their way through the mucous membrane of the intestine into the lymph spaces beneath, and thence into the circulation, particularly when the mucous membrane is unhealthy, abraded, or ulcerated; thus intestinal sepsis constitutes a danger heretofore but little appreciated.[1] In the future, therefore, the bacteriology of the intestinal canal will probably assume considerable importance, and the examination of the stools will take its rightful place.

Another striking feature about diseases of the intestines is the disproportionate amount of prostration which accompanies them—a point to which attention has already been drawn (§ 164). For instance, in a patient who is attacked by a slight but sudden diarrhœa or abdominal pain, the feeling of exhaustion, which in some cases may amount almost to collapse, is out of all proportion to the local mischief. This disproportionate degree of prostration or collapse is especially marked in early life, when "diarrhœa" is, mainly on this account, found to be the principal cause of death in children under two years of age. Again, among the acute specific fevers we find that the most fatal collapse and prostration occur in those in which the chief lesion is in the intestinal canal—in cholera, dysentery, and enteric fever. These facts are possibly accounted for by the circumstance that the chief centre of the sympathetic system (its "brain," so to speak) is found within the abdominal cavity, in close anatomical relation with the intestines which it enervates.

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[1] Compare §§ 169 and 170, Peritonitis, and Dr. William Hunter, Pernicious Anæmia, Path. Soc. Trans., 1901-1902, and the *Lancet*, 1900, vol. i., pp. 221, 296, 371; and 1902, vol. i., p. 1467.

PART A. SYMPTOMATOLOGY.

§ **213.** The cardinal symptoms of intestinal disorder are DIARRHŒA, CONSTIPATION, and ABDOMINAL PAIN.

ABDOMINAL PAIN is frequently present, especially in the more acute conditions, but by no means always ; and abdominal pain may be due to so many other diseased conditions within the abdominal cavity that it has been considered in Chapter IX. (the Abdomen, § 167).

DIARRHŒA is a cardinal symptom of intestinal disorders, and it will be fully discussed in Part C. of this chapter.

The same remarks apply to CONSTIPATION, and in this instance we shall have to distinguish simple constipation from that important surgical emergency, Obstruction of the Bowels. This also will be dealt with in Part C.

The GENERAL or REMOTE symptoms are sometimes, especially in acute cases, of a very severe character, in view of the profound PROSTRATION, which is associated with some intestinal disorders—to which allusion has just been made. PYREXIA is not usually a marked feature in intestinal diseases (see § 165). In the more chronic forms of intestinal disease EMACIATION is apt to ensue in course of time. Various NERVOUS DERANGEMENTS of a neurasthenic order are sometimes, as in gastric diseases, associated with disorders of the intestinal canal, consequent partly on malassimilation and intestinal toxæmia, and partly, no doubt, arising in a reflex manner by intestinal irritation. In rare instances these are of a most distressing nature, and in one case which I have seen, that of a medical man, who was unable to obtain relief, they led to suicide. Reflex symptoms of a less troublesome order—*e.g.*, vague pains, itching of the nose, or bad dreams—may be associated with intestinal parasites and some other intestinal conditions.

PART B. PHYSICAL EXAMINATION.

§ **214.** The physical investigation of the intestinal canal can only be accomplished by two means, the EXAMINATION OF THE ABDOMEN and the INVESTIGATION OF THE FÆCES.

The **Examination of the Abdomen** is not always easy, but it should never be neglected in suspected intestinal disorders. PALPATION and PERCUSSION will enable us to make out any generalised swelling or localised tumour. The tenderness which sometimes accompanies intestinal disorders may also be elicited. *Scybala* are often present within the colon, and must not be mistaken for the hard nodules of cancer or other tumour. Their mobility is a very deceptive feature, and the occasional association of diarrhœa may delude us. Their disappearance after active purgation is the only certain method of diagnosis. The reader is referred to Chapter IX. for further details as to examination of the abdomen.

§ **215. An Examination of the Stools** is always important, and sometimes absolutely necessary for the diagnosis of intestinal disorders. A great deal of information can also be thus obtained with regard to diseases of the other abdominal viscera. The fæces should be examined *first* as to their physical properties—colour, consistence, shape, size, odour, and reaction; *secondly*, for undigested food and other substances, such as mucus, gall-stones, or parasites; *thirdly*, for the presence of blood; and, *fourthly*, a microscopic examination is often necessary. It is only rarely that we can rely implicitly on a patient's statement, even as to the colour and appearance of the stools; and, however disagreeable it may be, we should, when thoroughness is desired, examine the fæces ourselves.

A small portion of the fæces may be brought in a tin box, but it is preferable to see them in bulk, the patient having used a night-stool. He should pass water before going to stool. A large wide-mouthed glass jar, closed at the top by a stopper, is a very convenient receptable for their preservation. Nothing should be added to the motion until the doctor has examined it. Then carbolic solution (1 in 100) may be added if we wish to detect mucus or to preserve the stool.

Physical Properties of the Stools.—1. The Colour of the fæces is normally dark brown. The degree of colour of the stools is a fair measure of the amount of bile which passes into the intestinal canal. When there is diarrhœa the stools, at first, are dark from excess of bile pigments; afterwards they become lighter on account of their dilution by the increased watery exudation and the presence of undigested food. (i.) *Clay-coloured stools* are found in cases of obstructive jaundice. (ii.) *Streaks of blood* may be present. (iii.) *Tarry stools*, of a dark or black colour, are due to the presence of blood which, entering the alimentary canal *high up* (as in cases of gastric ulcer), has undergone "digestion." (iv.) *Black fæces* are seen when the patient is taking iron, bismuth, or manganese internally. (v.) Colourless "*rice-water*" or *milky stools* are met with in cholera, severe dysentery, or severe enterocolitis, due chiefly to the presence of serum. (vi.) In infancy the stools are normally of an *orange-yellow* colour, but in "dyspeptic" diarrhœa or enteritis they are generally *green*.

2. The Consistence of the Stools is normally semi-solid, and the form is that of a rounded cylinder. (i.) When passed in hard, dry, roundish balls they are known as *scybala*. These are generally coated with mucus. Sometimes the irritation they cause sets up a false diarrhœa, and there are alternating conditions of diarrhœa and constipation, which can only be cured by a course of aperient medicines. (ii.) In typhoid fever the stools often present the appearance of *pea soup*. The *rice-water* diarrhœa of cholera has just been referred to. (iii.) In cases of stricture of the rectum—*e.g.*, from syphilis or cancer—the stools are *ribbon-like* in shape, and this forms an important diagnostic indication.

3. The Odour of the stools, which is due to skatol, does not give us

much information. There is a characteristic gangrenous odour in severe ulceration—syphilitic, cancerous, or dysenteric. An ammoniacal odour is never met with in human fæces. If this odour be present it can only arise from the presence and decomposition of urine.

4. The REACTION of the stools is normally feebly alkaline when first passed, but they may be acid, a feature which is common in infancy. In the course of a few hours the stools undergo acid fermentation.

VARIOUS SUBSTANCES may be found—

1. UNDIGESTED PARTICLES OF FOOD, if in excess, are indicative of imperfect digestion (gastric or intestinal), and, unless the food has been excessive, denote more especially intestinal or pancreatic disease (see also p. 324). In children this feature usually indicates over-feeding. Small, hard concretions, consisting of phosphates and other matter, are sometimes found, and may form the source of appendicitis.

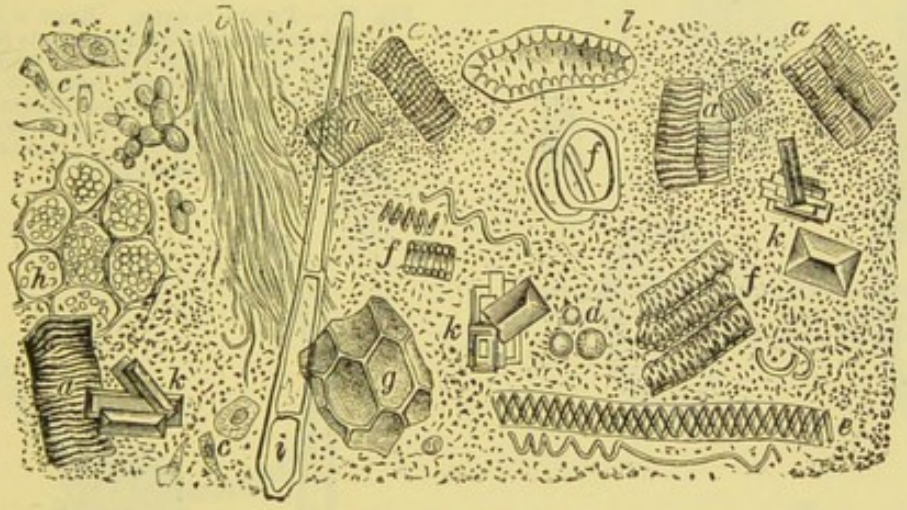


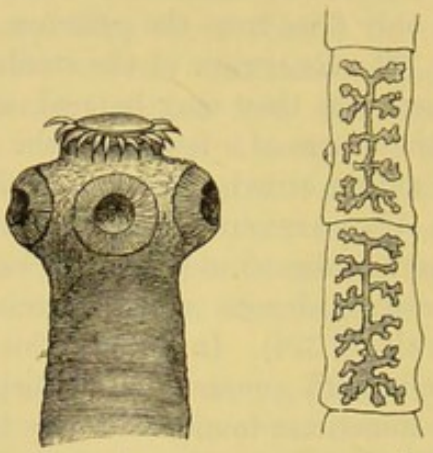
Fig. 63.—MICROSCOPICAL EXAMINATION OF FÆCES (after Von Jaksch).—Normal appearance × about 350. *a*, muscle fibres; *b*, connective tissue; *c*, epithelium; *d*, white blood cells; *e*, spiral vegetable cells; *f* to *i*, various vegetable cells; *k*, triple phosphate crystals in a mass of micro-organisms; *l*, diatoms.

2. MUCUS in the fæces is often overlooked unless specially sought for. To discover it satisfactorily *water must be added* to the fæces, when any mucus present will be seen floating about like small pieces of jelly. The presence of mucus in small amount is of no consequence; it is usual in constipation. When in quantity, and intimately *mixed with the fæces*, it indicates catarrh of the *small* intestine. When in *isolated masses* it signifies the presence of catarrh of the large bowel. In membranous, or mucous, colitis, *long cylinders* of mucus are passed, sometimes without much fæces. These cylinders are generally swarming with *B. coli*, which infest the colon.

3. BLOOD in the stools may appear either as streaks or in quantity, when from rectum or large bowel. If it comes from the stomach or small intestines it may have undergone partial digestion and give to the stools a tarry appearance (melæna). In either case it reddens the

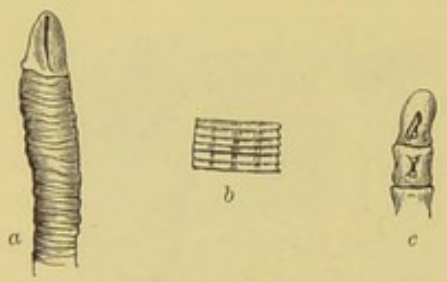
water in which the stool is placed, and gives the characteristic spectrum.[1] The *causes* are dealt with below (§ 224).

Fig. 64.—HEAD OF TÆNIA MEDIOCANELLATA.—Magnified about 10 times.



a b

Fig. 65.—TÆNIA SOLIUM. Head × 30 (*a*); and proglottides (*b*) or segments (slightly enlarged). In the latter the uterus has seven to ten lateral branches which *ramify*. But in T. Mediocanellata there are twenty to thirty lateral branches, which in turn terminate in two branches (dichotomous branching).



a b c

Fig. 66. — BOTHRIOCEPHALUS LATUS. Natural size. Head (*a*); proglottides (*b*); and tail (*c*).

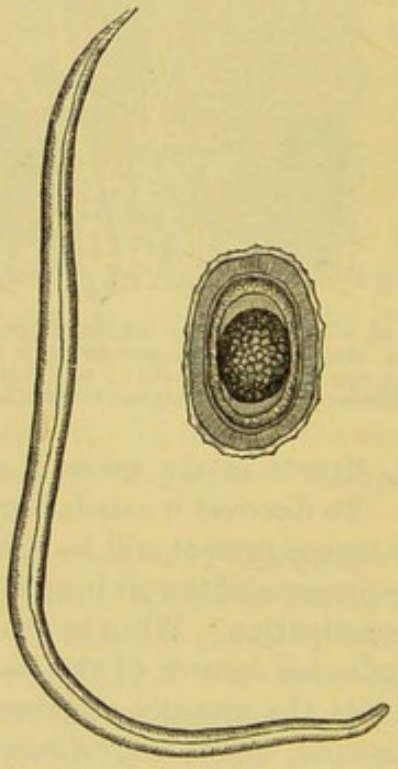


Fig. 68.—ASCARIS LUMBRICOIDES (Round Worm). About half normal size, and EGG × 75.

a

Fig. 67.—Female OXYURIS VERMICULARIS. × about 3 times. *a*, natural size, about $\frac{1}{8}$ inch.

4. PUS always indicates *ulceration* of the rectum or colon, which may be of syphilitic, cancerous, tuberculous, or dysenteric origin (§ 221).

[1] Cases have lately been recorded where, after standing for some time, the fæces developed on the exposed surface a colour resembling blood, but no blood was

Pus is difficult to detect when diarrhœa is present. When in large quantity, pus indicates an abscess bursting into the bowel, such as a pelvic or ischio-rectal abscess.

5. GALL-STONES may be found by mixing the stools with water, and passing the mixture through muslin or a fine sieve. Gall-stones sink in water when recently passed, though they float when dried. They are very friable, and any suspicious particles should be examined under the microscope for cholesterin, see p. 362.

6. WORMS, such as tapeworms and threadworms, may be found. It is of great importance to find the head of the tapeworm which is about

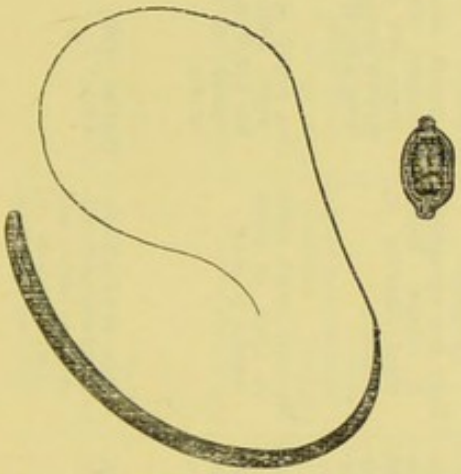


Fig. 69.—TRICOCEPHALUS DISPAR ("Whip-worm").—Magnified by 3, and EGG magnified about 100.

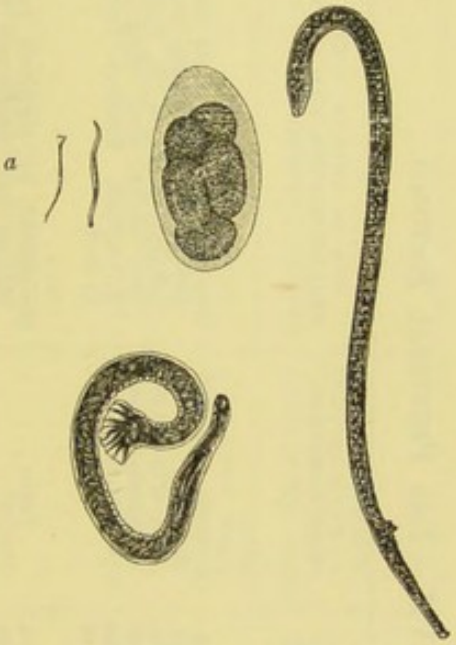


Fig. 70.—ANKYLOSTOMUM DUODENALE (male (smaller) and female).—Worm and egg × 175. *a*, natural size. The lower one is from a microscopic specimen for which the author is indebted to Dr. W. J. Tyson of Folkestone.

the size of a pin's head. This may be done by the method just described. Another method is, to mix the fæces with water and let the mixture stand. As the parasite sinks to the bottom the supernatant fluid should be carefully poured off, and more added, the process being repeated until the fluid becomes colourless. The various worms are given in the Table XVII.

Microscopic Examination of the fæces is often necessary, especially to find the ova of parasites. Place a small portion of the stool upon a slide, and if not sufficiently fluid, dilute with a quantity of normal saline solution ; cover and examine. If dysentery is suspected, the stool must be examined whilst still warm. NORMALLY, under the microscope (Fig. 63), the stool shows undigested particles of

detected by the spectroscope. It appears that in certain as yet unknown conditions some pigment is present in the fæces, which on exposure to the air becomes red like blood.—Carter and McMann, *Lancet*, November 25, 1899, p. 1432.

Table XVII.—The Principal Entozoa.

NAME.	CHIEF CHARACTERISTICS OF ANIMAL, AND WHERE FOUND.	CHIEF SYMPTOMS.	OVA OR EMBRYO; CHIEF CHARACTERISTICS, AND WHERE FOUND.	REMARKS.
Fam.: Cestodes.				
Tænia Mediocanellata. (Tapeworm in man). Fig. 64.	14 to 24 ft. long. Head, 4 suckers, no hooklets. Segments, over 1,000, show central stem uterus with 20 to 30 lateral *dichotomous* branches. Fastens itself to mucous membrane of intestinal canal in man.	Of reflex irritation, digestive or nervous disorders. Segments passed per rectum.	Recognised by segments containing ova discharged from alimentary canal in fæces. Ova more oval than in T. solium. Embryo found in beef.	Cattle the intermediate hosts. Found in Great Britain.
Tænia Solium. Fig. 65. (Tapeworm in man.)	About 10 ft. long. Head, 4 suckers, and row of 26 hooklets. Segments, about 850, show central stem uterus with 7 to 10 lateral *ramifying* branches. Fastens to mucous membrane of intestinal wall in man.	Ditto.	Recognised by segments containing ova discharged per rectum. A six-hooked embryo inside ovum; which eaten by pig, bores its way into the flesh.	Pig the intermediate host—"measly pork," where scolices abound in flesh. Animal takes 3 months to develop in man.
Bothriocephalus Latus. Fig. 66.	16 to 25 ft. long. Head club-shaped, with long lateral slits. No hooklets or suckers. About 3,000 segments; uterus, rosette-shaped. Found in intestinal canal of man.	Very few symptoms in adults. Intestinal disorder in children.	Segments containing ova discharged per rectum. Sometimes ova discharged alone; brown shelled; $\frac{1}{3\frac{1}{5}}$ in. long; with a lid at one end.	Ova hatched on reaching water, and eaten by fish, which act as intermediate host. Chiefly found in Switzerland and other parts of Central Europe.
Tænia Echinococcus. (Hydatid cyst in man; tapeworm of dog.) Fig. 75.	$\frac{1}{8}$ in. to $\frac{1}{4}$ in. long. Head pointed, with 4 suckers; double row of hooklets. Has 4 segments, the 4th longer than all others. Found in intestinal canal of dog or wolf.	Hydatid cysts form in liver, or other organs, in man.	Ova found in fæces of dog or wolf. Embryo reaches man by drinking water, and becomes encysted in various organs.	Man is the intermediate host; dog or wolf—the host. Man receives the embryo by drinking contaminated water—*e.g.*, mountain streams.
Fam.: Nematodes.				
Oxyuris Vermicularis. (Threadworm.) Fig. 67.	F. = $\frac{1}{2}$ in.; M. = $\frac{1}{4}$ in. in length. Found in large intestine, chiefly the rectum.	Reflex irritation. Worms tend to migrate at night, and cause itching of anus and genitals.	Worms easily seen after aperients.	Often trouble children. Found in all countries.

Ascaris Lumbricoides. (Round worm.) Fig. 68.	M. about 6 in. long; F. 12 in. Found in small intestine of man.	Reflex irritation, nervous and digestive.	Oval, $\frac{1}{25}$ in. to $\frac{1}{15}$ in. diameter. Hard, dark shell *with excrescences*. Found in faces.	Worms tend to migrate, and are sometimes vomited. Frequent in children. Are found in all countries.
OTHER FAMILIES.				
Tricocephalus Dispar. (Whip-worm.) Fig. 69.	1½ in. long. Anterior part fine and thread-like. Found in caecum.	Few symptoms.	Ova elliptical, with a projection at each end. Dark coloured, $\frac{1}{30}$ in. long. Found in faeces.	Confined to Egypt.
Ankylostomum Duodenale. Fig. 70.	F. = $\frac{1}{3}$ in.; M. = ½ in. Tail end broadest; mouth capsule distinct and provided with 4 tentacles, which clasp the villi of the sm. intestine. Found in jejunum.	Melaena, profound anaemia and weakness.	Ova about $\frac{1}{50}$ in. Clear and transparent shell, showing yolks through. Worms found in faeces.	Found in Egypt, Brazil, Jamaica, and Mining districts.
Filaria Sanguinis Hominis. (Filaria Bancrofti.)	3 or 4 in. long; fine and hair-like, lying in bunches together. Found in lymphatics.	Chyluria.	Embryos $\frac{1}{6}$ in. long, enclosed in thin envelope. Tapering tail and round head. Found in blood at night.	Mosquito acts as intermediate host, by taking embryo from blood. Chiefly exist in India and tropics.
Bilharzia haematobia. (Distoma haematobium.) Fig. 90.	M. = 15 mm. long; cylindrical, with gynephoric canal. F. = 20 mm. Found in blood of portal system, etc.	Hæmaturia and melæna.	Ovum has one terminal spine. Oval, 0·16 mm. long. Found in bladder and urine, and in faeces.	Intermediate host probably a fresh-water mollusc. Man gets parasite by water. Egypt, Africa, and Brazil chief countries.
Trichina Spiralis.	M. = $\frac{1}{6}$ in.; F. = ¼ in. Lives a few weeks in intestine of man, and then embryos migrate to muscles.	1st stage, gastro-intestinal irritation; 2nd stage, fever, tenderness of muscles, œdema. Often death in 5 weeks.	Larval form bores through alimentary canal, and is found coiled up within a cyst, in muscular tissue. Adult worm and occasionally embryo found in faeces.	Man gets parasite from uncooked pig flesh. Rabbits, mice, rats, sheep, also get the parasite. Found in Germany; rare in England.
Filaria Medinensis. (Guinea worm.)	F. = 13 in. to 30 in. Cylindrical, white, smooth. Found in subcutaneous tissues of limbs, especially the feet.	Local inflammatory symptoms; later, constitutional symptoms.	Embryo flattened, $\frac{1}{5}$ in. long, with long thin tail.	Presumably acquired by drinking water, which contains intermediate host. Found in Asia, Africa, Egypt, Brazil. Intermediate host—a fresh-water cyclops.

food, especially starch granules, muscle fibres, connective tissue, and fat cells; crystals of fatty acids, oxalate of lime, and other calcium salts. Hæmatoidin, phosphates, cholesterin, and Charcot-Leyden crystals are rare. Among the bacteria the Bacillus coli communis, various unnamed bacilli, cocci, and yeast are found. Blood corpuscles and intestinal epithelial cells may occur in small amount.

Among the ABNORMAL constituents which should be looked for are, *first* and chiefly, the presence of the ova or segments of the different entozoa (see below, § 215*a*).

2. Among the *undigested food products* an excess of undigested starch or of muscle fibre indicates disease of the small intestine or pancreas. An excess of fat in the fæces indicates either (i.) deficient bile secretion, or (ii.) disease of the pancreas.

3. The *Charcot-Leyden* crystals are the only abnormal crystals of any importance. They are very rare, and are found chiefly in association with worms, especially ankylostomum. Their presence is a useful indication that the parasite is still alive in the intestinal canal.

4. Various *bacilli*, such as those of typhoid and cholera, are present in the fæces in disease, but on account of the many extraneous microbes it is almost impossible to obtain specific cultures from the stools. The *b. coli* has its normal habitat in the colon.

5. The *amœba* of dysentery is more characteristic, and is now regarded as the pathognomonic feature of true dysentery. It was first found in the stools, and described by Lösch in 1875, and termed by him the amœba coli. They are generally found in fair abundance as roundish cell-like bodies of irregular oval form, which continually undergo amœboid movements. Their size varies from 10 to 20 μ. Koch was the first to suspect a definite relationship between dysentery and these organisms. Cunningham claims to have found them in cholera patients in Calcutta, and they are now generally believed to be the cause of hepatic abscess.

§ 215*a*. **Various Intestinal and Other Parasites,** or segments of them, or their ova, may be found in the fæces. These are described in Table XVII., and the accompanying illustrations. Seven of these *infest the alimentary canal of man:* The two common tapeworms (T. Solium and T. Mediocanellata), recognised by their segments in the fæces, naked eye; the tapeworm of central Europe (Bothriocephalus Latus), recognised by its segment, naked eye; the extremely common threadworm, (Oxyuris Vermicularis); and the common round worm (Ascaris Lumbricoides,) both of which may be seen by the naked eye, the former like small pieces of cotton, the latter as large as a garden worm; and two worms which are found mainly in Egypt, the Ankylostomum and the Tricocephalus, both of which, with their ova, need magnification for discovery. The symptoms and treatment of the commoner of these are given in § 226. The eggs of the Bilharzia are sometimes found in the fæces, enclosed in small fleshy masses, but they are chiefly found in the urine, associated with *hæmaturia* (§ 300). The ova are quite peculiar in having a spine-like projection at one end only (Fig. 89). Brief reference may here be made to the four other parasites, though they are not found in the intestines or fæces of man. Hydatid cyst, the cystic (alternate) form of the T. Echinococcus is found chiefly in the liver in man. Filaria gives rise to chyluria, and is only found in the blood at night. Trichina Spiralis is found in the muscles in pork-eating countries. Filaria Medinensis is found in the skin in tropical countries.

PART C. DISEASES OF THE INTESTINAL CANAL, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 216. **Routine Procedure, and Classification.**—Having first ascertained that the patient's LEADING SYMPTOM is referable to the intestinal canal; and secondly, by inquiries into the HISTORY of the illness, whether it came on *acutely* and suddenly or gradually in a *chronic* manner; we proceed, in the third place, to the PHYSICAL EXAMINATION

of the abdomen after the manner set forth in Chapter IX. (§ 166). If, in the course of these inquiries, definite disease is suspected in any particular organ, reference should afterwards be made to the appropriate chapter.

A. If **Diarrhœa** is the leading symptom :

If *acute*, or attended by choleraic or dysenteric symptoms	turn to	§§ 218-220
If *chronic*	,,	§ 221
B. If there is **Tenesmus** without diarrhœa ..	,,	§ 223
C. If **Blood** or some other **alteration in the stools** is the leading feature	,,	§§ 224-226
D. If **Constipation** is the leading symptom ..	,,	§ 227
E. If the **Stoppage in the Bowels** is complete ..	,,	§ 228

§ **217. Diarrhœa** is the frequent occurrence of loose or liquid motions ; it is the watery consistence of the stools which is the chief characteristic in diarrhœa. A frequent call to stool may arise from some local irritation (see Tenesmus), without any alteration in the consistence or form of the stool. This source of fallacy should be carefully guarded against. Many women speak of the tenesmus which sometimes accompanies the menses as " diarrhœa."

Causes of Diarrhœa.

	Acute.	Chronic.
Common.	I. Irritating food.	I. Acute causes becoming chronic.
	II. Water.	II. Local conditions about anus.
	III. Intestinal parasities.	III. Ulceration (colitis, tuberculosis, cancer and syphilis of the bowel).
	IV. Infantile diarrhœa.	IV. Chronic colitis or mucous colitis.
		V. Portal obstruction or congestion.
		VI. Dysenteric diarrhœa.
		VII. Nervous diarrhœa.
Rare.	V. Toxic blood conditions.	VIII. Amyloid disease.
	VI. Acute enteritis or " chill."	IX. Senile diarrhœa.
	VII. Acute ulcerative colitis.	X. Mineral poisons (*e.g.*, arsenic).
	VIII. Some causes of chronic diarrhœa.	XI. Pancreatic disease.
	IX. Dysentery.	XII. Psilosis.
	X. Cholera.	

The fæces should always be examined where it is possible (§ 215). Sometimes the situation of the disease may thus be discovered ; for instance, when the stools are coloured with bile, and contain undigested food, and *small pieces of mucus intimately mixed* with the fæces, catarrh of the small intestine may be suspected. When mucus or " slime " occurs in *larger masses*, in " strings " or " casts," there is probably disease of the large intestine.

§ **218.** In **Acute Diarrhœa** there is usually a good deal of pain and tenesmus (straining at stool) ; the tongue is usually furred, there is thirst, and may be vomiting. If there be much vomiting and prostra-

tion the diarrhœa is probably due either to the presence of some violent irritant, or to some serious organic lesion, such as injury to the bowel or peritoneum. In profuse diarrhœa the temperature is usually subnormal, and the urine diminished. It should be borne in mind that scybala retained in the intestines may give rise to attacks of diarrhœa alternating with constipation.

Causes.—I. The **food** taken, and the vessels in which it has been contained and cooked, should be the first questions in all cases of acute diarrhœa coming on suddenly in a healthy person. Collapse and many of the symptoms of cholera can be produced by food cooked in a new copper vessel. One of the irritant poisons may have been introduced into the food accidentally or designedly. This should be borne in mind; and in cases of sudden and unexplained diarrhœa the physician should go patiently through every article taken at every meal during the preceding twenty-four hours. Over-ripe or decomposing fruit, too much raw vegetable food, tinned meat—especially that which has been long in store, and has undergone a change resulting in the formation of ptomaines (see footnote, p. 287)—shellfish and bad cheese are also possible causes. The first or diarrhœal stage of trichinosis comes under this heading, and should be considered in pork-eating countries. In cases of acute diarrhœa in which trichinosis is suspected the worm should be sought in the fæces, for in the earlier stages of this disease treatment is so much more efficacious. The diarrhœa which precedes the intestinal obstruction caused by intussusception in children frequently follows a heavy meal of indigestible articles; and diarrhœa is itself a cause of intussusception.

II. The quality of the **water** is often responsible for diarrhœa, acute or chronic. This is frequently the case in malarial districts in the summer and autumn, especially when the temperature is high. Water containing much peat from the mountains may also cause diarrhœa; and thus the water supply of the town of Montreal frequently occasions diarrhœa in newcomers.

III. **Worms** may give rise to diarrhœa in children. They may be attended by uneasy abdominal sensations, night terrors, picking of the nose, itching of the anus, but sometimes the worms are discovered in the stools when there have been no symptoms pointing to their existence (§ 226).

IV. **Infantile Diarrhœa** occurs in at least three well-recognised clinical forms: (i.) Acute Dyspeptic Diarrhœa; (ii.) Inflammatory Diarrhœa or Entero-colitis; and (iii.) Epidemic Diarrhœa or "summer diarrhœa" (including Infantile Cholera)—mentioned in progressive order of severity.

(i.) In Acute Dyspeptic Diarrhœa the stools are offensive, frothy, of a greenish colour, and mixed with curds of undigested food. Vomiting may or may not be present. It is usually a transient condition if adequately treated.

(ii.) In Infantile Inflammatory Diarrhœa (Entero-colitis) the stools are green, slimy and often contain blood; there is some fever at the beginning, and abdominal distension. The inflammation attacks chiefly the colon, consequently there is tenderness on pressure over the region of the colon, and mucus in the stools. Adults also are sometimes affected. It lasts only one to three weeks if treated as described below.

(iii.) Epidemic Diarrhœa ("summer" or "autumnal" diarrhœa of children) is met with chiefly in childhood and infancy in the autumn months of the year, and is attended by catarrh of the mucous membrane of the bowel. The *symptoms* of a severe attack are: Watery stools, foul-smelling, of altered colour, containing lumps of mucus; vomiting; acute abdominal pain and tenesmus; prostration, collapse, subnormal temperature, with pinched aspect, rapid wasting, and often (after a course of a week or so) death from exhaustion. Infantile Cholera forms about 2 per cent. of "summer diarrhœa" cases. The stools are serous, persistent vomiting is a marked feature; great collapse rapidly supervenes, the temperature in the rectum is raised as in adult cholera, and death rapidly supervenes: some describe it as a separate affection, but it is probably a severe form of Epidemic Diarrhœa.

Etiology of Infantile Diarrhœa.—Any of the previously mentioned causes (I. to III.) are contributory, and very often exciting, causes in all forms of infantile diarrhœa; and especially dietetic errors. This latter is the sole cause in variety i., and probably in variety ii. These diseases affect chiefly hand-fed children, in warm weather, being probably in part due to dirty feeding-bottles, teats, sour milk, etc. Most of the cases occur in children under six months old. The causes of *Epidemic Diarrhœa* are far more obscure. (i.) Seasonal, epidemic, and microbic causes[1] have long been suspected on account of its prevalence during the summer and autumn months. It occurs chiefly after hot, dry summers, and the researches of Ballard seem to point to its being dependent upon some telluric condition. (ii). It occurs chiefly in towns, and certain *localities—e.g.*, Leicester—are notorious for a lethal epidemic each summer and autumn. Ballard found that the severity of the annual outbreak seemed to vary with the subsoil temperature; it started when the 4 feet earth thermometer read 56° F. Adults do not altogether escape the influence of these causes, and diarrhœa is widely prevalent in the hot, dry summer months in some years; but in children the death-rate is sometimes appalling.

In the *Treatment of Infantile Diarrhœa* astringents are not only useless, but harmful. Equal parts of lime-water and castor oil (F. 64), every two or three hours, until the stools became healthy is a most valuable prescription. This must be combined with appropriate diet. The

[1] Waldo considers epidemic diarrhœa to be due to local rather than to climatic conditions.—Milroy Lectures, *Lancet*, May, 1900.

milk must be sterilised and diluted with lime-water. Where cows' milk cannot be retained, condensed milk, whey, or raw meat juice may be tried. The vomiting may be checked by giving only barley-water with white wine whey (§ 212) for a time, and the usual stomach sedatives. In mild cases castor oil, followed by bismuth, rhubarb, soda, and cinnamon, or small doses of grey powder or thymol will effect a cure. In epidemic diarrhœa milk must immediately be stopped, and only whey, or albumin water (white of one egg to half a pint of water), or plain boiled water administered temporarily. With cessation of the diarrhœa, the return to milk should be gradual. In cases with collapse, brandy is to be given, and the child should be put into a warm mustard bath or hot pack until the skin, which is harsh and dry, becomes soft and elastic. The subcutaneous injection of from 5 to 10 ounces of sterile saline at a temperature of 100° F. is very efficacious. Calcium chloride is useful to check the serous exudation, by increasing the coagulability of the blood. If vomiting persists, it may often be checked or diminished by washing out the stomach. This is easily done by using a soft rubber tube with a large eye, and a funnel. For persistent offensive motions rectal irrigation with saline is useful.

V. **Toxic Blood States.**—Enteric fever is nearly always attended by diarrhœa; it sometimes complicates measles, and the other eruptive fevers (especially at their advent), Graves' disease, chronic renal disease, uræmia, pneumonia, and pyæmia. It may also be one of the effects of dissecting-room poisoning. Gouty people are often subject to attacks of diarrhœa, which are of a conservative nature.

VI. A **chill** to the surface in some individuals will determine an attack of acute diarrhœa.

VII. **Acute Ulcerative Colitis** is usually of sudden onset, with diarrhœa, and abdominal pain occurring in paroxysms. The motions are dark, offensive, and contain mucus and blood. There is tenderness over the colon, especially over its ascending portion, which is usually distended. The tongue is furred at first, and the breath very offensive. Pyrexia may be present, about 101° to 102°. The commonest complications are perforation, peritonitis, profuse hæmorrhage, and anæmia. Death may occur from exhaustion in one or two months. The *ulcerative colitis of asylums* is usually more severe, with vomiting and rigor, and may terminate fatally in a few weeks. Both diseases affect chiefly females about middle life. The cause is unknown.

VIII. In cases of acute diarrhœa in which the cause is obscure, reference should be made to the other **Causes** of **Chronic Diarrhœa**, any of which may from time to time give rise to an acute attack. **Dysentery** (§ 219) and **Cholera** (§ 220) are the commonest causes of diarrhœa in tropical climates, and are occasional in ours.

Prognosis of Acute Diarrhœa.—The causes of acute diarrhœa are for the most part removable; and though weakened by the attack, the

patient generally makes a good recovery. Acute Epidemic Diarrhœa in children, however, is a most fatal affection, and it leads to a higher death-rate in infancy, in Great Britain, than any other disease, accounting for nearly 3,000 *deaths annually in London alone.* Briefly, the prognosis in any given case depends upon (i.) the cause; (ii.) the severity of the symptoms and the evidences of weakness; (iii.) the state of the patient's hygienic surroundings; and (iv.) the effects of treatment. Infantile cholera is rarely recovered from. Dyspeptic diarrhœa may be cured in a few weeks, but if untreated, is apt to go on to subacute enteritis. Without treatment all forms of epidemic diarrhœa, even in adults, are serious. Should symptoms of prostration or collapse ensue, the outlook is bad; but it is only at the two extremes of life that this disease is so grave. Ulcerative colitis is very serious; if death does not occur from complications, it usually occurs from the exhaustion, anæmia, or relapses.

Treatment of Acute Diarrhœa.—The indications are (*a*) to *remove* any irritating matters present in the intestinal canal; (*b*) to ensure *rest* to the irritated parts; and (*c*) to *check excessive exudation.* (*a*) Thus, a simple acute diarrhœa in an *adult* following the ingestion of bad food is cured readily by a dose of castor oil (½ oz.) with tr. opii (♏ x.); or calomel. (*b*) Milk and bland food only can be taken; soups and beef-tea are not advisable. In severe cases withstanding treatment, the diet may be restricted to raw meat juice. Simple cases of dyspeptic diarrhœa in children are readily cured by grey powder every night, and alkaline carbonates during the day. If the stools are slimy, bismuth is needed. (*c*) After the expulsion of all irritant matters, a mild astringent, such as chalk or Dover's powder, is beneficial, and bismuth to soothe the congested mucous membrane. Astringents are contra-indicated in the *early stages* of diarrhœa, especially when due to (i.) irritants; (ii.) inflammation; or (iii.) portal obstruction. Only when the diarrhœa threatens to become chronic do we require to use astringents, such as catechu, kino, pulv. cretæ aromaticus, mineral acids, hæmatoxylin, and tannin. Opium allays irritation and checks peristalsis; It may be given as tr. opii or tr. chloroformi et morphinæ. Coto is a useful drug; it acts by diminishing the intestinal secretion. If the stools are very offensive, calomel, charcoal, carbolic acid, and creosote are useful; and a course of intestinal antiseptics may be given —salol (gr. x.), β-naphthol (gr. v.), thymol, etc. Lastly, when other means fail, rectal injections must be resorted to—opium with starch, or silver nitrate; but these are useful chiefly when the disease is in the larger bowel. In all severe cases, absolute rest must be insisted upon, with warmth to the abdomen.

The patient, who is or has been living abroad, complains of severe DIARRHŒA, WITH BLOOD, MUCUS, *and perhaps* PUS *in the stools.* The disease is probably DYSENTERY.

§ **219.** VIII. **Dysentery** is a form of diarrhœa which occurs in marshy and

malarial districts, attended by severe tenesmus and frequent stools, and generally with pyrexia, *due to ulceration of the large bowel*, and depends on the presence of one or more specific organisms. It is met with clinically in two forms—(*a*) *acute* and (*b*) *chronic*. Both are characterised by (i.) diarrhœa, (ii.) the passage of blood, and (iii.) of mucus, from the bowel.

(*a*) Acute Dysentery may be of sudden onset. The patient wakens in the early morning with griping pain, and tenesmus, and during the day there may be from ten to sixty scanty discharges from the bowel, containing blood, mucus, epithelial cells, and later on they acquire the appearance known as the "toad-spawn" discharge. In other cases there is abdominal pain and malaise for a few days before the onset of the diarrhœa. A moderate degree of fever may be present; if at all high, suspect the complication of malaria or liver abscess. In a favourable case the discharge ceases after a week or ten days.

(*b*) Chronic Dysentery may result from an acute attack, or it may be chronic from the onset. In the latter form the patient has a gradually increasing diarrhœa, the stools becoming frequent and scanty, with some tenesmus, and the passage of a little blood or flakes of mucus, the symptoms gradually becoming worse. Dysentery affects the rectum, sigmoid flexure, and descending colon. Sometimes the disease extends as far up as the cæcum, and may consist merely of a *catarrhal* state of the mucous membrane. Severer cases lead to *ulcerative colitis*, when shreds of mucous membrane may be passed, and this may result in thickening and cicatricial tissue, and stricture. The most severe variety, however, is the sloughing or *gangrenous* form, when large sloughs come away with an offensive odour, and are liable to set up septicæmia, or to cause perforation of the bowel, or even a fatal hæmorrhage.

Etiology.—Dysentery more often affects men, especially if intemperate. True or Amœbic Dysentery occurs in the tropics, where it is epidemic and endemic, and is due to the presence of the amœba coli (see § 215). It is supposed that it enters the alimentary canal by the drinking water. It is more apt to affect unhealthy persons, and is predisposed to by any disease or abrasion of the alimentary canal, such as occurs after eating unripe fruit, a chill, the abuse of purgatives, and especially constipation. In heart and kidney disease, secondary diphtheritic enteritis may occur, which is known as dysentery, but the description above refers mainly to true dysentery. Shiga has described a bacillus, known by his name, which is frequently found in epidemic and sporadic cases of dysentery, and is the probable cause of such cases as are not due to amœba dysenteriæ.

Diagnosis.—Acute dysentery resembles nothing for which it could be mistaken excepting acute diarrhœa, from which it is differentiated by the stools. But chronic dysentery should never be diagnosed before an examination has been made to exclude rectal cancer, polypus, piles, bilharzia, and, indeed, any of the other causes of diarrhœa (p. 325). Diarrhœa due to ulceration, occurring in a tropical climate, may be mistaken for chronic dysentery. The only pathognomonic feature is the presence of the amœba coli, or the agglutinative reaction to Shiga's bacillus. The ova of bilharzia hæmatobia may be found in small masses resembling polypi. These, on being broken up and examined by the microscope, show the ova (§ 300). The patient will also have a history of hæmaturia.

Prognosis.—An attack of acute dysentery in a healthy person may pass off in a week or so; but it requires care in a tropical country to prevent it passing on to chronic dysentery, a condition which is very difficult, often impossible, to cure. The sloughing form is extremely dangerous, being so often complicated by grave conditions, such as local abscesses. With chronic dysentery acute exacerbations frequently occur, and the patient becomes anæmic and greatly debilitated by the constant loss of blood. Complications arising in the course of chronic dysentery are ulcers, with consequent periproctitis, abscesses of the liver, cicatrisation with rectal stricture, peritonitis, multiple pyæmic abscesses, and pneumonia.

Treatment.—The main indication in both acute and chronic dysentery is to give rest to the inflamed part. This in the *acute* form is accomplished by keeping the patient absolutely at rest in bed, with no food except white of egg, barley

water, chicken broth, etc., for a day or two. The favourite remedy among English physicians is Ipecacuanha. A small dose of castor oil and laudanum may be given at the outset, and if this does not cut short the disease, ipecacuanha must be administered in doses of 25 to 30 grains at a time. The patient is so apt to vomit the remedy that special precautions must be taken in its administration. A dose of opium is administered, and the ipecacuanha given when drowsiness begins ; and no food should be taken for three hours before and after the drug is given. The patient must be kept lying down, the head low, and no movement permitted. French physicians advise magnesium sulphate (1 drachm every hour) till there is no more blood and mucus passed, and the temperature is normal. It rarely requires to be given longer than three days. Hot fomentations are used for the abdominal pain. Opium in starch enemata relieve the tenesmus ; or the bowel may be washed out with boracic solution.

In chronic dysentery the diet must be non-irritating, but it is not good to keep the patient too long on milk food. Sometimes the patient may recover rapidly on being sent a sea voyage. Constipation must be avoided. Astringents must very rarely be employed, though Bael fruit is advocated by some. Enemata of silver nitrate or sulphate of copper (starting with $\frac{1}{4}$ grain to the ounce), about 2 pints at a time, very slowly injected, are the most useful form of local treatment in the bacillary form, but for amœbic dysentery large enemata (1 to 2 pints) of solution of quinine sulphate are said to be more efficacious. They should be administered warm, beginning with a solution of 1 to 5,000, and gradually increasing to 1 to 2,500, and later to 1 to 1,000, twice daily (see also § 221, IV.).

The patient complains of ACUTE DIARRHŒA *coming on very suddenly, and attended with severe* COLLAPSE, *abdominal* CRAMPS, *and "rice water" stools.* The disease is probably CHOLERA.

§ **220.** IX. **Cholera** (synonym : Asiatic Cholera) is a disease, due to the comma bacillus of Koch, which commences with urgent vomiting, purging, and colourless evacuation, cramps and a tendency to collapse, and which, if not fatal in the first stage, is attended by secondary fever. The period of incubation is usually three to six days, but it may vary between one and ten.

There are three well-marked stages :

(*a*) *Stage of evacuation*, which lasts from two to twelve hours, or longer.—The patient is *suddenly* seized with violent vomiting, severe cramp, and profuse diarrhœa. The stools, after the first few, are colourless and opaque, resembling rice-water, and containing flakes of columnar epithelium and casts of villi ; and the *comma-shaped bacillus* (Chapter XX.). There is severe cramp in the fingers, toes and abdominal muscles, great exhaustion, small and weak pulse, and coldness of the body.

(*b*) The *algid stage*, cold stage, or stage of collapse.—The patient becomes like a corpse ; the surface temperature goes down, and the skin becomes a deadly livid hue ; the pulse cannot be felt at the wrist. The temperature is most remarkable, for in the rectum it may be as high as 105° F., while in the axilla it is only 90° F. During this stage the purging ceases, but the vomiting and cramps persist. The mind remains clear. There is suppression of urine and bile.

(*c*) *Stage of reaction.*—The pulse returns, the temperature rises, the bile reappears, the urine is scanty and deficient in urea. The temperature goes up, and may be attended by typhoid symptoms. The bowels are confined. There may be erythematous, urticarial, and other eruptions upon the skin. This stage is followed by great weakness.

The *Diagnosis* is easy in severe cases on account of its extreme suddenness and the severity of the symptoms. The only condition which resembles it is acute poisoning by arsenic, croton oil, and other irritants. The identification of the bacillus renders the diagnosis certain.

Etiology.—The disease occurs in great epidemics, but it fortunately has not visited this country, except sporadically, since 1865-6-7. Prior to that date there were epidemics in 1854, 1848, and 1832. In India it is endemic, just as

typhoid fever is endemic in London. As regards age, none are exempt. The season of the year in which all epidemics in this country have occurred has been the autumn and the end of the summer. A hot, dry summer predisposes, but the disease is seen in Russia during the winter. The exciting cause is a specific poison, which must be introduced into the alimentary canal. The disease is not communicable excepting by the evacuations from the bowels and stomach (like enteric), and cholera requires the same preventive measures (§ 390 *et seq.*). Fresh evacuations will not produce the disease, but only when they are slightly decomposed, for they take three, four, or five days to become infectious. The disease is usually communicated by drinking water which has become contaminated. But it may be conveyed in other ways through *want of cleanliness*. One attack does not give immunity from a second.

Prognosis.—It is a very serious disorder, and nearly all earlier cases of an epidemic are fatal. The average mortality is 40 to 60 per cent. Patients generally die in the algid stage. If they survive this, they generally recover, though relapses may occur. In the reaction stage uræmic coma, hyperpyrexia, or the typhoid state may cause death. *Untoward Symptoms* are blood in the evacuations, extreme collapse, extreme cyanosis, and absence of the pulse at wrist. Favourable signs are a perceptible pulse in the algid stage, the early occurrence of reaction, cessation of cramp, secretion of urine, and the occurrence of sleep. The commonest *Complications* are pneumonia, occurring in the reaction stage, bronchitis, pleurisy, parotitis, bed sores, diphtheritic inflammation of the pharynx, genitals, or bladder, and corneal ulcers.

There are two *subvarities* : (1) Choleraic diarrhœa, or " cholerine "—that is, cases like autumnal diarrhœa occurring during an epidemic of cholera. (2) Dry cholera, that is, where there has been no vomiting or diarrhœa, but all the other symptoms. These cases are rare.

Treatment.—The best treatment for the *preliminary* diarrhœa is to give a small dose of mild laxative, such as castor oil. This should be followed by astringents and other measures to check the diarrhœa, such as rectal injections of tannin, 20 grains to 2 pints of warm water, and accompanied by light milk diet. When the *stage of evacuation* supervenes, keep the patient warm in bed, with hot bottles and external warmth of all kinds. The denuded intestine may be protected by large doses of bismuth. A little stimulant may be given, in the form of camphor, sp. ammon. aromat., or alcohol. For the cramps, give morphia. In the *stage of collapse* medicines are useless, as they are not absorbed into the system. Packing in hot blankets and large subcutaneous injections of normal saline (0·75 per cent. solution of salt) constitute the best treatment. Chloroform inhalations have also been suggested. In the *stage of reaction* slight purging is not harmful. Give copious diluents, and if the urine does not appear, give an aperient.

§ 221. **Chronic Diarrhœa.**—The term chronic diarrhœa signifies the occurrence of frequent *loose* evacuations, say three or more in the twenty-four hours, extending over a period of weeks, months, or even years (as in Sprue). It is usually, though not necessarily, attended by tenesmus. The stools should be examined (§ 215) whenever the cause is doubtful. In all intractable cases the anus should be carefully inspected. Tenesmus points to the presence of disease of the rectum.

I. Chronic Diarrhœa may be due to some of the same causes as **Acute Diarrhœa** (*q.v.*). In children tuberculous ulceration of the intestine, intussusception, worms, or bad feeding ; and in adults, errors in diet, ulceration, and chronic irritant poisoning, should be remembered.

II. **Fissure of the Anus,** slight ulcers or abrasions, or even an inflamed pile, may cause a chronic diarrhœa, which baffles investigation for a long time. I well remember a woman, æt. sixty-three, who was a

patient in the Paddington Infirmary from March 14, 1887, to January 6, 1888. She had five to eight motions a day, quite loose, but otherwise normal, without mucus or blood on any occasion. There was no history of dysentery, syphilis, or tubercle. She was treated for fourteen weeks by a great variety of remedies. Then, on inspection of the anus, a slight fissure was discovered. She was put on biniodide of mercury ; the number of stools immediately fell to three, two, and one per diem, and she was cured in three weeks.

III. **Ulceration of some part of the Intestinal Canal** is perhaps the commonest cause of chronic diarrhœa in England, and it will be well to mention here all the ulcerating lesions which may affect the intestine, in order from above downwards. (1) Simple ulcer of the duodenum is a rare condition which may arise from burns, or from the same causes as simple ulcer of the stomach (§ 207). There may be few or no symptoms till sudden peritonitis or copious hæmorrhage and melæna occur. (2) Ulcer of the lower part of the ileum may be due to tuberculosis or typhoid fever. (3) Ulcer of the cæcum may arise from the pressure of inspissated fæces or some foreign body—*e.g.*, the bristle of a tooth-brush—which has been swallowed. (4) Ulcer of the vermiform appendix may similarly arise from foreign bodies or as part of appendicitis (*q.v.*). (5) Ulcer of the rectum is generally of malignant or syphilitic origin ; it is attended by the passage of blood and pus, and stricture may result. (6) Ulcers of the large intestine and rectum occur in the later stages of dysentery. These may contract on healing and produce stricture. (7) Cancer of the bowel may produce ulcer in any part of the bowel, but the most frequent situation is the sigmoid flexure. (8) To these some add catarrhal ulceration (§ 218, VII.).

The commonest causes of ulceration in this country are Colitis, Tubercle, Syphilis, Cancer, and in tropical climates Dysentery (§ 219).

1. Colitis, inflammation of the colon, occurs in two forms, ulcerative (§ 218, VII.) and mucous (see IV. below) ; and is one of the most intractable forms of chronic diarrhœa.

2. Tuberculosis of the lungs may be attended by diarrhœa, even without ulceration of the bowel, and in such cases the diarrhœa is considered to be one of the symptoms of the hectic fever in pulmonary tuberculosis, or due to swallowing of the infected sputum. Tuberculous ulceration is recognised by (i.) evidences of tuberculosis in the lungs or other part of the body ; (ii.) the presence of night sweats and intermittent pyrexia ; (iii.) the stools are watery and bilious, and there is rarely any pain. Relief is generally effected by quinine and opium internally, combined with appropriate dietary ; if these fail, recourse may be had to pernitrate of iron, opium, and lead.

3. Intestinal Cancer presents the following features : (i.) The patient is usually over forty-five or fifty, and there may be a family

history of cancer; (ii.) there may be cancer in the glands or other parts of the body, and there is almost always a history of *emaciation preceding the diarrhœa;* (iii.) paroxysmal abdominal pains are frequent, and if the disease is in the rectum there is great pain and tenesmus on passing a motion; if it be not in the rectum, a tumour can generally be made out through the abdominal wall; (iv.) the stools vary, but very often contain blood in considerable quantity.

4. In Syphilitic Ulceration of the bowel (i.) the motions often consist largely of pus and blood; (ii.) great pain and tenesmus are usual, combined with (iii.) other evidences and a history of syphilis. (iv.) Opium and antisyphilitic treatment are here of great value to check the diarrhœa.

IV. **Chronic Colitis** or **Mucous Colitis** is in its early stages frequently overlooked, when the patients complain perhaps only of "nerves." The symptoms are (i.) attacks of diarrhœa alternating with constipation. During the attacks (ii.) mucus is found in the stools. The mucus may be passed in masses, shreds, or casts several inches long. Occasionally blood is also passed, indicating ulceration. The fæces contain intestinal sand in one-tenth of the cases.[1] (iii.) The general health is lowered, associated with despondency and other symptoms of nervous prostration, together with (iv.) abdominal discomfort and sometimes paroxysms of pain. (v.) Examination may reveal a distended and tender colon, particularly over the sigmoid; and thickening of the descending colon or cæcum may be felt.

Course.—The attacks last at first a few days only, then for weeks or months; the patient may at times pass nothing but mucus by the bowel. The disease is not of itself fatal, but is often very intractable to treatment. It leads to neurasthenia, extreme emaciation, and death from asthenia or complications. Relapses may continue for ten years or longer.

Treatment.—During the attacks rest, warmth, bismuth, and milk diet is essential. Between the attacks indigestible food, seeds, and skins of fruit must be avoided. The chief indication is to keep the colon empty, which is best effected by ½ to 1 ounce doses of castor-oil every morning. Lavage with plain warm water (100° F.) should be employed every week. If the disease resist medical treatment, cæcostomy or appendicostomy may be performed, through which the colon is flushed out daily.

V. **Obstruction in the Portal Circulation** produces diarrhœa, due to the congestion of the intestinal wall. It is recognised by: (i.) A previous history of heart disease, or of intemperance and alcoholic dyspepsia; (ii.) other signs of liver or cardiac disease; (iii.) other evidences of portal obstruction, such as ascites, piles, and a large spleen (§ 233); (iv.) there is little or no pain, and the stools are liquid and dark, occasionally bloody. The *Treatment* requires caution, because the diarrhœa and hæmorrhage of themselves relieve the condition by diminishing the venous engorgement. (i.) If the diarrhœa has not lasted long, a large dose of calomel will relieve the portal congestion, and so cure the diarrhœa. (ii.) Magnesium

[1] Dr. Hale White, Allbutt's "System of Medicine," 1907.

sulphate (20 grains), with alum and dilute sulphuric acid, are recommended; bismuth and opium, with caution, are the most useful for checking the diarrhœa.

VI. **Dysenteric Diarrhœa** is a sequel of dysentery, which may perhaps have been contracted abroad many years previously. The laity, seeking a more elegant term, often speak of any form of diarrhœa as "dysentery." The characteristic symptoms here are: (i.) A previous history of acute dysentery, or a residence in dysenteric countries; (ii.) the tongue is generally characteristic, being very clean, red, and often sore; (iii.) the stools vary, but are generally pale, pasty, frothy and easily ferment; (iv.) slight errors in diet produce great aggravation of the diarrhœa. The *Treatment* consists almost entirely in regulating the diet. Only milk, farinaceous food, and eggs should be allowed; no meat, vegetables, or fruit. In severe cases the patient should live entirely on boiled skimmed milk, 4 or more pints a day. Rest and warmth are very important. Bismuth, with or without a little opium, may check the diarrhœa.

VII. **Nervous Diarrhœa** is a form of diarrhœa which may continue for years; it occurs in nervous people and has the following characteristics: (i.) The motions are often quite healthy, sometimes liquid, never attended by melæna or mucus. There is usually no pain or tenesmus. The diarrhœa is generally recurring or intermittent, occurring in the early morning, or when the patient is "nervous." Sometimes it follows each meal (*lienteric* diarrhœa). (ii.) It occurs for the most part in females of a neurotic type. (iii.) Diet seems to produce little or no influence, but the *attacks are determined* by mental emotion or bodily fatigue. A plain but generous diet is called for; and the administration of nux vomica, belladonna, and bromides are often more efficacious than astringents. Careful search should be made for any source of uterine or other reflex irritation. Arsenic (♏ii. Fowler's solution), with meals, is said to be a specific for lienteric diarrhœa.

The crisis of Locomotor Ataxy sometimes take the form of acute diarrhœa, with or without pain. In Hysteria acute attacks of diarrhœa, with noisy borborygmi, may occur, determined in the same way as other hysterical attacks.

VIII. **Amyloid Disease** of the intestines gives rise to a most intractable form of chronic diarrhœa. Indeed, this is the common mode of death in amyloid disease of the viscera. The characteristics here are: (i.) A history of long-standing purulent discharge, or of syphilis; (ii.) great pallor of the skin, accompanied by evidences of lardaceous disease in the spleen, liver, and kidney; (iii.) the stools are generally liquid and extremely offensive, sometimes attended by hæmorrhage. The *Treatment* is very unsatisfactory. Pernitrate of iron, sulphuric acid, logwood, acetate of lead, may be tried, and also opium, which does no harm, even when there is amyloid disease of the kidney, as there is no tendency to uræmia.

The *rarer* causes of chronic diarrhœa are:

IX. **Senile Diarrhœa** was, I believe, first described by Maclachlan, in his "Diseases of Old People." It occurs in persons over sixty or seventy, and is very chronic in its course, but the patient suffers very little. Careful examination for organic disease should be made before concluding that the condition is simply senile diarrhœa. Astringents and most other remedies fail to relieve it, and it may exist for many years without emaciation or danger to life.

X. **Mineral Poisons,** and especially arsenic and antimony, in small continuous doses may cause persistent diarrhœa. It was in this way that the celebrated Maybrick case was discovered.

XI. **Pancreatic Disease** has been associated with diarrhœa. Dr. Burney Yeo has described a case of chronic diarrhœa which resisted all treatment until pancreatic emulsion was administered. The diarrhœa returned when this was stopped, and ceased again on its administration. It may be assumed that only the chronic forms of pancreatic disease (*e.g.*, Fibroid Pancreatitis) would be attended by this symptom, and the diarrhœa is probably dependent upon the excess of fat and undigested muscle fibre in the fæces (§ 181).

§ 222. XII. **Psilosis** or **Sprue** is a condition met with in the tropics. It is characterised by diarrhœa and other symptoms of congestion of the alimentary canal usually running a prolonged, and often fatal, course.

The *Symptoms* consist of (i.) diarrhœa, which is very chronic and continuous, and attended by pale, copious, and frothy stools ; (ii.) there are also symptoms of dyspepsia, with distension of the abdomen ; and (iii.) tenderness of the mouth. At times these symptoms are exacerbated, and aphthous patches appear on the mucous membrane of the mouth and pharynx. In the course of time the patient becomes extremely feeble. Europeans in the tropics are often affected with slight diarrhœa, and Sprue is apt to supervene upon this. Any disease of the intestine, such as dysentery, and any cause of general weakness, predisposes to Sprue. Women are more often affected than men.

The *Diagnosis* is made by the history of chronic diarrhœa, with pale copious stools, and the tender state of the mouth. If untreated, the disease is usually fatal in one or two years. Even with treatment it may lead to death in six to ten years. Much depends upon the age of the patient. If in late middle-age cure is unlikely.

The *Treatment* consists in giving as much rest as possible to the alimentary canal. The patient must be put to bed, and kept on a restricted diet milk for six weeks or more. No solid foods must be allowed. The patient may gradually return in the course of a few months to ordinary diet, but meat and coarse vegetables must be taken only seldom, even after recovery. When milk disagrees, after trying condensed and peptonised milk, or milk and lime water, raw meat juice may be tried for a time, and then gradually return to milk. It is important not to give large quantities per diem, however much the patient may complain of hunger. To begin with only 2 pints of milk should be allowed. This may be increased, when the mouth is not tender, to 5 or 6 pints a day, in the course of two months. The juice of raw fruits, especially strawberries, has been recommended.

§ **223. Tenesmus** literally means stra ning at stool (τείνω, to strain or stretch) ; but in its widest sense it may be taken to mean any local rectal sensation of " bearing down " which results either in a constant desire to go to stool, or a straining when at stool. The latter may lead to prolapse of the rectum, especially in children. Diarrhœa is always attended by more or less tenesmus, but tenesmus is not always attended by diarrhœa. (1) Ascertain if the tenesmus is accompanied by diarrhœa—*i.e.*, are the motions frequent and liquid ? If so, refer to the section on Diarrhœa, § 217. (2) Particular attention should also be paid to the shape and consistence of the motions. (3) Examine locally for any anal or rectal condition such as fissures, piles, polypi, or ulcers. All the pelvic organs should also be very thoroughly investigated, especially in women, in whom the symptom is commoner than in men.

Causes.—Tenesmus (not necessarily accompanied by diarrhœa) may arise from four groups of causes :

1. Various conditions of the ANUS—pruritus, eczema, or fissure—may be overlooked for a long time, as in the case mentioned in § 221, II. Piles also, if internal, may be difficult to detect, but streaks of bright blood will appear in the motions from time to time in that case.

2. Various RECTAL CONDITIONS, especially stricture or ulceration. The former (usually of syphilitic origin) is attended by tape-like stools ;

the latter is attended by pus or blood, or both. Prolonged use of purgatives, or the constant use of the glycerine enema may result in straining at stool and prolapse of the rectum. Proctitis (inflammation of the rectum and anus) is another cause. In the aged, we should always suspect cancer of the rectum, although this is usually attended by actual diarrhœa.

3. PRESSURE on, or irritation of, THE RECTUM FROM WITHOUT, such as may be caused by chronic congestion, version, or other disease of the uterus. These in women, and congestion or new growth of the prostate in men, are both very common causes. Any bladder disease, such as stone—a frequent cause of tenesmus in children, and apt to result in prolapse of the rectum—or new growths. Chronic cystitis may cause this distressing condition. Ischio-rectal abscess, pelvic hæmatocele, and various ovarian and Fallopian tube lesions are all apt to cause tenesmus. The catamenial period is almost invariably attended by a certain amount of tenesmus, and in many women the bowels are only properly opened at such times.

4. IN HYSTERICAL AND NERVOUS SUBJECTS any fright or other emotion may at once determine tenesmus, which is spoken of by the patient as "diarrhœa." In tabes dorsalis the "rectal crises" may take the form of tenesmus.

Treatment.—The indications are (1) the removal of the cause, the treatment of piles and other causal conditions being found elsewhere; (2) the relief of any local congestion or irritation of the rectum. Fissure may require antisyphilitic remedies. In any case, morphia, belladonna, hydrochlorate of cocaine in the form either of suppositories or enemata will relieve the distress from which the patient suffers.

§ 224. **Blood in the Stools** is met with, as we have seen, in dysentery and some cases of simple diarrhœa; but it may be met with unassociated with the latter. The presence of blood in the stools may be recognised by the reddening of the water in which the stool is placed, or by the spectroscope. Clinically, blood in the stools may present two widely different characters: (*a*) When the blood is of *bright crimson colour* it indicates either that the bleeding comes from the rectum or the lower part of the large bowel; or, if it comes from the upper part of the intestinal canal, that it is too large in amount to be acted upon by the intestinal secretion. (*b*) *Melæna* (*tar-coloured stools*) is met with when hæmorrhage in moderate quantity has taken place in the stomach or the upper part of the alimentary tract, when the digestive fluids of the stomach and intestine acting on the blood give it this tarry colour. The causes of these two conditions may to some extent be interchangeable, for what will produce a large hæmorrhage at one time may at another produce only a little.

(*a*) **Bright Red Blood** may be due to the lesions of the lower part of

the alimentary canal. Of these, 1, 2, 3, 5, and 7 are referable to the anus or rectum, and may generally be discovered on local examination.

1. Hæmorrhoids, or Piles, are undoubtedly the commonest cause of blood in the stools. The blood is generally met with in streaks only, but the quantity may at other times be very large. This condition is fully described below, § 225.

2. Fissure of the Anus may also produce streaks of blood. It is a not infrequent condition, and is recognised by the excruciating pain during and after defæcation. The irritation it causes may give rise to a variety of false diarrhœa, as in the case narrated in § 221. The fissure can always be seen by *careful* examination.

3. Rectal Ulcers may give rise to streaks of bright blood in greater or less quantity, mixed with pus and mucus. They are usually of syphilitic, cancerous, or dysenteric origin, and can frequently be felt by digital examination (§ 221).

4. A discharge of blood-stained mucus, coming on somewhat suddenly in an infant, is highly suggestive of Intestinal Intussusception, which is one of the causes of acute obstruction (§ 228).

5. Rectal Polypi are met with chiefly in children.

6. Typhoid and Tuberculous Ulceration of the small intestine sometimes produce very profuse discharges of bright red blood, which comes from the lower end of the small intestine. Other evidences of these affections are present.

7. Bilharzia Hæmatobia causes hæmaturia, but it also gives rise to fleshy masses in the rectal mucous membrane, resembling piles which bleed. The ova are very characteristic, and can be detected in the fæces (§§ 215 and 300). It is only met with in persons who have been to South Africa or other tropical or subtropical countries.

8. Various General Blood Conditions may give rise to hæmorrhage coming from the rectum or elsewhere in the alimentary canal in varying amount. This is so in purpura, scurvy, hæmorrhagic forms of the specific fevers, acute yellow atrophy of the liver, and leukæmia.

(*b*) **Melæna** (*tarry stools*) is met with when bleeding takes place from the stomach or high up in the alimentary tract in moderate quantity. Its causes are as follows :

1. When coming from the stomach, it may be associated with profuse *hæmatemesis* (§ 192) ; the commonest causes of hæmatemesis are gastric ulcer and hepatic cirrhosis.

2. Portal Obstruction (§ 233) is one of the most frequent causes of melæna, especially that form due to alcoholic cirrhosis of the atrophic variety. It may also occur with the engorged liver and spleen of advanced cardiac disease. In either case the hæmorrhage in these circumstances is a natural safety-valve, and gives relief to the engorged state of the portal circulation.

3. Cancerous, Tuberculous, and other Ulcerations of the small

intestine (see §§ 218 and 221), and lardaceous disease of the bowel may also produce melæna.

4. The General Blood Conditions above named, when the hæmorrhage is small in amount, are attended by tarry instead of bright red stools. *Melæna neonatorum*[1] is a very rare condition, apparently due to some blood change, and is attended by pyrexia. It starts during the first week of life, and may terminate fatally in a few days. Nothing has been found post-mortem.

5. The Ankylostomum Duodenale (ankylostomiasis) is a frequent cause of profuse melæna in Egypt and other foreign countries, so profuse and continuous as to cause anæmia (§ 215).[2]

The *Treatment* of melæna should be directed to the cause, but the general principles are those laid down for hæmatemesis (§ 192). Turpentine (10 minims capsule), lead acetate, and opium are recommended. Suprarenal gland has recently been advocated as a remedy. *Ankylostomum* is readily destroyed by thymol. First empty the bowels by a brisk purge given the night before, then administer three or four doses of thymol (20 grains each) in cachets, at intervals of three hours. The bowels must be kept freely open all the while and alcohol and all fats carefully avoided, so as to prevent absorption and poisonous effects.

§ **225. Hæmorrhoids,** or Piles, consist of a varicose condition of the rectal veins. This varicosity forms a swelling of variable size, which may be altogether within the anus (internal piles), or partly internal and partly external. Internal piles may in some cases be seen, when the patient " bears down " as small purple swellings just protruding from the sphincter; in other cases internal piles are discovered only on digital examination of the rectum.

Symptoms.—(1) Streaks of *bright red* blood occur in the stools, and sometimes as much as ¼ pint of blood may be passed at one time. (2) There is pain on defæcation, the pain continuing for some time after the passage of a stool. When a pile becomes inflamed, or strangulated by the sphincter, severe pain and discomfort is experienced, and the patient may have to remain in bed for days. Pain may be referred to other parts of the body—*e.g.*, to the testicles, bladder, or loins. (3) Constipation nearly always accompanies piles, due partly to mechanical obstruction, and partly to the pain caused by defæcation. (4) In severe cases constitutional symptoms are developed, such as lassitude, irritability, headache, faintness, and later on, perhaps, anæmia, from loss of blood.

Etiology.—(1) Portal obstruction is itself a cause of piles, and in all marked cases we should seek for the other symptoms of this lesion (§ 233). (2) Habitual constipation, however, is undoubtedly the most common cause of hæmorrhoids, particularly in women, who in early life

[1] Osler, " Prin. and Pract. of Med.," p. 747, sixth edition.
[2] Other worms are mentioned by Dr. F. M. Sandwith in the *Lancet*, 1899, vol. ii.

are so apt to contract this habit. (3) Alcohol especially in the form of malt liquors, with excess of sugar, causes portal congestion, and thus becomes a source of piles. Alcohol in any form aggravates the condition. (4) Sedentary occupations and deficient exercise also produce piles. (5) Various local conditions, such as sitting on soft cushions which constrict the inferior hæmorrhoidal veins, uterine displacements, pelvic and other tumours, are all potent causes of hæmorrhoids.

Prognosis.—Hæmorrhoids are not usually regarded as serious, but they may be extremely troublesome, partly by the constant loss of blood, partly by their liability to repeated attacks of inflammation, and partly by the pain they cause.

Treatment.—Much may be done by three simple means: (1) The avoidance of alcohol (especially malt liquors) and sugar; (2) keeping the piles scrupulously clean; and (3) the bowels regularly and loosely open. Rich food and other causes of hepatic congestion must be forbidden. Hydragogue purgatives are best—such as Mist. Alba, Hunyadi, or Carlsbad waters every morning, or confect. sulph., or sennæ, with an occasional cholagogue at night. Regular exercise is desirable. Local applications should be of the simplest kind. The old-fashioned gall and opium ointment is now very properly replaced by hamamelis, with conium, morphia, or cocaine for the pain if necessary. Unguentum hamamelidis (B.P.) is an excellent preparation, and is best applied on a strip of lint inserted within the anus, and left there; or a suppository may be employed, containing 1 to 3 grains of hamamelin, and $\frac{1}{8}$ grain of morphia if requisite. Inflamed piles are very painful, and are best treated by warm hip-baths, frequent bathing, sitting over hot water in a bidet, warm fomentations with opium, belladonna, or cocaine. Incision may be required, but leeches are better. For the radical cure, removal by surgical measures is called for in some cases.

§ 226. Intestinal Worms may give rise to no symptoms at all. They are most frequently met with in children, and may remain undiscovered until they are found in the stools. The morphology, symptoms, and habitat of the various entozoa are described in Table XVII., p. 323. Threadworms (Fig. 67) and roundworms (Fig. 68) are the most common. It used to be considered that threadworms lived in the colon, but it is now believed they exist in the caput cæcum, and sometimes in the vermiform appendix. This fact explains those cases which appear to be cured for a time, but which continually relapse.

The *Symptoms* are very indefinite, and consist of: (1) Vague and persistent, though often paroxysmal, pains in the abdomen; (2) capricious and sometimes ravenous appetite, in spite of which the child becomes thin and sallow; (3) grinding of the teeth at night, picking of the nose, and other reflex phenomena; (4) irregularity of the bowels, or diarrhœa; (5) threadworms produce intense itching of the anus, and consequently fidgettiness, especially at night. They may wander forwards and cause vulvitis.

The *Treatment* differs for the different worms. For *threadworms* the best treatment consists of quassia injections. After an aperient, 1 ounce of powdered quassia to a pint of boiling water is, when cold, injected slowly into the bowel, and retained as long as possible. Common salt injections of the same strength may be used. The worms are destroyed with two or three such injections. Santonin (1 grain) in a powder with calomel is very efficacious; it should be given

on alternate days for three doses, followed by castor-oil. Where the worm has its habitat high up in the intestine (as the *tapeworm*), treatment is conducted in three stages. (1) In order to starve the parasite by keeping the alimentary canal as empty as possible, the patient should have no food after midday, and at night or the next morning a purgative must be taken. This leaves the worm uncovered, and thus readily acted upon by (2) the anthelmintic, which is given about an hour after purgation. The chief anthelmintic is ext. filicis liq., 1 drachm. Some recommend 30 minims of spirits of turpentine to be given with this; others give kousso (4 drachms) or pelletierine (2 grains of the alkaloid). (3) Two hours later give calomel with a saline aperient, to eject the worm from the body. The stools must be examined to see that the head is passed. If only segments are passed, the worm will grow again, and the same treatment will have to be repeated within three months. For the *round worm* the specific remedy is santonin, given in 2 grain doses to a child of three and upwards; for an adult 5 grains are given. For *Ankylostomum Duodenale*, see § 224.

§ 227. **Constipation** is insufficient action of the bowels, or the passage only of hard, dry, or ball-like masses of fæces (scybala), independent of organic disease within or outside the intestinal canal. This source of fallacy must be carefully excluded before diagnosing a case as one of simple constipation.

The *Symptoms* which accompany or result from constipation are sufficiently familiar—at first headache, languor, and depression, followed by a furred, coated tongue, dyspepsia, anæmia, sleeplessness, and eruptions, for the most part of an urticarial or erythematous nature. The temperature may rise a degree or so in certain conditions from temporary constipation, and I have met with one case where it went up to 102° F. The retention of hard fæcal masses may give rise to an alternating diarrhœa, which leads to error in diagnosis. Hæmorrhoids is another consequence of habitual constipation, and a distended ulcerated colon may result in some subjects. In women, in whom the condition is far more common than in men, a chronic torpidity of the bowels may predispose or even lead to uterine disease; and in both sexes varicose veins, œdema of the legs, sciatica, especially on the left side, and numbness of the legs are among its consequences.

For purposes of treatment we may consider the **Causes** of simple or uncomplicated cases of constipation under three headings:

(*a*) **Errors of Diet.**

(i.) Too bland food—*e.g.*, no vegetables, no food with coarse residue.
(ii.) Too dry food—*e.g.*, deficient fluid ingesta.
(iii.) Too little or poor food, or too great uniformity of diet.

(*b*) Causes of **Defective Vermicular Action** (other than errors of diet).

(i.) Sedentary habits.
(ii.) Advanced age, and other conditions where the general neuro-muscular tone is poor, as in melancholia, or anæmia.
(iii.) Prolonged disregard of calls of nature.
(iv.) Weak abdominal muscles.
(v.) Atony of the intestine, with or without chronic (mucous) colitis (§ 221, IV.).
(vi.) Diseases of the cord or brain—*e.g.*, cerebral tumour, tabes.
(vii.) Some febrile states.
(viii.) Spasmodic reflex conditions, as from uterine or ovarian diseases.
(ix.) Drugs, such as opium, lead, iron.

(*c*) **Deficiency of Bile,** or **Intestinal Secretions.**

(i.) Functional inactivity of the liver (§ 249).
(ii). Profuse vomiting.
(iii.) Excessive loss of fluid by skin or kidneys.
(iv.) Astringents, such as chalk or catechu. Hard waters also act in the same way.

Treatment.—Chronic constipation is serious in respect of the troublesome consequences mentioned above. In its treatment we should first endeavour to *find out the cause.* Examine the colon to see if it be distended; place one hand at the back, and press it forwards between the iliac crest and the last rib to meet the other hand, which is placed flat on the anterior abdominal wall, the patient being in the recumbent position. Having excluded local causes by a thorough examination, we should consider the various causes above mentioned. The treatment of constipation comes under six headings.

(1) *Dietetic Treatment.*—Increase the amount of fluid taken—*e.g.*, by sipping a tumbler of hot water slowly whilst dressing in the morning and undressing at night. Avoid large quantities of milk or hard water. Coarse foods should be eaten which stimulate the intestinal wall, such as oatmeal, wholemeal, or brown bread, green and raw vegetables, figs, prunes, and ripe fruits. A teaspoonful or tablespoonful of salad-oil at meal-times will often be very efficacious in cases due to deficient intestinal secretion. (2) Inculcate regular *habits*, even when there is no inclination to go to stool. Psychotherapy may also be employed. (3) Active *exercise* is advisable, excepting where uterine or ovarian irritation is in operation. A systematic exercise may be practised by lying flat upon the back and rising from a recumbent position without the aid of the arms ten or a dozen times each morning and evening. Systematised exercises of other kinds are now taught by many qualified teachers of physical culture. (4) Abdominal *massage* is often useful. Gently "rolling" the abdominal wall, or rolling a 7-pound ball over the abdomen, in the direction of the hands of the clock. (5) *Drugs.*—For occasional constipation, aloes with the evening meal and a seidlitz-powder in the morning are the most harmless. Calomel or other mercurial preparations should not be given habitually, but may be taken once a week for a few weeks. A useful vegetable pill is pil. col. co., pil. rhei co., aa gr. i., ext. hyoscyami, gr. $\frac{1}{2}$; two at bedtime. Belladonna and nux vomica in small doses undoubtedly promote vermicular action; the former is especially useful for women with pelvic irritation. A two-months' course of cascara sagrada, graduating the dose to the individual, will often break through a vicious habit of constipation. Jalap, elaterium, scammony, and gamboge are useful where drastic purgation is desired. Salines given daily for some weeks will often re-establish the functions of a torpid intestine (F. 46). These may be given in the form of the mineral waters, such as Carlsbad, which contains 13 grains of sulphate of soda to the tumbler, with alkalies (dose, one or two tumblers

twice daily) ; Friedrichshall, which contains 60 grains of the sulphates of magnesia and soda with alkalies (dose, half a tumblerful daily) ; Hunyadi water, which contains 200 grains of sulphates of soda and magnesia with alkalies (dose, quarter to half a tumbler). All of these are best given on an empty stomach (F. 57, 88, and 90 are also useful). An excellent aperient for children is cascara and maltine mixed together in the proportion of 10 to 20 minims of the ext. casc. sagrad. liq. to the teaspoonful of maltine. (6) *Enemata* are useful in certain conditions, though it must always be borne in mind that they do not empty the small intestine. They are, however, better than constant purgation in some cases. The ordinary soap enema or 1 or 2 pints of soapy water may be used. Half an ounce of glycerine is a very effective enema, but it should not be used longer than a few weeks, for it produces an irritable condition of the rectum. In cases of very prolonged constipation which resist all other means, I am in the habit of prescribing ¼ to ½ pint of olive-oil as an enema every night. If this be injected very slowly, it is retained, and after a course of one or two weeks it is wonderful how regularly the bowel resumes its functions. Another valuable enema consists of the following : mix two tablespoonfuls of sodium bicarbonate with two tablespoonfuls of olive-oil, adding just sufficient warm water to make a cream. Then stir in 1 pint of hot water, and inject slowly into the rectum, with the patient on his back.

The patient complains of SUDDEN STOPPAGE OF THE BOWELS *with inability to pass even flatus,* ABDOMINAL PAIN, *and* VOMITING *which gradually becomes stercoraceous ; his* PULSE IS RAPID, *and there is tendency to* COLLAPSE. The case is probably one of ACUTE INTESTINAL OBSTRUCTION.

§ **228. Acute Intestinal Obstruction** is one of the most serious medical or surgical emergencies to which a medical man can be summoned.

The *symptoms common to all forms* of acute obstruction are (1) complete *constipation, not even flatus being passed.* (2) The *pain* is at first paroxysmal, referred to the umbilicus, though it becomes continuous later on. There is not usually much tenderness. (3) The *vomiting* comes on earlier, is more urgent, and becomes more rapidly stercoraceous in proportion as the obstruction has taken place high up in the intestines. (4) Abdominal distension is generally present, and this may be one-sided, so giving us a clue to the position of the obstruction. (5) Constitutional symptoms gradually supervene, with prostration and a thready, *rapid pulse.* These also are more urgent when the small intestine is involved. The urine is diminished in proportion as the obstruction is near the stomach, for then the vomiting is more urgent.

Diagnosis of Acute Intestinal Obstruction.—When summoned to a case presenting these three symptoms—stoppage of the bowels, acute abdominal pain, and vomiting—the first step is to identify the case as

one of acute obstruction. In *colic* (renal, hepatic, or intestinal) all of these three symptoms may be present, but the patient's general condition is not so serious, and the bowels are readily relieved by purgatives or enemata. Moreover, the position of the pain in renal and hepatic colic is characteristic (see § 172). In *acute peritonitis* there is great tenderness over the abdomen, thoracic respiration, and some fever (see also § 170). But when there is *perforation* into the *peritoneum* collapse is present, at first without fever, and perforation is diagnosed with difficulty only by (i.) the passage of wind by the bowel; (ii.) the collapse being much greater even than that in acute obstruction; and (iii.) a possible history of the condition which has resulted in perforation or rupture (consult also § 169). It is sometimes impossible to diagnose these two conditions, and an exploratory operation should be undertaken without delay.

Causes of Intestinal Obstruction.—It is of some importance to ascertain the cause, for the prognosis and treatment differ somewhat in each case. (*a*) In *acute* intestinal obstruction, in which the symptoms come on *suddenly* in a person previously healthy, there are four *common* causes: (I.) External hernia; (II.) intussusception; (III.) internal strangulation; (IV.) appendicitis (perityphlitis). (*b*) Sometimes, however, acute will supervene on chronic obstruction, and the *common causes of chronic obstruction* (§ 229) are also four in number: (I.) Malignant stricture of the bowel; (II.) simple stricture; (III.) pressure of a tumour; and (IV.) dilatation of the bowel.

Features special to the several causes of acute intestinal obstruction.

I. External Hernia is known by the presence of a tumour in the femoral, inguinal, or umbilical region. No impulse on coughing is present. Obturator hernia is very rare, and can only be discovered at the time of operation.

II. Intussusception, or invagination of the bowel, is by far the commonest cause in childhood. According to Brunton, it is a cause of 43 per cent. of all cases of obstruction. True intussusception is always from the bowel above into the part below, and in more than half of the cases the lower part of the ileum becomes invaginated into the cæcum. In a third of the cases some other part of the ileum, and in about one-eighth some part of the colon, is implicated. The invaginated portion slowly sloughs, the two edges may be welded together, the slough may pass about the eighth or tenth day; thus spontaneous recovery may occur, though this is relatively rare. Death from perforation and collapse is more usual unless the case is dealt with surgically. Intussusception is known by (i.) severe tenesmus; (ii.) a rectal discharge of *blood and mucus;* (iii.) a sausage-shaped tumour may be felt, altering in position, on palpating the abdomen, and in extreme cases the invaginated portion of bowel is felt *per rectum;* and (iv.) the patient is a child, usually under two years of age.

III. Internal Hernia or Strangulation—*e.g.*, by bands of adhesion—is known by (i.) the urgency of the symptoms ; (ii.) the patient is an adult man, with (iii.) a history of old peritonitis. Volvulus (or twisting of the bowel) may be indistinguishable from the preceding—indeed, it practically results in strangulation—but (i.) it occurs in men over forty, usually with a history of chronic constipation ; (ii.) abdominal distension may be great ; (iii.) sometimes a tumour is felt over the sigmoid flexure, the usual site of volvulus.

Internal strangulation may also arise from (1) adhesion of the end of the appendix vermiformis through which a knuckle of the bowel gets nipped. (2) Agglutinations of the bowel. This is a cause of 3½ per cent. of the cases of acute obstruction. (3) Congenital deficiencies in the mesentery or bowel, or the foramen of Winslow.[1]

IV. Appendicitis (Perityphlitis) is known by (i.) pain and tenderness of a constant character in the right iliac fossa ; (ii.) the temperature is elevated if the inflammation has extended to the parts around ; (iii.) a history of previous attacks is often obtainable (see § 175).

The **rarer causes** of acute obstruction are three in number :

V. Impaction in the Bowel of a large Gall-stone. This is not so rare as might be supposed, and Dr. Murchison was able, without much difficulty, he stated, to collect thirty-four cases.[2] A large gall-stone escapes from the gall-bladder by ulceration into the bowel. The obstruction is high up in the small intestine, and consequently (1) the pain and constitutional symptoms are of extreme severity, and of very sudden onset. (2) The patient is usually a female (four females to one male), at or beyond middle age. (3) There may be a history of biliary colic, and in all cases there is a history of localised peritonitis some weeks or months before the seizure. (4) The symptoms may intermit, from the stone shifting its position.

VI. Obstruction of the bowel may sometimes be due to an Extravasation of Blood into the coats of the intestine. It occurs only in purpura, hæmophilia, and other blood disorders. Such cases are recognised by evidences of hæmorrhage in other positions—melæna, epistaxis, purpura.

VII. Among the still rarer causes of obstruction may be mentioned masses of round worms (Trousseau), concretions of ammonio-phosphate of magnesium (a frequent cause in horses, though rare in man), and other foreign bodies in the intestine.

Clinical Investigation and Diagnosis of the Cause of Obstruction.—If the case occur in a child, and there is a history of diarrhœa for the past few days, it is almost certainly intussusception ; in an old person suspect rectal stricture, impacted fæces, or volvulus ; in a young adult suspect strangulation, hernia, or perityphlitis. If the vomiting come on early and is urgent, it points to a tight constriction *high up* in the intestinal tube. If the distension is chiefly in the centre of the abdomen,

[1] The rarer conditions are connected with congenital malformations. For instance, an interesting case of patent Meckel's diverticulum into which the posterior wall of the ileum became intussuscepted, forming an umbilical tumour, in a male child, æt. six weeks, is published by Dr. Leonard Guthrie in *Pediatrics*, July 1, 1896, vol. ii.

[2] "Lectures on Diseases of the Liver," by Charles Murchison, M.D., LL.D., F.R.S., second edition, p. 497. London, 1877.

the obstruction is probably above the ileo-cæcal valve; if it is chiefly in the flanks, the obstruction is below the valve; if more in the right than in the left flank, the obstruction is probably in the sigmoid flexure.

When called to such a case, first examine for swelling in the positions of external herniæ. If the abdomen be distended, and present visible waves of peristalsis, inquire as to the causes of chronic obstruction (*infra*), as the case is probably an acute supervening upon a chronic obstruction. Always *examine per rectum*, for in acute intussusception the invaginated part of the bowel may be felt *per rectum*, and there may be a discharge of blood and mucus; or a stricture or other cause of chronic obstruction may thus be discovered. Next inquire into the past history—*e.g.*, for peritonitis (as this is a cause of internal strangulation), or for appendicitis or hepatic colic. Then examine the abdomen by palpation and percussion for tumour or tenderness. If the abdomen is distended only on one side, the site of the obstruction may be localised.

Prognosis.—The prognosis of obstruction of the bowels is always very serious. Death occurs in the natural course either from (1) gangrene and rupture of the bowel, or (2) exhaustion and collapse. The prognosis almost entirely depends in the present day upon the *stage at which the case comes under notice*, and the treatment adopted. All the acute cases require early surgical interference, and a surgeon should be summoned at once. The success and justification of such diagnostic operations form one of the chief triumphs of modern surgery. As regards the *Causes*, obstruction from a gall-stone is perhaps the most serious, then intussusception, then internal strangulation; perityphlitis being more favourable. Among the gradual causes, carcinoma of the bowel gives the gravest prognosis, and paralysis the most favourable. Cases in which the obstruction is high up are less favourable than those in the large bowel.

Treatment.—Acute intestinal obstruction is one of those serious conditions that demand the resources of both a physician and a surgeon, who should jointly undertake the management of a case. The indications are (1) to ascertain the cause; (2) to endeavour to remove the obstruction; and (3) in the meantime to support the strength and relieve the pain by controlling the peristalsis upon which it depends. Enemata may be given in all cases; purgatives should be avoided. Warmth is applied to the abdomen in the form of hot fomentations, turpentine, belladonna, or opium stupes. If there are signs of peritonitis, cold is said to be more efficacious. The question of the administration of opium is debated (see Appendicitis). Surgeons object that it masks the symptoms in cases likely to require operation, yet it is hard to know how otherwise to relieve the extreme pain in these cases. The diet should consist of fluids, such as iced milk, beef-tea, and stimulants, given in small quantities, and frequently.

In *external hernia*, after a warm bath, and taxis under chloroform,

proceed at once to operation. In *intussusception* some mild cases have a tendency to spontaneous recovery. Some surgeons recommend that an attempt should be made to reduce it by injections of warm saline or olive-oil, or by distending the gut with gas by bellows or a syringe under an anæsthetic, but it is better to proceed at once to laparotomy, which, in any case, should not be deferred for more than twenty-four hours. In *internal strangulation* or twisting it is best to operate without delay if an injection does not relieve and we are certain of the diagnosis. In cases of recovery without operation there has probably been a simple volvulus. But death almost always occurs in cases of internal strangulation if unrelieved. Manipulation, and inflating the bowel by means of bellows, have been suggested, but there is considerable risk attending these procedures. In *appendicitis*, see § 175. In *impacted gall-stone*, the progress is so rapid towards a fatal issue that operation, if undertaken, must be done immediately. The same remark applies to other foreign substances in the intestine.

The patient complains of CONSTIPATION *progressively increasing until he is unable to pass even flatus,* ABDOMINAL PAIN, *and from time to time* VOMITING ; *there is a rapid pulse, and general ill-health.* The case is one of CHRONIC INTESTINAL OBSTRUCTION.

§ **229. In Chronic Intestinal Obstruction** (1) the abdominal pain is generalised, intermittent, and of increasing severity. (2) There is constipation, or a history of alternate constipation and diarrhœa culminating in complete stoppage ; and (3) abdominal distension in most cases, and peristalsis in some, may be visible. The chief causes of this condition are four in number :

I. MALIGNANT STRICTURE by new growth in the wall of the bowel—*e.g.*, cancer. Its most common situations are the colon, especially the sigmoid flexure, and the rectum. This cause of obstruction may be recognised by (1) the presence of a tumour or stricture which may be felt on examination *per rectum*, and the distension of the abdomen being most marked in the flanks. When the tumour is situated higher up than the sigmoid flexure, it may generally be felt through the abdominal wall ; and when situated in the sigmoid flexure, it may be inspected by a sigmoidoscope. (2) When the sigmoid flexure or rectum is affected, the illness is often preceded by sciatica on the left side. (3) There are cancerous cachexia, the age of the patient, and perhaps hæmorrhage and fœtid discharge to aid in the diagnosis.

II. SIMPLE—*i.e.*, NON-MALIGNANT STRICTURE of the intestine may arise in consequence of dysenteric, syphilitic, or other ulceration, either in the colon or in the rectum. An ulcer alone is capable of producing symptoms of obstruction. This cause is recognised by (1) the absence of a tumour, and (2) a previous history of dysentery (perhaps only a mild attack), and residence in a tropical climate ; or a history of syphilis,

with a rectal discharge. Syphilitic stricture is rare, except between the sigmoid flexure and the anus.

III. Pressure on the Bowel by a Tumour or an enlargement of some viscus such as the uterus. This cause is recognised by the physical signs of tumour or enlargement respectively.

IV. Dilatation of the Bowel from paralysis of its coats. This is chiefly met with in the aged. It is differentiated from the other causes chiefly by (1) the absence of cachexia, tumour, emaciation, or other symptoms of the preceding causes, and an absence of a history of syphilis or dysentery. (2) The gradual formation of a soft fæcal tumour, situated in the descending colon. The *Diagnosis* of these causes is also discussed on p. 345.

V. Chronic Peritonitis (§ 176) causes a matting together of the intestines, and intestinal obstruction may result. Cancerous peritonitis is attended by much pain and the effusion of much fluid ; but in tuberculous peritonitis there are mostly adhesions, less pain, and less fluid.

VI. Chronic Intussusception is thus known : (1) It occurs usually in children ; (2) tenesmus is present ; (3) a tumour may be felt with characters similar to that met with in acute intussusception ; and (4) there is usually no marked distension (see also Acute Intussusception above).

VII. Appendicitis (§ 175) may occasionally cause chronic, but more often acute, obstruction.

Prognosis.—In all forms of chronic intestinal obstruction the symptoms of acute obstruction are apt at any time to supervene, from impaction of fæces above the narrowing lumen of the gut, but apart from this the prospect differs considerably in the different causes. A cancerous stricture is the most, a dilated colon the least, serious. Syphilitic stricture may be relieved by iodides ; dysenteric stricture is much graver, and irremediable. The course of a tumour varies with its nature. Chronic intussusception *may* spontaneously resolve, the invaginated part sloughing off and being passed by the rectum, but the outlook is always grave.

Treatment.—In most of the cases of chronic intestinal obstruction, surgical procedure is ultimately necessary, but at first the treatment consists in watching the patient until a diagnosis can be formed with as much accuracy as possible, and in giving digestible food, preferably such as leaves but little residue, the pain being relieved by opium and external applications (hot fomentations with turpentine or opium). In atony of the bowel, if oil enemata and other medicinal treatment fail, the fæces may require to be removed by mechanical means (scooped out). For simple stricture of the rectum gradual dilatation by bougies may be tried. In chronic intussusception operation is advisable. In cancerous stricture[1] an operation may prolong life by the formation of an artificial anus, and the longer the operation is delayed, the worse is the prognosis. It should never be delayed until vomiting has commenced. In some cases the bowel has been resected with success.

[1] For Treatment of Cancer, see § 415 .

CHAPTER XII

THE LIVER

We still remain in comparative ignorance of the functional disorders of the liver, but the structural diseases lend themselves more readily to physical examination and medical diagnosis. The fact that the liver is capable of containing a fourth of the blood in the body is sufficient proof of its importance. All the blood passing from the stomach and intestines circulates through the liver, after which it joins the general circulation considerably altered in its composition. Experimental researches show that the liver is concerned in the manufacture of urea or the antecedents of urea.[1] Degeneration or destruction of the hepatic cells is attended by a diminution in the quantity of urea excreted, and the amount of urea in the urine may be taken as a valuable prognostic guide in many hepatic disorders. The metamorphosis of the products of digestion in the course of their elaboration into urea is therefore one of the functions, probably the chief function, of the liver. Another important function of the liver is the manufacture of glycogen; the third and least important function is the secretion of bile.

PART A. SYMPTOMATOLOGY.

The symptoms due to disorders of the liver are not so clearly defined as those of cardiac or pulmonary diseases. The cardinal symptoms of *structural* disease of the liver are PAIN IN THE HEPATIC REGION, JAUNDICE, and a group of symptoms due to PORTAL OBSTRUCTION, which include Ascites. When the liver cells become gradually destroyed, as in cirrhosis, serious disturbance of the general health ensues, and in the later stages of that and of some other hepatic disorders LETHARGY passing into coma supervenes. The chief symptom of *functional* derangement of the liver is, according to Murchison,[2] an excess of lithates

[1] If blood containing no urea be passed artificially through the liver, it is found that the effluent fluid contains urea, and therefore it must have been added to it in the liver. Again, if the portal vein be connected to the jugular vein, thus shutting out the liver from the general circulation, the formation of urea ceases.—Solnikoff and others, see Alexander Hill's "Physiologists' Notebook," p. 82. Cambridge, 1893.

[2] "Clinical Lectures on Diseases of the Liver," second edition. Longmans and Co., London, 1877.

in the urine, LITHURIA, consequent on an excess of lithic or uric acid in the blood (lithæmia). How far lithæmia is really due to hepatic disorder is still a debated point (§ 249); it may certainly arise in other ways. Functional derangement of the liver is always attended by DEPRESSION, which may amount to hypochondriasis, and vague DIGESTIVE DISTURBANCES.

§ **230. Pain and Tenderness over the Liver** is very marked in PERIHEPATITIS and any other condition in which the capsule is involved, and sometimes radiates upwards towards the right scapula. The onset of pain in the course of a liver complaint may therefore be of considerable importance; for example, in hydatid of the liver, the natural course of which is painless, it would point to a danger of rupture of the cyst. When the upper surface of the liver is involved, the pain is very often *referred to the right shoulder;* it is, indeed, a symptom of phrenic (diaphragmatic) irritation. The most severe form of pain, however, is that which occurs in connection with the passage of GALL-STONES (*biliary colic*). In a considerable number of hepatic disorders pain may be completely absent. There is, however, in many cases of marked disease or enlargement of the liver a feeling of weight or fulness in that region, accompanied by an inability to lie on the left side.

Hepatic pain may be *simulated* by Pleurodynia (rheumatism of the intercostal muscles), Intercostal Neuralgia, Pleurisy, Dyspepsia, and various gastric conditions, and by Intestinal or Renal Colic.

§ **231. Jaundice** is the term applied to the yellow pigmentation of the skin and other tissues due to the non-elimination of bile. It appears first in the urine, in which bile pigments and acids may be detected (§ 282), next in the conjunctivæ, then in the skin universally and uniformly.

FALLACIES.—The yellow coloration of the conjunctivæ differentiates jaundice from all similar pigmentations of the skin. (1) Excess of *subconjunctival fat* may simulate jaundice, but this is readily distinguished by its unequal distribution. (2) The *sallowness* of the skin in chlorotic young women is easily distinguished from jaundice by the absence of pigment from the conjunctivæ, and the absence of bile from the urine. (3) The *cachexia* of carcinoma, malaria, and certain other forms of visceral disease, is differentiated in the same way. (4) The *bronzing* of the skin in Addison's disease is hardly likely to be mistaken for jaundice. (5) *Santonin* and *rhubarb*, administered internally, colour the urine, but do not give the reaction for bile in that fluid.

Symptoms accompanying Jaundice.—(1) Flatulent dyspepsia, and a bitter taste in the mouth. (2) Pruritus, which may be very troublesome in some cases; eruptions, such as xanthelasma, are less common. (3) The temperature, as a rule, is subnormal, and the pulse slow; (4) general debility and emaciation ensue in prolonged cases; (5) head symptoms, such as delirium and coma, may appear towards the end and xanthopsy — *i.e.*, a yellow vision — is sometimes present. (6) Hæmorrhages, either subcutaneous or from mucous membranes, are liable to occur in severe cases.

Recent experiments have shown that jaundice can no longer be

divided into *obstructive* and *non-obstructive*, for all jaundice is obstructive,[1] nor into *hepatogenous* and *hæmatogenous*, since no bile pigments can be formed when the liver has been removed. Jaundice may be divided into that due to **extra-hepatic obstruction,** and that due to **intra-hepatic obstruction,** or **toxæmic jaundice.** In the latter group the action of the poison is, first, blood destruction (hæmolysis), resulting in increased formation of bile pigments ; and, secondly, increased viscidity of the bile, which causes a temporary obstruction and absorption of bile pigments. Clinically, jaundice due to extra-hepatic obstruction is distinguished by the *colour of the stools*, which are pale, slate, or clay-coloured, from the absence of bile in the intestinal canal. There are four separately named forms of jaundice—Catarrhal Jaundice (§ 239), Epidemic Jaundice, or Weil's disease (§ 240) ; Malignant Jaundice, Icterus Gravis, or Acute Yellow Atrophy of the Liver (§ 247) ; and Icterus Neonatorum (§ 232).

(*a*) **Jaundice due to Extra-hepatic Obstruction** may be produced in three ways—Obstruction within the bile-duct, disease in the wall (II. and III. below), or pressure outside the bile-ducts.

I. Foreign Bodies within the duct, such as (1) gall-stones and inspissated gall ; (2) hydatids, round worms, distoma,[2] and other parasites ; (3) foreign bodies from the bowel.

II. Catarrhal Inflammation of the bile-ducts, usually spreading from the duodenum. This, which is known as Catarrhal Jaundice, is one of the commonest forms of jaundice (§ 239).

III. Stricture, or obliteration of the duct owing to (1) congenital absence ; (2) perihepatitis ; (3) cicatrisation after ulcer of the duodenum ; (4) ulceration of the bile-duct, which may produce obstruction by the swelling around, or lead to stricture ; and (5) spasmodic stricture (?).

IV. Tumours pressing on the duct, such as (1) cancer and other tumours of the liver ; (2) enlargement of the glands in the transverse fissure of the liver ; (3) tumours of the stomach, pancreas, kidney, great omentum ; (4) fæcal masses in the intestines ; (5) pregnant uterus ; (6) ovarian tumours ; and occasionally (7) tumours growing from the walls of the ducts.

(*b*) **Jaundice due to Intra-hepatic Obstruction (Toxæmic Jaundice)** may arise in (1) cirrhosis of the liver (occasionally) ; (2) pneumonia ; (3) other acute specific fevers, especially tropical fevers, yellow fever, relapsing fever, and pyæmia ; (4) animal poisons, such as ptomaines or snake-bite ; (5) chemical poisons, such as phosphorus, mercury, antimony, arsenic, copper, chloroform and ether ; (6) acute yellow atrophy of the

[1] "Stadelmann, Der Icterus und seine verschiedenen Formen," Stuttgart, 1891.

[2] If the patient has resided at the Cape, or other country where the parasite is found, the stools may be examined for the eggs of the *Distoma hepaticum*. In these cases the eggs are very plentiful, and contained in every stool. Besides jaundice, which is often of an intermittent character, the patient complains of a dull aching in the hepatic region, and gastric disturbance.

liver; (7) protracted constipation. Jaundice also arises in newly-born children (icterus neonatorum), and in states of emotion and concussion of the brain, in a manner not understood.

To *diagnose which of the causes* of jaundice is in operation: 1. If possible, EXAMINE THE FÆCES, which are slate or clay-coloured in complete obstruction, and of normal colour in toxæmic jaundice. But it must be remembered, as possible fallacies, that the fæces may become stained if mixed with urine; and that the bile-duct may be only partially obstructed, and enough bile may thus escape to colour the fæces.

2. Inquire as to the HISTORY OF THE ATTACK. Jaundice coming on suddenly, especially in a middle-aged female patient previously in good health, almost invariably indicates obstruction by gall-stones (rare cases of nervous shock excepted). Jaundice coming on slowly, and ultimately becoming intense, is very generally due to a tumour pressing on the hepatic duct. A well-marked jaundice persisting some weeks is almost certainly obstructive. A history of previous temporary attacks points in adult life to gall-stones; in youth to "catarrhal jaundice."

3. EXAMINE THE HEPATIC REGION CAREFULLY. If the liver is enlarged, cancer is the most probable cause; interstitial hepatitis less commonly. If ascites be present, the diagnosis rests between cancer and cirrhosis.

4. Inquire as to PAIN AND CONSTITUTIONAL SYMPTOMS. Pain of a spasmodic and severe character accompanies jaundice due to gall-stones and cancer. It is more constant and gnawing in character in congestion of the liver and catarrh of the bile-ducts. The temperature is not often elevated, but it may be so in catarrhal jaundice, jaundice due to poisons in the blood, pyæmic hepatitis, tubercular affections, and local pus formations, such as inflamed hydatid. Cerebral symptoms are very rarely present, except when a fatal termination is at hand, unless the jaundice occurs in the course of pneumonia, fevers, or in that rare disease, yellow atrophy of the liver.

The *Prognosis* and *Treatment* of jaundice depend on its causal diseases (*q.v.*). The disappearance of bile from the urine indicates that the attack is coming to an end, though it may be some weeks before the skin clears. The flatulent dyspepsia and many of the concurrent symptoms may be relieved by the administration of ox-gall (5 to 10 grains or more) with meals, together with carminatives. The itching of jaundice is often a most troublesome symptom, but it can generally be relieved by pilocarpine.

§ 232. **Icterus Neonatorum** is a mild transitory form of jaundice which affects a very large number (estimated by various observers at from 70 to 90 per cent.[1]) of new-born infants. It appears usually on the second or third day of life, is not

[1] These statistics were taken from hospital cases, which were placed in circumstances where a better light probably resulted in the detection of the slightest tinging of the skin.

generally very intense, and rarely lasts longer than one or two weeks. The fæces are normal in colour, and apart from the jaundice the infant presents no other symptoms. The cause of the condition has been the subject of considerable debate, but the question is almost entirely an academic one, and the reader is referred to systematic works or to a publication by Runge.[1] The *Treatment*, if any is required, is the same as that for catarrhal jaundice.

A *severer form* of the same condition, sometimes erroneously called Icterus Gravis Neonatorum, occurring during the first week of life, may be due to (1) Congenital stricture of the bile-ducts by syphilitic perihepatitis ; (2) congenital absence of the duct ; (3) septicæmia ; (4) Winckel's disease (an epidemic form) ; or (5) acute fatty degeneration of the new-born (Buhl). The first and second are diagnosed by the intensity of the jaundice and the absence of bile from the fæces ; the remainder present other symptoms, such as hæmorrhages, purpuric spots, and (4 and 5) cyanosis.

PART B. PHYSICAL EXAMINATION.

The liver lies chiefly in the right hypochondrium ; the left lobe extends across the epigastrium above the stomach into the left hypochondrium. The gall-bladder lies below, in contact with, the liver, and is situated under the ninth right intercostal cartilage (see Figs. 61 and 71).

The routine methods of examination of the liver consist of INSPECTION, PALPATION, and PERCUSSION.

§ **233. Inspection** locally teaches us but little, as a rule, unless the symmetry of the abdomen as observed from the foot of the bed be altered. However, the presence or the absence of *jaundice* should always be noted in cases of suspected hepatic disease. If slight, it may be noticeable only in the conjunctivæ and urine. Note also if there are venous stigmata in the face or enlargement of the veins of the abdominal wall, such as occur with cirrhosis and portal obstruction.

§ **234.** During **Palpation** the patient should be placed in the recumbent posture, and, in order to obtain complete relaxation of the abdominal walls, he may be asked to " let his breath go." If this is not sufficient, the knees should be drawn up and the shoulders supported. Standing on the right side of the patient, place the palmar surface of the hand, previously warmed, on the right side of the abdomen, immediately above the iliac crest, pressing it firmly yet gently inwards. The tips of the fingers should be inclined slightly upwards and inwards towards the median line, and the *upper margin of the index finger* should be pressed firmly down, working little by little upwards towards the costal margin. In this way the upper border of the index finger, always held perfectly flat, will come in contact with the margin of the organ if it be enlarged. But if it is not enlarged, the liver cannot be felt, for it lies altogether beneath the costal margin in the adult. In young children, however, the liver is proportionately larger in all its dimensions, and the lower edge normally protrudes beneath the costal margin. If the liver is enlarged, try to feel its surface by gently dipping

[1] " Die Krankheiten der ersten Lebenstage," Auflage 2, 1893.

the fingers down. Notice if its surface is smooth (as in fatty liver) or nodular (as in cancer), or simply rough ("hobnail"). When there is fluid in the peritoneal cavity, this method of "dipping" the fingers (suddenly) is also useful; but in most cases the finger tips only excite contraction of the abdominal muscles, and so frustrate our object. The other fallacies of hepatic enlargement are mentioned under *Percussion* (below).

The *gall-bladder*, if enlarged, may be discovered as a round elastic tumour, projecting beneath the ninth rib, at its junction with the cartilage.

§ **235. Percussion** should be light, so as to elicit only the superficial or absolute dulness of the organ. In percussing the upper margin, start

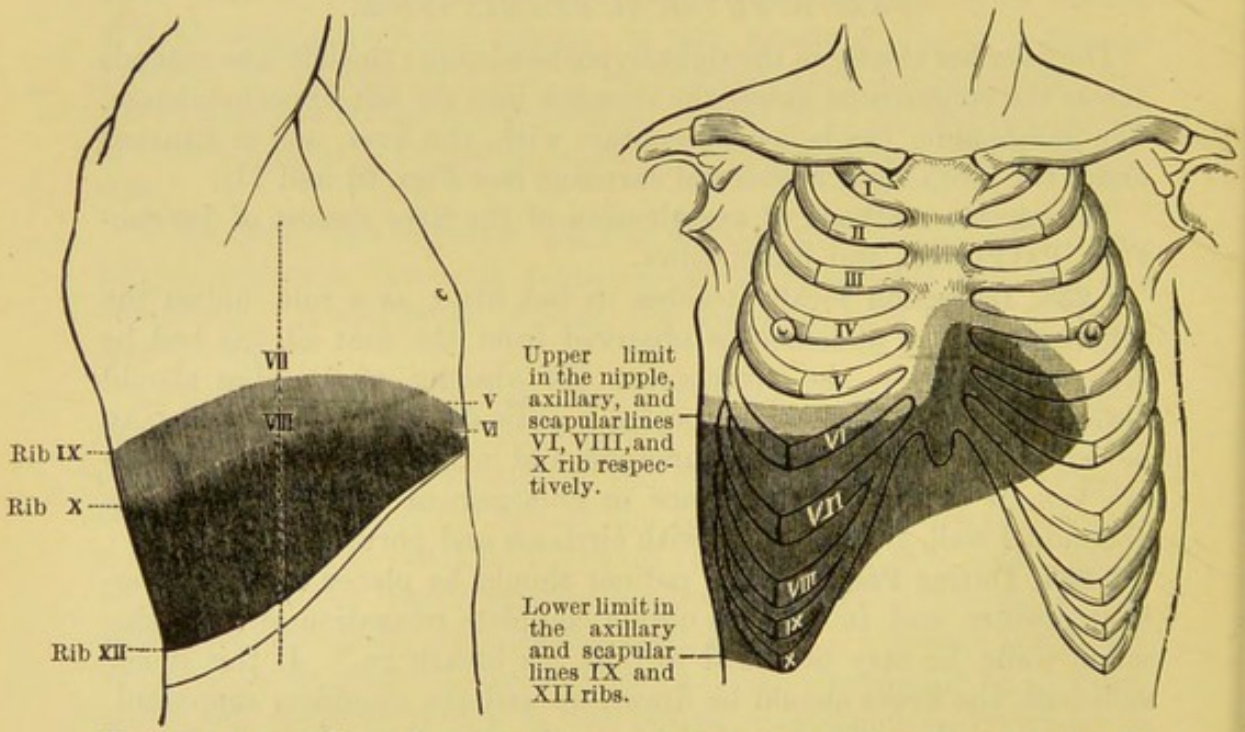


Fig. 71.—Area of Liver Dulness.—The *superficial* (or absolute) dulness corresponds to the deep shading; the area of *deep* (or relative) dulness is larger and includes the lighter shading.

where there is a good lung-note above, and percuss down from rib to rib in the nipple, mid-axillary, and scapular lines. Then repeat the process from space to space. In defining the lower edge, still lighter percussion should be used, and the examination should proceed from the tympanitic note of the intestine upwards towards the hepatic region. But the more certain method of detecting the lower edge is by palpation.

Normally, in the nipple line the *superficial* or absolute *hepatic dulness* commences two fingers' breadth below the nipple, and measures $3\frac{1}{2}$ to 4 inches, and in a routine examination this is the most important measurement to obtain. The normal boundaries of the liver are given in Fig. 71.

The *lower border* arches upwards just beneath the right costal margin and crosses the epigastrium, where the hepatic dulness becomes continuous with the cardiac dulness. In the mid-sternal line the dulness extends from ½ inch above the base of the xiphoid cartilage to about midway between the umbilicus and the xiphoid, where the lower edge may be felt by careful palpation when the abdominal wall is very lax. Thus the *absolute* dulness measures on an average about 2 *inches in the mid-sternal line* and 4 *inches in the nipple line.*

These landmarks do not indicate the deep dulness of the liver, which is more difficult, and in most cases less useful, to determine. But in some cases, such as abscess or hydatid, it is desirable to make out the deep (or *relative*) dulness of the liver by heavy percussion. The extreme height of the liver, as thus made out, corresponds to the fifth rib in the nipple line, seventh space in the mid-axilla, and ninth space in the scapular line.

Light Percussion Boundary (the one ordinarily used) gives the superficial or absolute dulness—*i.e.*, where the liver is in contact with the ribs.	Nipple line.	Mid-axillary line.	Scapular line.
Upper margin situated at the	6th	8th	10th rib.
Extent of dulness in vertical line	3½	4	3 inches.

Heavy Percussion Boundary gives the deep or relative dulness where the liver is covered by lung.	Space.		
Upper margin situated at the	5th	7th	9th rib.
Extent of dulness in vertical line	4	5	4 inches.

Fallacies.—The physician should never feel satisfied with mapping out the liver once only, because the organ may be temporarily affected by many varying conditions, and the *percussion* boundaries by no means always give us a true index. Thus the lower edge may be masked by the dulness of the stomach after a full meal, by an accumulation of fæces in the colon, or by a thickened omentum. Great rigidity of the muscles, or œdema of the abdominal walls, may also obscure the lower edge of the liver.

The beginner, by *palpating* with his finger tips, which excite muscular contraction, generally fails to make out the lower margin of the liver, even when the organ is enlarged. By *percussing too heavily he fails to get the absolute dulness.*

Apparent *diminution* of the liver may arise from (i.) distension of the stomach or intestines with gas ; (ii.) by contractions of Glisson's capsule, especially on the under surface, giving rise to puckering or distortion of shape anteriorly ; or (iii.) emphysema of the lungs, which obscures the *upper* border very much. Great diminution or absolute loss of the liver dulness, owing to gas in the peritoneal cavity, is a diagnostic feature of perforation of the stomach or intestine.

Apparent *enlargement*, when attention is paid solely to the lower edge of the organ, may be due to a *displacement* of the liver downwards (i.) by pleuritic effusion, emphysema, or pneumo-thorax ; (ii.) intrathoracic tumours ; or (iii.) enlargement of the heart or hydro-pericardium. These and other fallacies may arise from paying attention solely to the *lower edge* of the organ ; and, finally, the liver may in rare cases be dropped or "*floating.*" "Riedel's lobe" is mentioned under Abdominal Tumours.

§ **236. Fluid in the Peritoneum** (Ascites) is a frequent accompaniment of some hepatic disorders, and its presence or its absence must always be carefully noted. The methods of investigating this important matter have already been given (§ 184).

Ascites (Dropsy of the Peritoneum) is one of the **Evidences of Portal Obstruction,** and these are more frequently associated with some disease of the liver than of any other organ. Sometimes they are the only evidences we have of hepatic disorder.

The Signs of Portal Obstruction are, in the order in which they appear : (1) A liability to attacks of *gastric* and *intestinal catarrh*, as evidenced by irritable dyspepsia, and the vomiting of mucus, streaked perhaps with blood, in the early morning before breakfast. (2) *Hæmorrhage*, sometimes in very large quantity, from the stomach and the bowels. (3) *Hæmorrhoids* may occur in other diseases (§ 225), but they are frequently associated with portal obstruction. (4) Attacks of *congestion of the liver.* (5) Congestion, and therefore enlargement, of the *spleen.* (6) *Ascites* (see below). (7) *Enlargement of the veins* of the abdominal wall from the establishment of a collateral circulation. (8) *Œdema* of the legs is a secondary and indirect result of the pressure of the ascitic fluid on the large veins within the abdominal cavity. (9) *Albumen* in the urine may arise in the same way, or from concurrent disease of the kidney.

Ascites, it will be observed, is a late sign of portal obstruction. It has already been fully described (§ 185), and it will be remembered that its *three principal causes* were Cardiac, Hepatic, and Renal disease. (1) In *cardiac disease* the ascites will have been preceded by dropsy of the legs ; (2) in *hepatic disease* the ascites is the predominating feature, though it may be followed by dropsy of the legs ; while (3) in *renal disorders* the ascites is only part of a dropsy which is general from the outset. *Cancer of the peritoneum* may also produce ascites, but here the nodules of cancer will probably be felt on palpation, and there will be other symptoms of cancer. Ascites may have to be diagnosed from an ovarian or other large abdominal cyst, and from fat in the omentum (§ 182).

Portal obstruction, and consequently ascites, are not present with equal frequency in all diseases of the liver, and in some they are absent. In order of frequency they are as follows :

(1) Cirrhosis, or an increase of the interstitial tissue (usually due to alcohol), is by far the commonest cause of portal obstruction, by producing pressure upon the minute branches of the portal vein within the liver.

(2) In Cancer of the liver portal obstruction and ascites are fairly frequent, but they are due not so much to the cancer within the liver as to the pressure of enlarged glands in the transverse fissure of the liver upon the portal vein, or to secondary involvement of the peritoneum.

(3) Perihepatitis may occasionally produce constriction of the portal vein by puckering at the fissure.

(4) Simple Congestion of the liver may be attended by hæmorrhoids and gastric catarrh, but rarely by much ascites.

(5) Fatty and Waxy Liver, Abscess, and Hydatid are hardly ever attended by portal obstruction.

The other *Causes*, the *Prognosis*, and *Treatment* of Portal Obstruction have been described under Ascites (§ 185).

In cases of hepatic disease the **urine** should always be tested for *bile* (§ 282) and for urates (§ 291), sometimes for leucin and tyrosin ; and the amount of urea may need to be estimated (§ 280).

PART C. DISEASES OF THE LIVER.

§ **237. Routine Procedure.**—First : Ascertain *what is the patient's Leading Symptom.* The symptoms of disorder of the liver we discussed in Part A.—*e.g.*, gastric disturbance, pain (or a feeling of weight or discomfort in the hepatic region), or jaundice. If there be severe and paroxysmal pain, turn first to biliary colic (§ 241).

Secondly : Learn the *History* of the patient's illness, eliciting the facts in chronological order, and in this way ascertain the important fact whether the disease be *acute* or *chronic*, because disorders of the liver may be conveniently classified into these two groups.

Thirdly : The Examination of the Liver must next be made. The routine method is as follows :

1. Ascertain whether the liver is *enlarged* or *diminished* (by percussion in the nipple line, and abdominal palpation), and whether there is any *pain*, *tenderness*, or other abnormality.
2. Ascertain whether there be any *fluid* in the peritoneum (§ 184).
3. Ascertain if there is any *jaundice* (§ 231), and examine *the urine* for bile pigments, lithates, and the diurnal amount of urea.

Classification.—For clinical purposes, diseases of the liver may be conveniently divided into Acute and Chronic Disorders.

If the illness is one of long standing, and has come on insidiously, the reader should turn to **Chronic Diseases of the Liver** (§ 248). The **acute diseases** will be first described.

ACUTE DISEASES OF THE LIVER.

If the illness has come on more or less suddenly, and is attended by considerable malaise or other constitutional symptoms, it is one of the **acute diseases of the liver or bile ducts**, probably : I. Acute Congestion ; II. Catarrhal Jaundice ; or III. Gall-stones. The less common acute diseases are : IV. Perihepatitis ; V. Abscess ; and VI. Acute Yellow Atrophy.

I. *The patient complains of* pain *or* discomfort in the hepatic region, *the liver area may be* increased, *slight jaundice and numerous vague* dyspeptic symptoms *are present, but there is little or no fever.* The disease is probably Acute Congestion of the Liver.

§ 238. Acute Congestion of the Liver.—Clinically, there are two kinds of congestion of the liver—an active or acute congestion, and a passive or mechanical congestion. *Active* or *arterial* congestion (with which we are now concerned), is usually met with in the form of acute attacks due to dietetic errors ; though it may sometimes occur as a more subtle and sometimes latent condition in a subacute or chronic form which eventuates in cirrhosis. *Passive* or *venous* congestion is due to obstructed venous return (mostly in chronic cardiac or pulmonary disease) ; it is in the nature of things a chronic process, and will be considered under Chronic Diseases (§ 255).

TABLE XVIII.—ACUTE DISEASES OF THE LIVER.

	Jaundice.	Enlargement of the Liver.	Ascites.
I. ACUTE CONGESTION.	Not very great.	Slight increase.	Usually absent.
II. CATARRHAL JAUNDICE.	Always present and marked.	Slight increase.	Absent.
III. GALL-STONES.	Very marked in most cases.	May be considerable increase.	Absent.
IV. PERIHEPATITIS.	Absent.	None unless another cause.	Usually absent.
V. ABSCESS OF LIVER.	Generally present.	Moderate and irregular enlargement.	Usually none.
VI. ACUTE YELLOW ATROPHY (very rare).	Very marked.	Liver diminished in size.	Absent.

Symptoms.—(1) The onset is usually somewhat sudden, after a series of indiscretions in diet, especially in the matter of alcohol. The patient complains of pain, or a feeling of weight or uneasiness in the region of the liver, and he may be unable to lie on the left side. (2) There is generally a slight but uniform enlargement of the liver, and some degree of tenderness. (3) Slight jaundice is present on the second or third day in the majority of cases, but it is never so intense as in catarrhal jaundice or gall-stones. The fæces are dark in colour, owing to the presence of bile. (4) Certain gastro-intestinal symptoms are present—nausea, headache, furred tongue, a bitter taste in the mouth, and flatulence ; the bowels are usually constipated ; the urine is scanty, high coloured, and deposits lithates on standing ; and there is usually some depression of spirits and irritability of temper.

Etiology.—(1) By far the most frequent cause is alcoholic excess. Constant indulgence in rich foods containing fat, sugars, and spices may also produce congestion. (2) Residence in hot climates, especially when associated with malaria and dietetic errors ; but many attribute to the climate what is really due to alcohol or faulty diet. (3) Suppres-

sion of an habitual discharge, especially bleeding piles or menstruation ; and (4) dysentery and febrile states are often accompanied by congestion. (5) Sudden or protracted chill and (6) injury have been mentioned as causes. An attack of acute congestion may be *predisposed to* by (i.) the presence of chronic congestion (§ 255) ; (ii.) previous attacks of malaria ; (iii.) indolent or sedentary habits.

The *Diagnosis* is based upon the occurrence of symptoms of gastro-intestinal disturbance in association with pain and enlargement of the liver. In *perihepatitis* the first-named are absent, the pain is much more acute, and syphilis is probably in operation. The diagnosis from the other acute hepatic disorders is given in Table XVIII. (*ante*). The symptoms of *pleuro-pneumonia* at the onset may include jaundice and the other symptoms of acute congestion of the liver, for which, indeed, this disease may be mistaken. It is important, therefore, to examine the base of the right lung in all such cases when associated with jaundice.

Prognosis.—Acute congestion is very apt to recur, especially if the patient continues his dietetic indiscretions. The intervals between the attacks become shorter, and the condition is followed by subacute or chronic congestion, and, eventually, cirrhosis. An attack of moderate severity rarely lasts more than a week or two. Unless a condition of cirrhosis is reached (when enlargement is checked to some extent by the shrinking of the newly-formed fibrous tissue), the degree of congestion may be fairly estimated by the amount of enlargement.

Treatment.—The indications are (1) to relieve the congestion of the portal system, and (2) to correct dietetic errors. To relieve the congestion, saline purgatives are specially indicated, such as the sulphates of magnesia, potash, soda, or the bitartrate of potash. Carlsbad, Friedrichshall, or Hunyadi Janos water should be taken every morning early, and a full dose of calomel, podophyllin, or pil. hydrarg. at night. In severe cases leeches, or dry or wet cupping in the region of the liver, may relieve the pain considerably. Leeches are sometimes applied to the margin of the anus, but this is not always convenient. Murchison recommended ammonium chloride in doses of 20 grains two or three times a day, to induce free diaphoresis, and diminish the portal congestion and pain (F. 46, 51, 53, and 66 may be useful). Ipecacuanha is in great repute among Indian physicians, and is given in large doses, as in dysentery (20 to 30 grains every six or twelve hours), preceded, half an hour before each dose, by ½ grain of opium to prevent vomiting. For the gastric symptoms, alkalies, the alkaline salts, carbonate of magnesia, bismuth, and hydrocyanic acid are useful. Nitro-hydrochloric acid and nux vomica are also useful in convalescence. The diet during the attack should be of the simplest, consisting at first solely of 2 pints of milk a day. Alcohol in any form should be strictly forbidden. As the patient recovers, a simple regimen should be prescribed (§ 212).

II. *The patient, who is* YOUNG, *has suffered from* GASTRO-INTESTINAL DISTURBANCE *for some days or weeks, when* JAUNDICE, WITH CLAY-COLOURED STOOLS, *sets in somewhat suddenly, without local pain, and with little or no enlargement of the liver.* The disease is probably CATARRHAL JAUNDICE.

§ **239. Catarrhal Jaundice** (Acute Cholangitis) is jaundice due to inflammatory swelling of the lining membrane of the bile-ducts, and the consequent obstruction to the outflow of bile.

Symptoms.—(1) The jaundice is usually of sudden onset, though it is preceded for a shorter or longer time by signs of gastro-intestinal disorder. (2) The jaundice is often very intense, but in mild cases the degree corresponds to that of congestion of the liver. It generally begins to subside in the course of two or three weeks. If it lasts longer, some other cause (§ 231) should be suspected. (3) The stools are pipe-clay-coloured, and the urine is dark with bile. (4) Nausea and loss of appetite, flatulence, and constipation are generally present. (5) A feeling of uneasiness or weight in the hepatic region is usually complained of. There may be slight enlargement of the liver, the edge being smooth, firm, and tender. The spleen may be slightly enlarged. (6) There may be slight fever at the commencement, but it usually subsides before the patient is seen, and the pulse is abnormally slow.

Etiology.—(1) Extension of inflammation from the stomach and duodenum along the bile-ducts secondarily to gastric derangement is the most common cause of catarrhal jaundice. (2) It is the by far commonest form of jaundice met with in children and young adults. (3) Exposure to chill. (4) It may be secondary to congestion (§ 238, *ante*) or cancer. (5) Catarrhal jaundice frequently follows the passage of a gall-stone. (6) In the adult gout or gouty conditions are said to predispose.

Diagnosis.—Catarrhal jaundice may have to be diagnosed in an old person from *cancer*, but in the latter the jaundice comes on slowly, with pain, and it lasts many months. In *gall-stones* there is biliary "colic" (§ 241). In *congestion* of the liver the jaundice is less marked, and the fæces are not clay-coloured.

Prognosis.—The disease is never fatal. It usually terminates in a few weeks, after the gastric disorder has been relieved. The outlook is unfavourable only when catarrhal jaundice complicates other maladies, such as cancer or gall-stones.

Treatment.—(1) Remove any cause of the concurrent gastro-enteritis, and allay the condition with alkalies, alkaline carbonates, rhubarb, or bismuth. (2) A brisk mercurial purge, followed by a saline once or twice a week, helps to relieve the congestion, both of the intestines and the liver. (3) Ox-gall, creosote, or salol are sometimes useful as intestinal antiseptics. Sodium salicylate and ammonium chloride (10 grains of each) is very effective. (4) Rectal injections of one or two

pints of water daily (60° to 90° F.), retained as long as possible, have been recommended to allay any intestinal irritation. The prescriptions and much of the treatment for Congestion (*supra*) are applicable.

§ **240. Epidemic Jaundice** (Synonyms : Weil's Disease, Septic Jaundice (Fraenkel), Infective or Febrile Jaundice) is characterised by a sudden sharp febrile attack, accompanied by severe pains in the limbs, and rapidly followed by jaundice, swelling of the liver and spleen, and nephritis ; and ending gradually in recovery about the tenth or fifteenth day. Clinically, it resembles a severe transitory form of catarrhal jaundice occurring epidemically. Cases of what was probably the same disease were first described by Weiss in 1866.[1] In 1886 Professor Weil, of Heidelberg,[2] described four cases of a peculiar form of acute infective disease characterised by jaundice, which most German authorities regarded as a disease not hitherto observed.

Symptoms.—The malady comes on suddenly, with marked prostration, headache, and sometimes delirium. The muscular pains, especially in the legs, are among the most noticeable features, and may obscure the other symptoms. The jaundice appears on the second or third day, reaches a moderate degree, lasts about fourteen days, and then disappears gradually. The stools are generally clay coloured. The liver is considerably enlarged and tender, the spleen enlarged, and the urine contains albumen, epithelial casts, and sometimes blood. The temperature reaches 103° or 104° F. on the second or third day, and begins to fall between the sixth and ninth. Various rashes and occasionally epistaxis have been observed.

Etiology.—There seems to be no doubt that the disease is infectious. It has always occurred in an epidemic form, chiefly amongst men between the ages of fifteen and thirty, and especially working-men. Most epidemics have occurred in the summer months. The nature of the infection is not yet identified, though Jaeger[3] succeeded in discovering an organism, to which he gave the name of bacillus proteus fluorescens, in the urine and organs in several cases. The same observer found that the ducks and geese about the river in which the affected patients had bathed were subject to a fatal disease with marked jaundice. Dr. William Hunter points out (*loc. cit.*) that a very similar jaundice can be produced experimentally in dogs by toluylendiamin, in whom it also produces swelling of the spleen and liver, and nephritis. In both Weil's disease and poisoning by this reagent the duodenum is frequently found to be the seat of considerable congestion.

Inferentially, the *Treatment* of this Epidemic Jaundice would appear to resemble that of Catarrhal Jaundice.

III. *The patient, usually an elderly female, is suddenly seized with* PAROXYSMS OF SEVERE PAIN *in the hepatic region, and in the course of twelve to twenty-four hours she becomes* JAUNDICED, *the stools becoming clay-coloured.* The attack is one of BILIARY COLIC.

§ **241. Gall-Stones** and **Biliary Colic.**—Gall-stones are concretions which form in some part of the biliary passages, most commonly in the gall-bladder. CHOLELITHIASIS is the condition in which gall-stones are developed. When gall-stones move along any of the ducts, they give rise to Biliary Colic.

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[1] "Zur Kenntniss und zur Geschichte der sogenannten Weilschenkrankheit," *Wien. med. Woch.*, 1890, Bd. xl.

[2] "Ueber eine eigenthümliche, mit Milztumor, Ikterus, und Nephritis einhergehende akute Infectionskrankheit," *Deut. Archiv für Klin. Med.*, vol. xxxix., 1886.

[3] Quoted by Dr. William Hunter in Allbutt's "System of Medicine," vol. iv., p. 95.

GALL-STONES may vary in size from particles hardly larger than a sand-grain to the size of a golf-ball. When they are solitary, they are round or oval in contour. It is important to notice the presence of facets or flattenings of their surface, caused by the pressure of one against the other, because this indicates that there has been more than one stone in the gall-bladder or bile-ducts. Their colour varies from a yellow to a dark brown, and their chief physical characteristics are the smooth " soapy " surface, the ready way in which they crumble between the thumb and finger (though sometimes they are very hard), and their lightness as compared with renal calculi. They generally consist chiefly of cholesterin mixed with a combination of calcium and bile pigment, but are sometimes either pure cholesterin, pure bilirubin, or pure calcium carbonate. Strong sulphuric acid when added to cholesterin crystals gives a ruby red at the junction. But the *appearance* of the crystals is the most characteristic feature about them, consisting as they do of rectangular plates, broken by irregular rectangular fractures (Fig. 72).

Biliary Colic.—Symptoms may be absent when the stone is at rest, but when it begins to move (i.) the pain is agonising; it starts in the epigastrium and shoots into the right hypochondriac region towards the spine and up to the right shoulder, but never passes downwards.

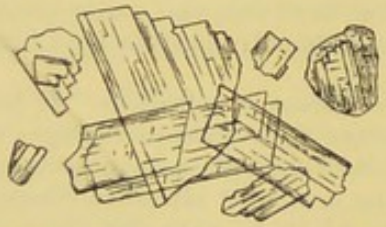

Fig. 72.—Cholesterin Crystals. Microscopic appearance presented by fragments of gall-stones in the fæces.

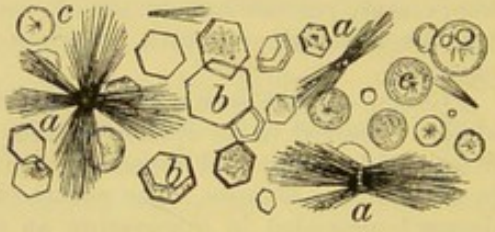

Fig. 73.—*a*, TYROSIN, in bundles of needle-shaped crystals, and *c*, LEUCIN, spherical crystals with concentric markings, found in the urine in rare cases of acute yellow atrophy of the liver. *b*, CYSTIN (clear six-sided plates), is a rare urinary deposit due to an inborn error of metabolism. It may form renal calculi.

The paroxysm is usually so severe that the patient is in a state of partial collapse, with vomiting, hiccough, subnormal temperature, and a quick weak pulse. Sometimes there is a rigor, and the temperature rises a few degrees. Between the paroxysms of acute pain there is a constant dull aching and tenderness over the hepatic region. The attack lasts from a few hours to a few days. (ii.) The liver may be enlarged, and if a stone becomes impacted in the hepatic duct the enlargement may be considerable. (iii.) Jaundice usually appears twelve to twenty-four hours after the paroxysm, and lasts from a few days to a few weeks. It is most intense when the stone is impacted in the common duct.

The *Symptoms* which arise vary somewhat with the *position of the gall-stone* (Fig. 74). Thus: (i.) If a stone is impacted in the *common duct*, there are biliary colic, marked jaundice, and a distended gall-bladder, and if the impaction continues the liver becomes enlarged. (ii.) If a gall-stone be impacted in the neck of the gall-bladder (*i.e.*, in the *cystic duct*), biliary colic is present without jaundice. In time the

gall-bladder may be distended with mucus, and form a definite abdominal tumour. (iii.) Stone impacted in the *hepatic duct* is rare. It causes biliary colic and jaundice, but the gall-bladder is not distended. (iv.) Stones occasionally form in the radicles of the hepatic ducts, and give rise to indefinite symptoms, sometimes without pain, and usually without jaundice. (v.) Sometimes small particles of cholesterin (biliary sand) in the *gall-bladder* may give rise to recurring paroxysms of pain, unaccompanied by any other symptoms, which defy diagnosis.

Diagnosis of Biliary Colic.—It is distinguished from the two other forms of colic in Table XIII., § 172. The severity of the pain and its paroxysmal character usually distinguish it from all other acute diseases of the liver. Pseudo-biliary colic is sometimes met with in nervous women. The diagnosis from *cancer* of the liver may be very difficult.

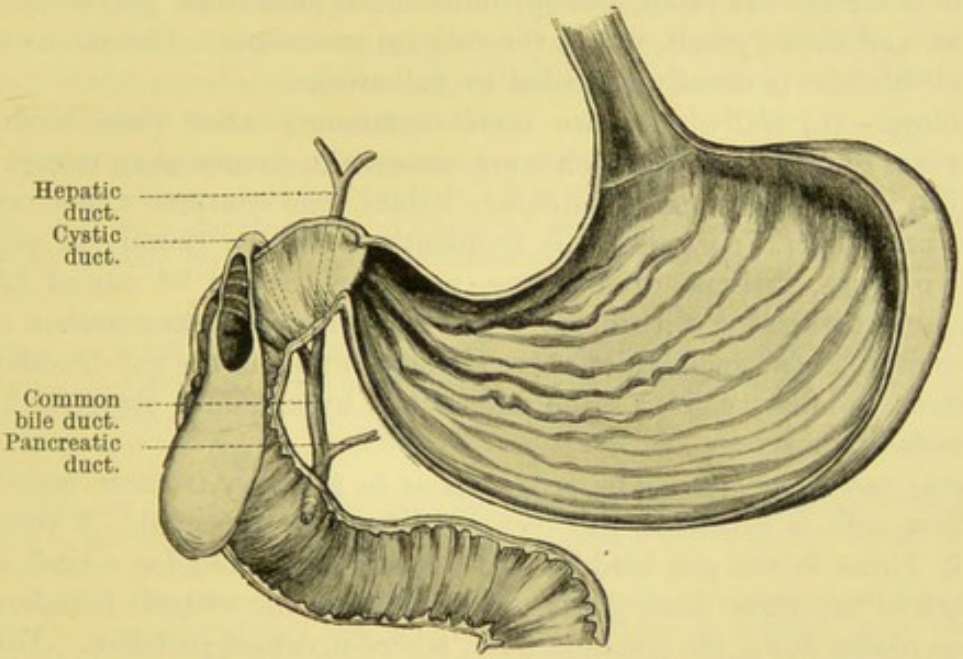

Fig. 74.—The STOMACH AND DUODENUM opened to show the ducts in connection with the Liver and Pancreas.

Both occur at the same age, and both cause jaundice; further, cancer may follow after years of trouble from gall-stones. In cancer, the jaundice comes on and steadily gets more and more intense. It must be remembered that in some cases gall-stones are passed without colic, but with jaundice; consequently, *recurring attacks* of jaundice in an elderly woman should lead one to suspect gall-stones. In all suspected cases the stools should be carefully examined for stones. *The presence of ascites* points to cancer, for it rarely exists long without the effusion of fluid into the peritoneum.

The *Symptoms* of GALL-STONES AT REST in the GALL-BLADDER (CYSTO-CHOLELITHIASIS) are often very obscure, and occurring as they do in elderly females are very apt to be mistaken for cancer. (1) Enlargement and tenderness of the gall-bladder can generally be made out

below the ninth costal cartilage, unless it is obscured by adhesions, or by Riedel's lobe, a local hypertrophy of one lobe of the liver sometimes associated with chronic cholelithiasis (see § 188). But, on the other hand, the enlargement of the gall-bladder may be mistaken for cancer, from which it can be distinguished only by the long duration of the illness. (2) Attacks of "biliary fever"—*i.e.*, "chills," or shivering, with slight rises of temperature of a malarial type—at intervals for months or years are perhaps the commonest complaint. (3) Local pain or discomfort is not always present, but, like (1), it may from time to time be produced or aggravated by exertion. (4) For the rest, the symptoms are negative—no jaundice, ascites, or other symptoms—only a condition of general ill-health, due to septic absorption from the ulcerating and irritated gall-bladder. (5) The stones may on rare occasions become encysted, but far more often ulceration, perforation, abscess, and fistula result, unless the surgeon intervenes. Carcinoma of the gall-bladder is usually preceded by gall-stones.

Etiology.—(i.) Gall-stones are much commoner after than before fifty years of age ; (ii.) are much more common in women than in men ; and (iii.) in stout persons of sedentary habits, who consume a diet rich in fat and sugar. (iv.) There is frequently a history (family or personal) of gout, asthma, or migraine, and Sénac found 98 out of 128 cases were associated with urinary gravel. They are less common in hot countries. When gall-stones are already formed in the gall-bladder, an attack of biliary colic is often determined by a sudden strain or an overloaded stomach.

Course and Prognosis.—The prognosis as to recovery from an attack of biliary colic is excellent, but recurrence may be expected. A stone usually forms in the gall-bladder and becomes impacted for a time in the neck of the cystic duct, giving rise to biliary colic without jaundice. It then passes down the common duct, where it causes jaundice. This rarely lasts more than a few weeks, but rare cases have been reported where it lasted two years. Impaction for any length of time leads to consequences, which may be classified thus : (i.) *Ulceration* of the ducts, with pyrexia, or abscesses of the liver and bile-ducts, and consequent subacute pyæmia ; (ii.) *perforation* into adjacent tissues, leading, for example, to fatal peritonitis ; (iii.) inflammation and *abscess* of the gall-bladder, which may open externally, perforate into the peritoneum, or ulcerate into the intestines ; (iv.) formation of *fistula* between the gall-bladder and the colon or duodenum, through which stones can pass of such a size that they may cause intestinal obstruction.[1]

Treatment.—(*a*) *During the attack* anodyne treatment is called for. Opium or hypodermic of morphia and atropine should be given (F. 25).

[1] Large gall-stones may gradually ulcerate through from the gall-bladder to the duodenum, in some cases almost without symptoms. Murchison collected some thirty-four of such cases where the gall-stone was large enough to give rise subsequently to intestinal obstruction.

Chloroform inhalations are used in severe cases. Other drugs recommended are chloral, spiritus ætheris, antipyrin, and extractum belladonnæ. Of late years olive-oil, in doses of at least 6 ounces, has been strongly recommended as causing rapid passage of the stone into the duodenum, but the author's experience does not support this. Hot water, with 1 drachm of sodium bicarbonate to the pint, may be tried. If the patient is put into a warm bath (100° F.), and kept there till he shows signs of weakness, an attack of pain may be warded off. Hot turpentine stupes may give relief.

(*b*) *Between the attacks* the habits of the patient must be corrected. Wine, beer, and sugar must be avoided. A prolonged course of alkalies, and sodium salicylate, or of such mineral waters as Vichy and Carlsbad, is advisable. Turpentine (℥ x.), in capsules, is said to aid the alkaline treatment. The treatment for Congestion (§ 238) is applicable. Surgical treatment is necessary whenever there is suppuration, when the gall-bladder remains distended, when the common duct is blocked, or when biliary colic frequently recurs.

§ 242. **Diseases of the Gall-bladder** are chiefly manifested by pain and enlargement or swelling of the gall-bladder, which first appears just beneath the tip of the ninth rib. For the fallacy of Riedel's lobe see Abdominal Tumours, §§ 187, 188. Mayo Robson[1] gives the following classification of diseases of the Gall-bladder and Bile-ducts :

A. **Catarrhal Inflammations** : (*a*) Acute Catarrh (which corresponds to Catarrhal Jaundice, § 239) ; (*b*) Chronic Catarrh. B. **Suppurative Inflammations** : (*a*) Suppurative Catarrh, which may consist of—(α) Simple empyema, and (β) suppurative cholangitis ; (*b*) Ulceration, Perforation, and Stricture of the gall-bladder and bile-ducts ; (*c*) Acute Phlegmonous Inflammation and gangrene of the gall-bladder.

Chronic Catarrh of the gall-bladder presents symptoms resembling those due to gall-stones within it (*vide supra*), but there is less pain, very slight jaundice, and no tenderness or pressure over the region of the gall-bladder.

Simple Empyema of the gall-bladder, without involvement of the hepatic ducts, is nearly always due to gall-stones. There is swelling, with continual localised pain and tenderness ; and the abscess may burst in various directions, or point externally.

Suppurative Cholangitis is practically indistinguishable from pyæmic abscesses (§ 244).

Ulceration of the gall-bladder is referred to above under Gall-stones (§ 241), and Perforation is usually a result of the same lesion.

Stricture is generally also a consequence of the ulceration following gall-stones, but it may sometimes be due to other lesions (see § 231). The result depends upon the position of the stricture. In the *cystic* duct it leads to distension of the gall-bladder. In the *common* duct it leads *both* to distension of the gall-bladder and considerable enlargement of the liver. It is rare in the *hepatic* duct, where it produces enlargement of the liver.

Acute Phlegmonous Inflammation of the gall-bladder (phlegmonous cholecystitis) is a rare affection (Courvoisier collected only seven cases). It comes on suddenly, with symptoms resembling perforative peritonitis, and is difficult to diagnose from acute appendicitis. It is usually rapidly fatal.

Membranous or Fibrous Cholecystitis has been recorded by Dr. H. D. Rolleston secondary to retained gall-stones.[2]

[1] Allbutt's "System of Medicine," vol. iv.

[2] Report of Path. Soc. London, the *Lancet*, May 24, 1902.

The less common **Acute Disorders** of the Liver remain to be considered, viz., PERIHEPATITIS, ABSCESS OF THE LIVER, and ACUTE YELLOW ATROPHY.

IV. *The patient complains of* PAIN AND TENDERNESS *in the hepatic region, aggravated by movement. There is* NO JAUNDICE, *and other hepatic symptoms are absent.* The malady is probably PERIHEPATITIS.

§ 243. Perihepatitis is inflammation of the capsule of the liver, which becomes opaque and thickened, and by its contraction may lead to considerable distortion of the shape of the liver.

Symptoms.—(i.) Acute attacks usually set in suddenly, with pain in the hepatic region, radiating to the shoulder, and there is tenderness, increased on movement, pressure, or cough. (ii.) Fever is absent as a rule, and the patient may appear to be in his usual health. (iii.) Friction may be felt or heard. (iv.) Unless some other disease is present, there is no jaundice. Recurrent attacks lead to thickening of the capsule, recurring ascites, necessitating repeated tapping, and occasionally jaundice. The puckered liver, with its thickened, rounded, distorted edge, can sometimes be made out. The history of a *Cause*, especially *syphilis*, is usually obtainable. It is sometimes part of an inflammation of the liver itself, or is associated with an abscess, tumour, or cirrhosis. Sometimes the inflammation extends from adjacent organs, as in pericarditis, pleurisy, or gastric ulcer, or it may be part of a general peritonitis. Perihepatitis occasionally complicates acute or subacute rheumatism.

Diagnosis.—The characteristic pain and the absence of jaundice differentiate it from many other liver diseases. The conjunction of syphilis is also very helpful. Cases of cysto-cholelithiasis (p. 363) or gumma of the liver may at times be mistaken for perihepatitis.

Prognosis.—Simple cases tend to recover. In cases which have lasted for a long time a certain amount of cirrhosis of the liver ensues. Portal obstruction may ultimately result from puckering at the fissure, and considerable distortion of the liver may result in the same way.

Treatment.—The diet must be spare, and the patient must be kept warm. Salines are given with blue pill and rhubarb. Externally, hot fomentations and poultices give relief, and if the pain is severe, leeches are recommended. The cause when known must be treated—*e.g.*, syphilis with iodide of potassium.

V. *There is* ENLARGEMENT *of the liver, accompanied by* PAIN *and tenderness, and the boundaries of the area of dulness are* IRREGULAR; *there are* SHIVERINGS, SWEATING, *and* INTERMITTENT PYREXIA. The disease is ABSCESS OF THE LIVER.

§ 244. Abscess of the Liver.—Solitary or multiple collections of pus may occur in the liver, due to septic infection, to suppuration of the bile channels, or portal vein, or more rarely to suppuration of pre-existing morbid conditions, such as hydatids or gummata. "Tropical" abscess occurs after dysentery, and is due to the amœba coli; it is usually solitary, whilst pyæmic abscesses are usually multiple.

Symptoms.—(i.) The onset is usually *acute*, with pain and tenderness of the liver, accompanied perhaps by a dry cough, with shallow respiration and digestive disturbance. The pain is affected by respiration, and is worst when the patient lies on the left side. (ii.) The liver is enlarged, and the enlargement may extend downwards, or more often upwards, even to the nipple. There may be fluctuation. (iii.) More or less jaundice is present as a rule. (iv.) Constitutional symptoms are marked. There is usually high fever, continuous at first, then with increasing oscillations. Rigors and sweats are common. Later on the patient falls into the typhoid state, with emaciation, vomiting, diarrhœa, and delirium.

Besides the acute type just described, there is an *asthenic* variety, with insidious

onset, general failure of the health, and periods of continuous or intermitting fever, followed by intervals of apyrexia resembling ague. Cough and dull aching over the liver and in the right shoulder are generally present from the beginning.

Diagnosis.—(i.) The pain and pyrexia distinguish abscess from *hydatid* (when not in a suppurating condition). (ii.) A distended and *inflamed gall-bladder* is recognised by a history of gall-stones, and its outline may be palpable on examination. (iii.) Abscess is often mistaken for severe *ague*. But ague is amenable to quinine, the elevations of temperature are periodic, and each paroxysm has three stages. (iv.) A hepatic abscess may be diagnosed from other swellings of the liver by exploratory aspiration, giving the reddish " anchovy sauce " coloured pus, which is distinctive. (v.) A low form of pneumonia at the base of the right lung so frequently accompanies liver abscess that its presence is an important aid to diagnosis in obscure cases.

The insidious cases of liver abscess are always difficult to diagnose, and where health is deteriorating, with obscure pyrexial conditions from time to time, almost every general or local inflammatory disorder may be suspected before liver abscess. Manson advises the physician in the tropics to suspect liver abscess in all obscure abdominal cases with evening rise of temperature. On the other hand, cases have occurred where medical men, diagnosing abscess, have explored the enlarged livers of *leucocythæmia* and *pernicious anæmia*. This mistake may be avoided by examining the blood before resorting to puncture.

Etiology.—(i.) Hepatic abscess may arise by the suppuration of a pre-existing tumour, such as hydatid, or gumma, or the ray fungus. The general opinion is that gummata of the liver do not suppurate ; yet they suppurate elsewhere, as in the skin. (ii.) Ulceration of the biliary passages, as from gall-stones or ulcerations of the alimentary canal, dysenteric or simple ulcers, are potent causes of the multiple or " pyæmic abscesses." (iii.) Occasionally operations on the rectum are followed by abscess of the liver. (iv.) Injury to the liver. (v.) Pyæmia. (vi.) The chief form of abscess in warm climates is the large and solitary *tropical* abscess which is associated with dysentery. As many patients with dysentery never develop liver abscess, it is now believed that, while dysentery is the predisposing cause, there must also be some other agency as a determining cause. That hepatic congestion plays some part is borne out by the fact that, whereas abscess rarely follows dysentery in women, children, or natives, it is the usual sequence of dysentery in men who have indulged in alcohol and highly-spiced foods.

Prognosis.—(1) The case mortality varies from 57 to 80 per cent. Death usually takes place in three weeks in cases with multiple abscesses. The pyrexia increases, and the patient dies in the typhoid state. (2) Solitary abscess may lead to death in a month, or the patient may live for one or two years, with obscure symptoms as described above. (i.) The abscess may burst into the peritoneum, pericardium, or alimentary canal, with a fatal issue, or it may open externally and gradually recover by free discharge. (ii.) Frequently the abscess, especially a " tropical " abscess, bursts into the right lung. The patient develops a severe cough, with signs of consolidation of the right pulmonary base, and the abscess contents are brought up as a red-coloured sputum. Recovery may result, or the continued discharge may lead to death from exhaustion or lardaceous disease.

Treatment.—When the evidence points simply to acute inflammation of the liver, before the temperature leads one to suspect pus formation, anodyne treatment, such as cupping and hot poultices are employed. Ammon. chlor. (20 grains t.i.d.) or ipecacuanha (if dysentery be present) are the best drugs. Saline purgatives, spare diet, and absolute rest in bed are necessary. As soon as an abscess is suspected, exploratory puncture must be performed ; at least six punctures should be made before abandoning the attempt to find pus. If an abscess is discovered, free drainage must be established. Sir Patrick Manson advises the use of a large trocar, through which a drainage-tube is inserted.[1] Recently Leonard

[1] Report of Med. Chir. Soc., the *Lancet*, May 17, 1902.

Rogers has replaced drainage by flushing out, two or three times daily, with a solution of quinine.

§ **245. Subphrenic Abscess** (Abscess beneath the Diaphragm).—The *Symptoms* are much the same as those of tropical liver abscess. When occurring above the right lobe, the liver dulness is continued up in the axilla, perhaps as far as the level of the nipple, and is convex, or dome-shaped, upwards. The base of the right lung shows signs of congestion, and there are evidences of pleurisy at one or both bases.[1]

Etiology.—In men the most common causes are appendicitis and ruptured duodenal ulcer; in women gastric ulcer. Other causes are extension of hepatic abscess, empyema perforating the diaphragm, extension of kidney or pelvic abscess, and local tuberculous or (rarely) cancerous processes.

Diagnosis.—In a right-sided *empyema* of the chest the upper border of the dulness, when continuous with that of the liver, is concave, being higher towards the spine. In *hepatic abscess* the liver is tender and enlarged below the costal margin, but it is often impossible to distinguish subphrenic from hepatic abscess. A variety containing air so greatly resembles pneumothorax that it is called *pyo-pneumothorax subphrenicus*. Exploratory needling under chloroform should be employed to complete the diagnosis. It is usually due to perforated gastric ulcer or abscess of the lung.

The *Prognosis* is fair if surgical treatment is carried out thoroughly and in time.[2]

§ **246. Actinomycosis of the Liver** is a condition which may be mistaken for abscess of the liver. It is due to the absorption of the ray fungus from the intestines, and starts as one or more foci in the liver substance, which slowly enlarge into spherical masses, and which may undergo suppuration, though the frequency of this latter is debated.

The *Symptoms* consist of vague uneasiness referable to the liver, with gradually increasing enlargement—at first uniform, later on unequal, the organ becoming

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[1] Mr. H. L. Barnard has described six clinical varieties, according to the position of the abscess. They depend upon the existence of six areas on the under surface of the diaphragm, four intraperitoneal and two extraperitoneal. The four intraperitoneal forms are the right and left anterior and right and left posterior, and are divided by the cruciform arrangement of the ligaments of the liver (falciform, coronary, right and left lateral). The extraperitoneal forms are the right, between the layers of the coronary ligament, and the left, above the left kidney. The commonest are the right and left anterior intraperitoneal forms, due chiefly to appendicitis, gastric ulcer, and duodenal ulcer. The next most common is the right extraperitoneal due to hepatic abscess. The *local* signs vary with the position and with the presence or absence of gas in the abscess. *In the right anterior intraperitoneal form*, if due to appendicitis, there are a dome-shaped extension upwards of the liver dulness, and evidence of compression and pneumonia at the base of the right lung, whilst the liver is not displaced downwards; if due to infection from in front, as from a gastric ulcer, the abscess presents at the right costal margin as a dull, tender, fluctuant swelling, limited by the mid-line of the abdomen. The *left anterior intraperitoneal form* presents in the left subcostal zone in a similar manner. A *right extraperitoneal* abscess gives signs of compression at the base of the right lung, and sometimes a fluctuant tumour in the epigastric notch. The liver is pushed down, even as low as the umbilicus. In the *right posterior intraperitoneal form* there are diffuse tenderness and rigidity along the right costal margin, spreading back to the right loin. A *left posterior intraperitoneal abscess* occupies the lesser sac of peritoneum, and may present between the stomach and liver, stomach and spleen, stomach and transverse colon, and beneath the transverse colon. A *left extraperitoneal* abscess points in the left lumbar region. The two anterior intraperitoneal forms are those which usually contain gas, and thus a circular area of tympany is produced in the centre of the dull area to the right or left of the epigastric notch. Metallic tinkling, splashing, and coin sound may be heard over the tympanitic area, which changes its position when the patient is rolled over (*Brit. Med. Journ.*, 1908, vol. i., pp. 205, 371, 429).

[2] Cantlie, *Brit. Med. Journ.*, 1899, vol. ii., p. 647.

prominent in one place. Exploration with trocar may yield no results; but if the tumour is laid open, the characteristic greenish fluid with yellow specks is obtained in which the ray fungus is found, which clinches the diagnosis.

VI. *The patient is a pregnant women, and the illness has been ushered in by deep* JAUNDICE *and* PROFOUND CONSTITUTIONAL SYMPTOMS. The disorder is ACUTE YELLOW ATROPHY OF THE LIVER.

§ 247. **Acute Yellow Atrophy** (Malignant Jaundice, Icterus Gravis[1] is a disease characterised by intense jaundice and cerebral symptoms, extensive necrosis of the liver cells, with rapid diminution in volume of the organ, often occurring in pregnant women, and usually ending fatally.

Symptoms.—(i.) The premonitory symptoms may be slight, resembling a catarrhal jaundice. There is increasing tenderness over the liver. (ii.) In a few days or weeks severe symptoms set in, with deepened jaundice, headache, and delirium, and the patient passes into the typhoid state. (iii.) Hæmorrhages occur from the stomach, bowel, and bladder, and there may be petechiæ under the skin. (iv.) Fever is usually absent during the course of the illness, but at the end it may be high. (v.) With the onset of the severe symptoms the liver dulness begins to rapidly diminish. The spleen is usually enlarged. (vi.) The urine is characteristically altered, having a marked diminution in uric acid, urea, and salts, while leucin and tyrosin are found crystallising out on evaporating a few drops of urine on a slide (Fig. 73).

Diagnosis.—Acute Yellow Atrophy is not likely to be mistaken for any other liver disease after the acute symptoms set in. Phosphorus poisoning may closely resemble it, but in that condition the liver is enlarged, and signs of irritant poisoning precede the onset of the jaundice.

Etiology.—Predisposing Causes.—(i.) Acute Yellow Atrophy is most common under middle age, though rare in children; and (ii.) in women, especially during pregnancy. (iii.) Dissipation and excesses of any kind are said to predispose. *Exciting Causes.*—It is said that the onset of this disease has often been preceded by severe mental emotion; and malarial, influenza, and other blood poisons have been said to determine its onset. The malady is probably microbic in origin.[2]

Prognosis.—The disease is very fatal. After the severe symptoms set in the patient usually dies in a comatose condition within a week. Pregnant women usually abort.

The *Treatment* is very unsatisfactory. During the preliminary stage the disease is treated as in catarrhal jaundice. Warm baths, diaphoretics, and diuretics may be tried.

CHRONIC DISEASES OF THE LIVER.

§ 248. **Routine Procedure.**—It will be remembered (§ 237) in the physical examination of a patient suspected to be suffering from hepatic disease that the *first* and most important question to investigate is whether there is *any enlargement of the organ* (by palpation and percussion). (2) The question next in order of importance is whether there is any *pain or tenderness* in the organ, for reasons which will be apparent below. And then (3) is there any *jaundice?* (4) Is there any *ascites?* (5) In every case of suspected liver disease the spleen (§ 258) and the urine should be carefully examined.

[1] "Icterus gravis" is a term used sometimes in a generic sense for any very severe jaundice tending to a fatal issue. Acute yellow atrophy is, according to modern researches, only one form of it (see Discussion at Clin. Soc., London, the *Lancet*, December, 1900).

[2] See Report of the Path. Soc. London, the *Lancet*, November 4, 1900.

The numerous *fallacies* in the alteration of the size of the liver dulness must be carefully studied (p. 355).

Classification.—By common consent chronic diseases of the liver are divided into those in which the AREA OF DULNESS IS NOT INCREASED, and those in which the AREA OF DULNESS IS INCREASED; and these latter are grouped into painful and painless enlargements.

A. The organ is of **normal** or **diminished size in**—

I. Functional derangement of the liver .. .. § 249
II. Atrophic (alcoholic) cirrhosis .. .. .. § 250

B. The organ is **increased in size,**—

a. Without pain or tenderness—

I. Hypertrophic cirrhosis (alcoholic and other) .. .. § 251
II. Fatty liver .. .. § 252
III. Lardaceous liver .. .. § 253
IV. Hydatid and other (in England) rare conditions .. § 254

b. With pain or tenderness—

I. Chronic congestion .. .. § 255
II. Cancer of the liver .. .. § 256
III. Abscess of the liver .. .. § 244

TABLE XIX.—CHRONIC DISEASES OF THE LIVER.

	Size *and* Surface.	Pain.	Jaundice.	Ascites.
I. ADVANCED CIRRHOSIS OF THE LIVER (*atrophic alcoholic cirrhosis*).	After enlargement it becomes DIMINISHED. Surface irregular (hobnail).	None.	Generally absent, never marked.	A very prominent symptom.
Ia. HYPERTROPHIC CIRRHOSIS. Of alcoholic, syphilitic, or biliary origin.	Enlargement may be very great. Surface hard and may be nodular.	Varies.	Varies.	Usually absent.
II. CHRONIC CONGESTION.	Slight enlargement. Surface smooth.	Present, but slight.	Slight.	Usually some.
III. FATTY LIVER.	Moderate enlargement. Surface smooth.	Absent.	Absent.	Absent.
IV. LARDACEOUS or Amyloid LIVER.	Enlargement may be very great. Surface smooth.	Absent.	Absent.	Absent.
V. CANCER OF LIVER.	Great enlargement. Surface uneven.	Severe.	Usually present.	Usually present.[1]
VI. HYDATID LIVER (rare in this country).	Outline of dulness arched or distorted.	Absent unless near surface.	Usually absent.	Absent.

A. In the first group, in which the liver is of **normal** or **diminished** size, there are only two disorders, I. FUNCTIONAL DERANGEMENTS; and II. ALCOHOLIC CIRRHOSIS; and these are two of the commonest hepatic disorders met with.

[1] The presence of jaundice and ascites depends on enlargement of the glands in the fissure, generally considerable in the later stages.

I. *There is* **no alteration in the size of the liver,** *but the patient complains of* LETHARGY, *vague digestive disturbances, sleepiness after meals, furred indented tongue,* CONSTIPATION, *headaches, and there is a frequent deposit of* LITHATES IN THE URINE *on cooling.* There is probably FUNCTIONAL DERANGEMENT OF THE LIVER.

§ **249. Functional Derangement of the Liver**[1] certainly constitutes one of the commonest of the minor ailments that affect a highly civilised community. Very careful percussion may perhaps detect slight enlargement, but generally, if there is any enlargement present, it indicates congestion, Active (§ 238), or Passive (§ 255).

Symptoms.—There are two manifestations of functional derangement which deserve special notice—constipation and lithuria. 1. The common complaint, "My liver is sluggish," is often equivalent to saying that the bowels do not act properly. Certainly, *constipation,* attended by pale-coloured fæces, due to a deficiency in the amount of contained bile, is a frequent accompaniment of disordered liver. The amount of bile in the stools is not, however, an absolute guide to the activity of the liver. *Diarrhœa* alternating with constipation, and flatus passed per rectum, may be present.

2. *Lithuria—i.e.,* excess of urates in the urine, which appear when the urine cools as a pink or orange deposit—is evidence, according to Murchison[2] and many of his followers, of defect in the liver function, more especially of that function which is concerned in the disintegration of nitrogenous foodstuffs. Such deposits are met with when the quantity of urinary water is markedly deficient, or when the proportion of proteid in the diet is in excess; but excluding these causes, there are three pathological conditions with which lithuria is specially associated—(*a*) *Febrile diseases—e.g.,* ordinary febrile "catarrh"—where the liver cells may become granular, and the whole organ may be enlarged and congested; (*b*) *structural diseases of the liver,* especially such as are attended by congestion; and (*c*) *functional derangement* of the liver (Murchison). Under these circumstances, which more particularly concern us now, the deposit of lithates in the urine is a "manifestation of a morbid condition of the blood and of the entire system," for which Murchison suggested the term lithæmia. It is due to the presence in the blood, not necessarily of lithic acid, but of numerous partially elaborated products belonging to the chemical series which connects proteid food on the one hand with uric acid, and urea on the other. It is accompanied, according to the same authority, by a great variety of symptoms—depression of spirits, irritability, lethargy, a disinclination for work, aching pains in the limbs, headache, vertigo, sleeplessness sometimes, undue drowsiness at others, dyspepsia,

[1] The introductory remarks at the head of this chapter may well be perused in this connection.

[2] *Loc. cit.*

palpitation, irregularity of the pulse, and high tension, or sometimes enfeeblement of the circulation and general enfeeblement of the body.

3. " Functional derangement of the liver may exist for years without any other symptom than the frequent deposit of lithates, and occasionally lithic acid, in the urine. But if neglected it may ultimately be the means of developing *gout*, structural diseases of the liver and kidneys, or some other serious malady " (Murchison, *loc. cit.*, p. 615).

4. *Sugar in the Urine—i.e.*, temporary or permanent glycosuria (diabetes), may in many cases, if not in all, be regarded as a manifestation of functional derangement of the liver ; but we are still in the dark concerning the pathology of this symptom.

It may, however, be assumed that glycosuria might arise in one or more of three ways : (*a*) Imperfect glycogenesis in the liver, the sugar passing through the liver unchanged ; (*b*) increased conversion of glycogen into sugar, which results whenever the circulation through the liver is increased—*e.g.*, by vaso-motor paralysis of the hepatic artery ; and (*c*) diminished destruction of sugar in the blood or tissues.

Etiology of Lithæmia.—Functional disorder of the liver (and consequently lithæmia and the other symptoms above named) may be *secondary* to (*a*) the continual over-functioning of the organ, (*b*) to diseases of the alimentary tract, (*c*) to diseases of the heart or lungs, and (*d*), as above mentioned, to pyrexia. When *primary*, its principal causes are—(1) *Errors of diet*, especially rich, sweet, greasy foods, and alcoholic beverages. Alcohol *combined with sugar* (*e.g.*, port and other fruity wines) are specially injurious ; or taken in the form of undiluted spirit, particularly on an empty stomach, is infinitely more harmful than dilute alcohol at meal times. (2) *Deficient supply of oxygen*, such as deficient exercise, or confinement in ill-ventilated rooms. (3) *Tropical climates*, especially when combined with indulgence in unsuitable food or alcoholic excess. (4) " Prolonged *mental anxiety*, worry, and incessant mental exertion " (Murchison). (5) Certain *constitutional* peculiarities, for the most part *inherited*, may render one person much more susceptible than another to any of the above causes.

Treatment of Lithæmia.—(1) *Diet* is certainly the most important feature of the treatment. Avoid particularly sugars, fats, and alcohol. All highly seasoned and rich foods, sweets, pastry, butter, and, in severe cases, potatoes and fruits may have to be given up. " In most cases of lithæmia, a diet consisting chiefly of stale bread, plainly-cooked mutton, white fish, poultry, game, eggs, a moderate amount of vegetables, and weak tea, cocoa, or coffee answers best ; while in others the patient enjoys best health on a diet composed of milk, farinacea, vegetables, eggs, and occasionally fish."[1] Haig's diet for uric-acidæmia, as he terms lithæmia, is still more rigorous,[2] and undoubtedly the quan-

[1] Murchison, *loc. cit.*, p. 615.

[2] Dr. Alexander Haig's diet for uric-acidæmia consists of : Bread, 10 ounces ; oatmeat, 2 ounces ; milk 2 pints ; cheese, 2 ounces ; rice, 2 ounces, vegetables and fruit, 12 ounces. Vegetables, fruit, and bread may exceed these quantities, but

tity as well as the quality of the food must be regulated. There is no doubt that many sufferers from lithæmia take more food than can be dealt with by the liver. It is here that inherited peculiarities play such an important part, for what is too little food for one man may be too much for another. Various dietaries are given in § 212. (2) Abundant exericse in the open air to supply the necessary oxygen is only second in importance to diet. (3) Hydragogue and cholagogue aperients—*e.g.*, the regular administration of salines (Hunyadi, Carlsbad, or Friedrichshall waters) every morning, and calomel once or twice a week (F. 46, 51, and 67).[1] (4) Personally I have found bark and mineral acids (especially nitro-hydrochloric) and bark, taken shortly before meals, very efficacious in some cases. (5) Among the other drugs, chlorides, iodides, and bromides are recommended by Murchison for the various conditions, as indicated by the symptoms. Opium is contraindicated. If this treatment fails, turn to that of Acute Congestion (§ 238).

II. *The area of liver dulness is* **diminished,** *and if the surface can be felt it is* HARD AND UNEVEN (*hobnail*) ; ASCITES *is probably present, but no very distinct jaundice ; the spleen is enlarged, and the patient is subject to* HÆMORRHOIDS, *and* HÆMORRHAGES *from the stomach and bowel.* The disease is ATROPHIC ALCOHOLIC CIRRHOSIS.

§ **250. Atrophic Cirrhosis of the Liver,** or, as it is sometimes called, Alcoholic Cirrhosis, Interstitial Fibrosis of the Liver, or Interstitial Hepatitis, consists of a progressive degeneration of the liver cells, with an increase of the interstitial fibrous tissue, leading to portal obstruction, and a shrinkage of the organ. Pathologists are now agreed that the interstitial fibrosis is secondary to the atrophic degeneration of the hepatic cells. Fibrosis or cirrhosis of the liver must still be regarded as mainly the result of alcoholic excesses, especially the habit of dram-drinking on an empty stomach. Clinically there are two varieties of Alcoholic Cirrhosis—the *Atrophic* form, which is a very common condition ; and the *Hypertrophic* form, which is relatively rare. The adjectives have reference *to the size of the organ*, for whereas the former

this observer maintains that the nearer a patient adheres to this dietary, the less likely is he to suffer from uric-acidæmia, gout, rheumatism, and allied diseases. These diseases he regards as being largely dependent on faulty diet, and especially the consumption of animal food (proteids) in excess, and such as contain uric acid and its antecedents (*e.g.*, xanthin). The foods quite free from these substances are bread, macaroni, rice, and other cereals, potatoes, vegetables, nuts, and fruit.

[1] Luff found that the gelatinous Sod. biurate is precipitated in crystalline form on making alkaline a blood serum with sod. bicarb. But if Pot. bicarb. is added, this action is *delayed*, and the precipitate *less in quantity*. Thus is explained the advantage of Pot. salts in acute and subacute gout, and the use of vegetables which are rich in Pot. salts. In gouty people the blood is not less, but really more alkaline than normal, from excess of soda salts, which hasten the crystalline deposit. "Causation and Treatment of Gout," *Lancet*, November 18, 1899, p. 1361, and *Brit. Med. Journ.*, 1899, vol. ii., p. 1163. See also Discussion, Brit. Med. Asso., *Lancet*, vol. ii., 1899, p. 441, and "Uric Acid," by Haig.

soon becomes diminished, the latter is enlarged throughout the disease. The hypertrophic form is further distinguished by a tendency to jaundice without ascites; and histologically the fibrosis has a uni-lobular distribution, instead of being multi-lobular as in atrophic cirrhosis.

Symptoms.—(1) In the early stage of the disease the organ may be enlarged, though rarely much so; but in the second and third stages the liver dulness is diminished. The liver is small and hard, and the surface is often nodulated, hence it is known as the "hobnail," or "gin-drinkers'" liver. There is a feeling of uneasiness and weight in the hepatic region. (2) The onset of the disease is very slow and insidious, extending sometimes over years. Gastric symptoms, such as *morning sickness*, and the other symptoms of alcoholic dyspepsia, are alone complained of for a considerable time. These are followed by symptoms of chronic gastritis, debility, and emaciation. The patient's aspect is very characteristic, with dilated *venous stigmata* in the cheeks. (3) Jaundice appears in the later stages of the malady in about one out of three cases. (4) Symptoms of portal obstruction occur (§ 236), and hæmatemesis is sometimes the first obvious symptom; the spleen becomes enlarged, and ascites (which is present in 80 per cent. of the cases) may be very considerable in amount. (5) In the concluding stages of this disease, when the secreting tissue of the liver is destroyed, the patient falls into a comatose state, with muttering delirium, which resembles uræmia and the typhoid state, except that there is pyrexia in the latter. This precise clinical resemblance is quite in keeping with the fact that the liver takes part in the elaboration of urea; so that when its cells are destroyed the blood becomes charged with a number of nitrogenous products, which cannot be eliminated.

Etiology.—(1) Cirrhosis of the liver is most common between thirty-five and sixty; it is rare under twenty-five. Men are much more frequently affected than women. (2) Alcohol is undoubtedly the most usual cause of atrophic cirrhosis, especially when taken in small quantities, frequently, or when taken *neat on an empty stomach*, the patient perhaps never becoming intoxicated.

Diagnosis.—*Cancer* of the liver is only difficult to diagnose from cirrhosis in the early stages; but usually it runs a more rapid course, and is accompanied by more pain, and more intense jaundice. The spleen is not usually enlarged in cancer. In *passive congestion* of the liver with ascites there are evidences of a cause, such as heart or lung disease.

Prognosis.—The disease has a slower and more insidious onset than hypertrophic cirrhosis (below), and is in most cases a more serious condition. If the patient is seen before signs of portal obstruction supervene much can be done, but if not until afterwards, the prognosis is grave. The outlook is more favourable in patients who are young (under thirty), and where the general health is good. *Untoward*

Symptoms.—Although cases are reported where restoration to comparative health has occurred after the development of ascites, it remains true that, as a general rule, with the onset of rapid ascites the end is in view, the patient rarely living more than a few months. The question has been raised whether such cases resulting in recovery were not syphilitic (§ 251, Ic). When there is rapid reaccumulation of fluid after paracentesis, and little benefit is derived from treatment, the course tends towards an early death.

Treatment in the early stages is practically the same as that employed for chronic congestion of the liver, and chronic gastritis (§§ 255 and 209). The habits of the patient must be corrected, and the diet reduced to the simplest elements; milk should be the staple diet in advanced cases. Alcohol must be completely cut off, and regular exercise taken. A course of salines should be ordered to be taken in the early morning, and rhubarb or mercurial pills at night. Ammonium chloride and iodide of potassium are valuable remedies in the stage of enlargement of the liver. If portal obstruction and ascites have set in, see § 185. Patients sometimes recover after repeated tappings, which gives time for the establishment of the collateral circulation; and recently surgical measures have been adopted for the artificial production of peritoneal adhesions for the establishment of the collateral circulation.[1] The one usually adopted is "epiplopexy" or stitching the omentum to the anterior abdominal wall.

We now turn to those chronic liver diseases in which **the area of dulness is increased.** These may be divided into two groups—those WITHOUT PAIN AND TENDERNESS, are described immediately below. If the enlargement is attended WITH PAIN AND TENDERNESS, turn to § 255.

There are four diseases with **enlargement** of the liver **without pain and tenderness**: I. HYPERTROPHIC CIRRHOSIS; II. FATTY LIVER; III. LARDACEOUS LIVER; and IV. HYDATID and other cysts. In CATARRHAL JAUNDICE (§ 239), CHRONIC CHOLELITHIASIS, and some other disorders, the liver is somewhat enlarged, but this is not their main feature.

I. *The* **liver is enlarged** *and* PAINLESS; *its surface is hard,* JAUNDICE IS PRESENT, *but little or no ascites, and there is a long history of failing health.* The disease is probably HYPERTROPHIC ALCOHOLIC CIRRHOSIS.

§ **251. Hypertrophic Cirrhosis of the Liver** is a term employed in a generic or clinical sense to indicate a progressive enlargement of the liver, due to an increase in the connective tissue of the organ with a tendency to jaundice. The condition may occur under at least five different aspects, due respectively to Alcoholism, Syphilis, Gall-stones, Chronic Heart-disease, and Malaria. Biliary cirrhosis may also be

[1] Rolleston and Turner, the *Lancet*, December 16, 1899.

associated with Splenic Anæmia. A rare variety of hypertrophic cirrhosis accompanied by pigmentation of the skin or bronzing, has been described. Sometimes this is attended by glycosuria, and has been called "BRONZED DIABETES." The pigmentation resembles that of Addison's disease, but the liver is larger than in that disease. The pigment contains iron.[1]

I*a*. HYPERTROPHIC ALCOHOLIC CIRRHOSIS (synonym: Biliary Cirrhosis).—In this form of cirrhosis the fibrous overgrowth occurs around single lobules, hence the name "uni-lobular cirrhosis." The disease is now classed by some authors as a distinct form of alcoholic liver, in which the organ is enlarged *throughout the whole course* of the disease; and there is a great tendency to jaundice, and but little ascites—features which contrast with those of the more common condition, Atrophic Alcoholic Cirrhosis, just described. Others hold that it is an early stage of the atrophic form.

Symptoms.—(1) The symptoms come on very insidiously, with a failure of the general health. The patient rarely applies for medical aid until (2) jaundice has set in, which may be very pronounced. (3) Fever in some cases occurs at intervals, and may be as high as 103° F. (4) In spite of the intense jaundice there are few or no signs of portal obstruction, and ascites is rarely, if ever, present. (5) The liver is uniformly, and may be considerably enlarged, hard, and sometimes rough. There is no tenderness and no actual pain (except during the feverish attacks), though a dull weight may be complained of in the hepatic region. The spleen is normal or only slightly enlarged. (6) A history of alcoholism is usually present, but in many cases the cause is obscure.

Diagnosis.—In *atrophic alcoholic cirrhosis*, after some preliminary enlargement, the liver undergoes contraction (§ 250). The percussion area of the liver, except in the early stages, is therefore normal or diminished. There is less tendency to jaundice, but ascites occurs in most of the cases. *Fatty* and *amyloid* livers are not acompanied by jaundice. *Cancer* has a more rapid and painful course. And see Table XIX., p. 370.

Prognosis.—Sometimes patients die within twelve months, with an acute onset of the typhoid state, but most live for a number of years, with signs of progressive emaciation. The prognosis is more favourable than in the atrophic form of cirrhosis; recovery is fairly common.

The *Treatment* of Congestion (§ 238), or Atrophic Cirrhosis, is applicable, according to the predominating symptoms.

I*b*. CARDIAC VALVULAR DISEASE results, as we have seen, in very considerable congestion of the liver. Long-continued passive engorgement of the liver gives rise to changes known as the "nutmeg liver,"

[1] Osler, *Lancet*, August 12, 1899. p. 440; *Lancet*, August 26, 1899; and *Brit. Med. Journ.*, 1899, vol. ii., p. 1595.

accompanied by more or less enlargement of the organ ; and this may be attended by a considerable degree of fibrosis. The diagnosis depends on the presence of cardiac valvular disease and other features (see Passive Congestion, § 255).

Ic. Syphilitic Disease of the liver generally takes the form of a diffuse hypertrophic fibrosis ; or it may be met with in the form of *gummata*. Undoubtedly, hepatic fibrosis may result from both hereditary and acquired[1] syphilis, though probably the gummatous form is commoner in the latter. In the inherited variety two forms of fibrosis occur. In one there is fine diffuse fibrosis between the individual cells (unicellular fibrosis) producing a uniformly smooth, firm liver ; in the other, coarse fibrosis, with perihepatitis and much distension, occurs, as in the acquired disease.

The *Symptoms* are somewhat variable. The liver is moderately enlarged ; there is not much tendency to jaundice and portal obstruction excepting in the final stages. There may be actual pain, especially when the capsule of the liver is involved ; but as a rule there are only indefinite sensations of illness, accompanied in the gummatous cases by a slight degree of intermittent pyrexia. In the gummatous form nodular projections may possibly be made out on the surface of the organ. The presence of such projections, accompanied by intermitting fever and a history of syphilis, in a young adult practically make the diagnosis certain.[2] In the absence of a syphilitic history the occurrence of pain and local tenderness at intervals points to syphilitic rather than to alcoholic cirrhosis, because *perihepatitis and the involvement of the capsule* are prominent features of syphilitic cirrhosis.[3] In the diagnosis from cancer we have mainly to rely on the efficacy of iodide, and the (usual) absence of jaundice and ascites in syphilitic disease.

The *Prognosis*, as a rule, is good, if the nature of the disease be discovered and it is treated by large enough doses of potassium iodide and mercury.

Id. Cirrhosis of Biliary Obstruction.—Hypertrophic cirrhosis has been produced experimentally in one half of the liver by ligature of one hepatic duct, and some observers have met with it clinically in association with gall-stones. When acting as clinical clerk to the late Dr. Charles Murchison, I had the opportunity of observing a case of this kind which was under his care. The patient was a woman, aged forty-five, with a history of repeated attacks of biliary colic. There was great enlargement of the liver, with jaundice of three years' duration. Cancer was excluded from the diagnosis, as the patient lived for some years afterwards. Hypertrophic cirrhosis due to biliary obstruction was Dr. Murchison's diagnosis, possibly due to stenosis of the hepatic duct.

Ie. Malarial Cirrhosis.—Subjects of prolonged malarial poisoning have an enlarged liver, which is believed to be due to cirrhosis. Alcohol may possibly be a contributory cause in these cases.

II. *The* **enlargement of the liver** *is* PAINLESS *and uniform ; the surface is smooth and soft ; there is* NO JAUNDICE OR ASCITES, *and the* SPLEEN IS NOT ENLARGED ; *there is a history of alcoholism, or the patient is suffering from phthisis.* The disease is probably Fatty Liver.

§ 252. **Fatty Liver** is a condition in which fat is deposited in the hepatic cells, commencing in the periphery of the lobules. It is nearly always associated with some other disease.

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[1] I have met with several cases of marked diffuse fibrosis of the liver, due to acquired syphilis. Glisson's capsule was extremely thick, and large ramifying bands passed from it into the organ in all directions.

[2] A case of this kind is recorded by Bristowe in the Clin. Soc. Trans., vol. xix., p. 249.

[3] Cheadle, Lumleian Lectures, *Brit. Med. Journ.*, 1900, vol. i., p. 756.

Symptoms.—(1) The liver is enlarged uniformly and is quite smooth. (2) Pain, jaundice, and portal obstruction are absent. (3) The accompanying symptoms are due to the cause of the fatty liver, and may consist, therefore, of debility, anæmia, etc. (4) The history of a *Cause* is important—viz. (i.) Chronic wasting disease, such as phthisis. (ii.) Fatty liver appears in association with fatty heart (*q.v.*) and general obesity. (iii.) It often occurs consequent on chronic alcoholism; and a mixed degeneration of fat and fibrosis is not uncommon.

The *Diagnosis* from the painful enlargements of the liver is not difficult (see Table XIX.). From *lardaceous* liver it is known by the absence of signs of lardaceous spleen or kidney, and by the absence of its cause.

The *Prognosis* and *Treatment* depend upon the primary disease—*i.e.*, the cause. It is hardly likely that the fat can be removed.

III. *The* **enlargement of the liver** *is* UNIFORM *and* PAINLESS; *the surface is smooth and hard; there is* NO JAUNDICE, NO ASCITES; *the* SPLEEN IS ENLARGED; *there is a history of prolonged purulent discharge or constitutional syphilis.* The disease is LARDACEOUS DEGENERATION.

§ 253. **Lardaceous (Amyloid or Waxy) Liver** is a condition in which the liver tissue is replaced by lardaceous material, which starts in the capillaries and smaller arteries of the organ, leading sometimes to an immense enlargement.

Symptoms.—(1) The liver is enlarged uniformly and smoothly, and feels firm and resisting; (2) pain, jaundice, and portal obstruction are absent; (3) the constitutional symptoms are due to the presence of the causal condition, and to the presence of amyloid disease of other organs.

Etiology.—(i.) Long suppuration and purulent discharge, as from necrosed bone; (ii.) constitutional syphilis; and (iii.) tuberculous disease of the lungs or elsewhere may produce amyloid disease here as elsewhere. Amyloid liver has become much rarer since chronic suppurations have been obviated by improved surgical methods.

Diagnosis.—The presence or history of a cause renders the diagnosis of amyloid disease comparatively easy (see also Table XIX.).

The *Prognosis* depends upon the amount of amyloid disease elsewhere. Diarrhœa, indicating amyloid changes in the intestines, abundant pale urine, with albuminuria, indicating amyloid disease of the kidneys, are untoward signs. If the cause is remediable, as by surgical treatment, the liver may decrease in size.

Treatment.—The indications are (i.) to remove the cause, and (ii.) to keep up the strength. The former is attained by administering potassium iodide in the case of syphilis, and by surgical treatment in the case of long-standing discharges. Tonics, such as iron and quinine with cod-liver oil are useful.

IV. *The* **enlargement of the liver** *is* PAINLESS, *but* NOT UNIFORM, *and the upper margin of the liver dulness is perhaps* ARCHED; *there is no jaundice or ascites and the spleen is not enlarged; a thrill or vibration is felt on percussion.* The disease is HYDATID CYST.

§ 254. **Hydatid Tumour of the Liver** depends on the presence in the liver of a parasite, rare in this country, though common in Australia, India, and Iceland, where dogs live in close association with man, and in Russia, where wolves are common.

Symptoms.—(i.) There is a slowly increasing enlargement of the liver, which is smooth, globular, and elastic, sometimes fluctuating. The right chest may be bulged outwards, with dulness in the axilla. When the fingers of the left hand

are laid on the tumour and tapped with those of the right hand, the "hydatid fremitus," or "vibration," is felt in some cases. (ii.) Pain is absent unless the tumour is very near the surface, when great pain may be present, because *the capsule* is involved. (iii.) No constitutional symptoms appear unless the tumour becomes so large as to press upon the surrounding structures, or becomes inflamed and suppurates.

Etiology.—The parasite enters the alimentary canal of man by means of drinking water contaminated by the fæces of the dog or wolf, containing the ova of the tænia echinococcus, a tapeworm which may infect the dog. The embryo is carried to the liver, where it encysts and grows. The cyst so developed has a firm gelatinous wall, and contains a clear fluid ; and from the wall a number of proscolices or embryonic heads develop, each with a crown of most characteristic hooklets.

Diagnosis.—*Abscess* of the liver produces pain and fever, and on aspiration yields grumous material like anchovy sauce. *Pleuritic effusion* on the right side, leading to dulness in the axilla, may resemble hydatid. In such cases a *bulging* outwards of the lower ribs over the liver points to the presence of hydatid. A *renal cyst* has resonance in front, due to the colon. A history of residence in Australia, etc., should lead one to suspect hydatid in cases of slowly increasing enlargement of the liver, *with few other symptoms.* Exploratory puncture settles the diagnosis. This must be performed by a long trocar and cannula pushed in many directions—some seven at least, if not at first successful. The fluid withdrawn is pathognomonic. It is clear, opalescent, of low specific gravity, and contains a large excess of chlorides, no albumen (unless inflammation has taken place), and—most characteristic of all—echinococcus hooklets (see Fig. 75).

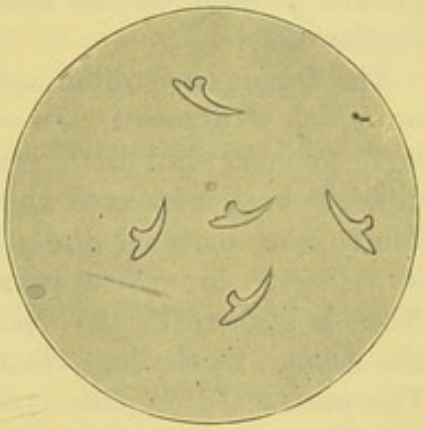

Fig. 75.—Hooklets from an HYDATID CYST in man ; the TÆNIA ECHINOCOCCUS. — Magnified about 150 times. These form the crown of hooklets around the anterior end of the scolex, and are absolutely distinctive of hydatid fluid. From a photomicrograph by Mr. Frederick Clark.

Prognosis.—The patient may live for several years with no other symptoms than a slow increase in the size of the liver. Occasionally the tumour undergoes spontaneous cure by drying up. In some cases hydatids appear in other parts of the body, as in the lung, peritoneal cavity, brain, or other organs. Jaundice may very occasionally be caused by cysts lodging in the bile ducts. The cyst may suppurate, giving rise to the symptoms of liver abscess, or pyæmia may be set up. Sometimes death occurs by the sudden rupture of the cyst into the pleura or peritoneum.

Treatment.—Success has been attained in some cases by drawing off a few ounces of the fluid, the cyst then having shrivelled up. Free incision and drainage is the best method of treatment.

There are three diseases in which **enlargement of the liver** is attended **with pain and tenderness** : I. CHRONIC CONGESTION, II. CANCER OF THE LIVER, and III. ABSCESS OF THE LIVER. In CHRONIC CHOLELITHIASIS and several ACUTE DISORDERS the liver may be slightly enlarged and tender.

I. *The* **enlargement** *is moderate, smooth, and uniform,* PAINFUL, *and* TENDER ; *some jaundice and ascites may be present, the* SPLEEN IS ENLARGED, *and there are signs of congestion of the abdominal viscera.* The disease is probably CHRONIC CONGESTION OF THE LIVER.

§ **255. Chronic Congestion** of the liver is a condition in which the enlargement is due to venous obstruction (passive congestion). Compare the opening remarks in § 238.

Symptoms.—(i.) The liver is tender, and a sensation of weight and fulness is complained of in the hepatic region. Pulsation may be conveyed to the palpating hand synchronous with the heart in the early stages, but as the organ becomes firmer this is lost. (ii.) Signs of general venous obstruction appear. (iii.) Ascites develops, and the spleen is enlarged. The yellow discoloration of jaundice may arise. (iv.) Gastro-intestinal disturbances are common.

Etiology.—Passive congestion is the result of any backward pressure due to obstruction of the circulation. In most cases this is caused by heart or lung disease, and especially mitral valvular disease. Any growth pressing on the inferior vena cava above the diaphragm has similar effects.

The *Diagnosis* is often aided by the recognition of the heart disease on which it depends. In some cases of *ascites* with anasarca of the legs, we may find both *hepatic enlargement* and *albuminuria*, and a difficulty may arise as to which was the primary cause of the condition—heart, liver, or renal disease. This difficulty is still further increased if extensive bronchitis prevents accurate auscultation of the heart. Now, in such cases, *the liver* may be excluded as the primary cause, if the dropsy in the legs clearly preceded the dropsy of the abdomen. The presence of hepatic enlargement is then a sign of great value as helping to exclude *renal* mischief, because enlargement of the liver is not a usual sequence of kidney disease, although it is a fairly constant result of *cardiac* valvular disease.

Prognosis.—The prognosis is altogether influenced by the cause of the congestion; and the state of the heart is generally the measure upon which the patient's chance of a longer or shorter life depends. In mitral stenosis an enlarged liver with ascites is less grave than in mitral regurgitation, because it normally occurs at an earlier stage in stenosis (Broadbent). It is most serious in aortic disease, and especially regurgitation.

The *Treatment* is that of the cause, and our attention must be directed to the heart and lungs. Purgatives and light foods are necessary in order to relieve the strain on the portal system. Leeches over the liver or bleeding may be indicated (see also Acute Congestion, § 238).

II. *The* **enlargement of the liver** *is* IRREGULAR; *the* PAIN *and tenderness may be great;* JAUNDICE *and* ASCITES *are present; the spleen is not enlarged; the patient is advanced in years, feeble and emaciated.* The disease is CANCER OF THE LIVER.

§ **256. Cancer** of the liver may be primary, but is usually secondary to disease elsewhere; generally the stomach, rectum, or other part of the alimentary canal.

Symptoms.—(i.) Pain is an almost constant feature of cancer of the liver; it is continuous, with exacerbations, and is independent of food or position. A certain amount of tenderness develops. (ii.) The enlargement of the liver is irregular, and nodules may be made out. These are of a hard consistence, and increase rapidly. There is also a less common diffuse form of cancer in which there are no nodules, and in which the liver is only slightly and uniformly enlarged. In the nodular form the liver may be enormously enlarged. (iii.) Jaundice is usually present, *sooner or later*, and is intense and progressive. An intense jaundice persisting over five to seven weeks in an old person should indeed always lead one to suspect cancer. Ascites generally occurs, either from involvement of the glands in the fissure, or the peritoneum.[1] The spleen is not enlarged. (iv.) The general health of the patient is bad, and emaciation and cachexia may be present before any local signs are discovered. Cancer may be present in another part of the body. Fever occurs at intervals, especially in cases of primary cancer.

Causes.—Cancer occurs after middle life; it is rare before thirty-five. It is liable to occur secondarily to cancer of the stomach or rectum. When a patient has been the subject of gall-stones for a long period of time, cancer of the liver is apt to result in later life.

Diagnosis.—Jaundice is very rarely entirely absent in cases of cancer. This and the cachexia alone may justify a diagnosis of the condition. The diagnosis from *cirrhosis* may be difficult when nodular enlargement cannot be definitely made out, and when considerable ascites is present. In cirrhosis there is little or no pain and tenderness, the history of the illness is of longer duration, the spleen is enlarged, and the jaundice is not so intense. The *inflammatory thickening* under the liver after a long history of gall-stones may resemble cancer, and can be distinguished only when time shows little or no increase in the enlargement. *Syphilitic* liver has not so much pain and tenderness, is of slower growth, and rarely produces ascites.

Prognosis.—Cancer of the liver is usually fatal within six to twelve months, death taking place from exhaustion. Untoward symptoms are rapid growth, ascites, or respiratory difficulties due to extension of the disease to the lungs.

Treatment can be palliative only. Morphia or opium is administered for the pain, and attention must be given to the relief of the symptoms of gastric distress, and to aid nutrition. With rest and care the patient may have periods during which the disease makes no progress, and which hold out to the patient false hopes of his ultimate recovery.

III. **Abscess of the Liver** also produces considerable hepatic enlargement, which is PAINFUL and TENDER. It has already been described among the Acute Diseases, § 244; but sometimes it runs a very chronic course.

[1] Dr. Charles Murchison used to teach that *jaundice* with *ascites* in an *old person* usually indicated cancer.

Tumours of the Liver, other than CANCER (§ 256), HYDATID (§ 254), and GUMMA § 251, Ic.), are more rare. Their presence is manifested by *enlargement of the organ,* which may be regular or irregular, accompanied in some cases by constitutional symptoms. When, as in some cases of ACTINOMYCOSIS (§ 246), they assume an inflammatory form, pyrexia, accompanied by sweatings and rigors, is present. SARCOMA OF THE LIVER is occasionally met with—*e.g.*, Lympho-sarcoma—but it is most often secondary to deposits elsewhere, and the liver condition is only a subordinate part of the case. The patient may be younger than in the other form of malignant disease. Chondro-sarcoma, Melano-sarcoma, Tubercle, and Fibroma occur very rarely.

Floating Liver (Dropping or Ptosis of Liver, Hepatoptosis) is probably a somewhat rare condition which is due to a laxity of the ligaments. It has lately been receiving some attention (*vide* the *Lancet*, May 12, 1900). It is apt to be mistaken for enlargement of the organ, and *vice versa*. The condition has been referred to under Abdominal Pain, because, if attended by symptoms, this is the principal one. There may also be vague neurasthenic symptoms.

THE SPLEEN.

There is still some doubt as to the precise part which the spleen plays in the economy, and symptoms may be altogether wanting when it is diseased. Great diminution in size of the organ has been found *post-mortem* without any symptoms during life ; and the spleen has been removed by operation without a fatal issue.[1] Sometimes, however, profound disturbance attends enlargement of the organ.

PART A. SYMPTOMATOLOGY.

§ **257.** In addition to the local pain and discomfort due to the enlargement of the organ, the symptoms which arise may include extreme pallor of the skin (the most intense anæmic pallor known is that associated with splenic leukæmia), great weakness, and enormous increase in the number of leucocytes in the blood ; but we are not sure that all of these are results of splenic disease. In other instances, in " ague cake " for example, very great enlargement takes place without any symptom beyond the inconvenience due to the size of the organ. The *symptom* which is found to be most constantly associated with disease of the spleen is anæmia, the various " causes " of which will be discussed elsewhere (§ 401).

PART B. PHYSICAL EXAMINATION.

§ **258.** The only physical signs which can be relied upon as diagnostic of splenic disease are the signs of enlargement of the organ, and this is most readily made out by **Palpation.** When the spleen is enlarged,

[1] Spencer Wells has reported several cases of removal of the spleen, and in each instance the patient seemed in good health for, at any rate, some years after the operation. More recently Mr. Bland-Sutton has reported nine cases of extirpation of the spleen for various maladies (Report Clin. Soc., London, the *Lancet*, 1900, vol. ii.).

the anterior edge of the organ, being free, makes its way downwards and forwards towards the umbilicus. The *notch* in the anterior border is so characteristic that it forms a strong point in diagnosis of any splenic tumour. METHOD.—Stand on the right side of the patient, who should be lying on his back. Pass the left hand across the abdomen, and lay it posteriorly over the eleventh rib on the left side, and place the right hand flat upon the anterior surface of the abdomen, with the tips of the fingers just below the eleventh rib. By gently dipping them down into the abdomen, and tilting the organ upwards with the left hand during inspiration, the splenic notch may be felt if the organ is enlarged. It is more readily palpated when the patient draws a deep breath. Normally, the spleen cannot be detected by palpation, and even slight enlargements may not always be appreciated. An enlarged spleen always has a space between its posterior edge and the erector spinæ behind, into which the fingers can be dipped—at any rate, in spare subjects. *Fallacies.*—Without being enlarged, the spleen is readily palpable when it is displaced downwards, or is "floating." It is sometimes displaced downwards in cases of deformed chest (*e.g.*, rickets), large pleuritic effusions, and emphysema.

§ **259.** The **Percussion** of the spleen is attended with some difficulty. The organ is situated in the left hypochondrium, between the upper border of the ninth rib and the lower border of the eleventh; and roughly between the mid-axillary and scapular lines (Fig. 37, p. 135). It extends obliquely forwards and downwards nearly to the costal margin. It lies wholly beneath the ribs, and the upper third is overlapped by the lung. Percussion does not afford a very accurate means of investigation, but it is well to remember that *a straight line drawn from the centre of the left axilla, obliquely downwards and forwards to the umbilicus, should be resonant in its entire length* (Gairdner's line). The spleen normally lies altogether behind this line, but if it be enlarged this line is impinged upon by dulness at the junction of its middle and lower thirds.

The SURFACE LANDMARK of the spleen may be said to form an oval, lying *obliquely* between the post-axillary and mid-axillary lines, and having, for purposes of description, four borders. The procedure for percussing out the *anterior* and *lower* borders differs from that used to elicit the *upper* and *posterior*, because the latter recede from the surface, the lung intervening. It is best to percuss at the end of an expiration, because the spleen is then less covered by lung. As mentioned above, palpation is preferable, but to define the *anterior* and *lower* limits by percussion the patient should lie on his back. (1) *Anterior border*—percuss *lightly* along the tenth rib, starting at its anterior end, and the note will be found to become dull about the *mid-axillary* line. (2) For the *lower border* percuss, also lightly, along the posterior-axillary line from below upwards, and the lower border should be reached about the lower edge of the eleventh rib. (3) To define the *upper* and *posterior* limits is very much more difficult, and very often—in fat subjects for instance—it is impossible. Fortunately, it is not of so much importance to map out the posterior border. The patient must either sit up or lie in a semi-prone position, resting on his right scapula. If he turns completely on to his right side,

the spleen may fall away from the left side. His left hand should be placed on his head. *Upper border*—percuss with a *heavy* stroke just behind the post-axillary line, starting from the angle of the scapula and working vertically downwards. After repeating this several times, it will be noticed that the pulmonary resonance is impaired at the upper border of the ninth rib. (4) *Posterior border*—similarly with heavy percussion, by starting over the neck of the tenth rib and continuing along that rib anteriorly, you may elicit a change of note just in front of the scapular line.

Fallacies.—The dulness of *splenic enlargement* may be simulated by pleuritic effusion or consolidation of the left lung. The area of splenic dulness may be *diminished* by emphysema of the lungs, or distension of the stomach or the colon by gas. The splenic dulness may be altogether absent when there is a wandering spleen, or congenital absence of the organ.

PART C. DISEASES OF THE SPLEEN.

§ **260.** The diseases of the spleen are all—if we except the relatively rare cases of wandering spleen and atrophy—comprised under the causes of **enlargement of the organ,** and its **diagnosis** therefore becomes a matter of considerable importance. Enlargement is detected by palpation aided by percussion as above mentioned. The mechanical effects of pressure, when the spleen is very much enlarged, are mainly dyspnœa and gastro-intestinal disturbance.

Splenic Enlargements have three chief characteristics: (1) The splenic *notch* is felt on its anterior border; (2) the mass moves with respiration; (3) it is dull to percussion because the resonant colon does not lie in front of splenic tumours, as it does in front of renal tumours, Gairdner's line of percussion resonance (*vide supra*) being thus impinged upon. (4) It is also worth bearing in mind that when an area of dulness is due to splenic enlargement, its outline *resembles in shape* that of the normal spleen. A splenic enlargement or tumour may further be diagnosed from other abdominal tumours by remembering (5) its mobility on bi-manual palpation, unless bound down by adhesions; and (6) its surface, which in simple enlargement of the organ is distinguished from neoplasms of the peritoneum, stomach, intestines, etc., by being smooth and firm.

Splenic enlargements or tumours may have to be diagnosed from the following conditions: (1) *Renal tumours*, and especially movable kidney, in which (i.) there is resonant intestine in front of the tumour, and (ii.) there is not, as in splenic tumours, any resonant percussion in the flank; (2) *enlargement of the left lobe of the liver*, in which (i.) the dulness is continuous with that of the right lobe, whereas splenic dulness rarely reaches to the middle line, and (ii.) the splenic notch is absent; (3) *cancer of the cardiac end of the stomach*, in which (i.) the dulness is less absolute, (ii.) there is no notch, and (iii.) there is coffee ground vomiting, etc.; (4) *ovarian tumour*, which (i.) will have grown from below upwards, (ii.) the hand cannot be pushed between the tumour and the pelvic brim as it can in the case of a splenic tumour, and (iii.) can be felt on vaginal examination; (5) *accumulation of fæces*, in which

(i.) the tumour has an irregular outline, (ii.) doughy consistence, and (iii.) a course of purgatives and enemata will remove it; (6) *post-peritoneal tumour*, in which (i.) there is no notch, and (ii.) no resonance behind it; (7) *abdominal aneurysm*, which, when of sufficient size to be mistaken for the spleen, is attended by (i.) severe pain in the back, and (ii.) evident expansile pulsation; (8) *deep-seated abscess in the abdominal parietes*, in which the swelling is (i.) tender and has a vague irregular outline, and (ii.) situated more superficially than a splenic tumour.

The **Causes of Enlargement of the Spleen** may be grouped as follows:

a. **Acute Enlargement.**

I. Acute infections.
II. Embolism of the spleen.

b. **Chronic Enlargement.**

I. Cardiac disease.
II. Cirrhosis of the liver.
III. Lardaceous disease.
IV. Spleno-medullary leukæmia.
V. Hodgkin's disease.
VI. Splenic anæmia.
VII. Erythræmia.
VIII. "Ague cake."
IX. New formations—hydatid, cancer.
X. Chronic infections—tubercle and constitutional syphilis.
XI. Rickets.
XII. Splenic anæmia of Infants (pseudo-leukæmia of Von Jaksch).

If the illness of which the patient complains came on gradually and is running a **chronic** course, turn to § 262; but if it came on more or less suddenly, commence at **acute enlargements,** below.

§ **261. The Causes of Acute Enlargement** *of the spleen—i.e., coming on rapidly with acute symptoms, and often preceded and accompanied by pain and tenderness of the organ—come under two headings.*

I. Acute Infections.—The spleen is enlarged more or less in nearly all the acute specific fevers, especially typhoid, typhus, relapsing fever, pyæmia, malarial affections, and pneumonia. This form is recognised by (i.) the presence of symptoms of the primary malady; (ii.) the presence of pyrexia; (iii.) the subsidence of the enlargement with improvement in the primary disease.

II. Splenic Embolism may be either *benign* or *septic* in character. (*a*) If *benign* it follows as a complication of cardiac valvular disease, especially mitral stenosis; and is recognised by (i.) the enlargement of the spleen rarely being great, and soon passing away; (ii.) a history of sudden onset of *acute pain* in the left hypochondrium, which rapidly subsides; (iii.) the absence of hepatic or pulmonary disease, and the presence of symptoms of cardiac valvular disease. (*b*) If of *septic* origin, it follows as a complication of malignant endocarditis (§ 39*a*)

or some other septicæmic process. Here inflammation of the splenic substance may, though rarely, proceed to the formation of an abscess. Septic embolism is characterised by the same symptoms as simple embolism, and in addition we have evidences of the pyæmic process, and symptoms of septic emboli in other parts.

§ 262. *The* **Causes of Chronic Enlargement** *of the spleen are more numerous than those of acute. In adults the commonest perhaps is* CONGESTION, *due to chronic* LIVER *or* HEART *disease; but in children* SYPHILIS *and* RICKETS *are more frequent.*

I. In CARDIAC DISEASE the spleen shares in the general congestion of organs in the later stages, but it is a relatively unimportant feature, and the enlargement is rarely great in uncomplicated cases.

II. CIRRHOSIS OF THE LIVER and other causes of PORTAL CONGESTION are attended by splenic congestion, and may result also in a fibrosis of the organ; but here again the splenic condition is only one of the aids to the diagnosis of the primary condition.

III. LARDACEOUS or WAXY DISEASE is one of the commonest causes of splenic enlargement. The swelling may be considerable, but it is rarely as large as V. or VI. below. It is recognised by (i.) the surface being smooth and uniform; (ii.) painless enlargement (waxy disease) in other organs, such as the liver; (iii.) profuse diarrhœa, due to waxy disease of the intestines; (iv.) the history of a cause—viz., prolonged purulent discharge (*e.g.*, chronic phthisis), or of syphilis.

IV. In SPLENO-MEDULLARY LEUKÆMIA the spleen may be very large, extending even to the pelvis, and weighing over 10 lbs.; (i.) there is great pallor and debility; (ii.) leucocytosis is present, with changes in the corpuscles (see Leukæmia, Chapter XVI., § 407); (iii.) there is great tendency to hæmorrhage from the nose and other mucous membranes; (iv.) frequently diarrhœa is present, and, if the disease lasts for long, ascites may supervene.

V. In HODGKIN'S DISEASE the spleen is occasionally the seat of lymphadenomatous enlargement. It is recognised by (i.) the enlargement being slight; (ii.) the presence of enlarged lymphatic glands in other parts of the body; and (iii.) the paroxysms of pyrexia and glandular swelling.

VI. In the SPLENIC ANÆMIA of adults there are (i.) leucopenia and secondary anæmia; (ii.) debility; (iii.) tendency to hæmorrhages; (iv.) cirrhosis of liver associated with jaundice and pigmentation—Banti's disease (§ 409).

VII. In ERYTHRÆMIA there are (i.) cyanosis; (ii.) a large excess of red blood cells (polycythæmia) (*vide* Cyanosis, § 26).

VIII. "AGUE CAKE" is a term used to indicate the persistent enlargement and induration of the spleen due to malaria. (i.) The organ is smooth and hard, and it may be of very great size, although causing comparatively little inconvenience. (ii.) There is a history of ague, or of exposure to malaria, and great debility and anæmia are usually present. (iii.) There is an absence of heart or liver disease. (iv.) There is little or no increase in the number of white corpuscles, and no other signs of leukæmia are present.

IX. NEW FORMATIONS of the spleen are recognised chiefly by the irregularity of the enlargement to which they give rise.

1. Hydatid of the spleen is extremely rare, and if present usually exists concurrently with hydatid of the liver, and is recognised by (i.) the enlargement not being uniform ; (ii). the evidences of a fluid cyst, fluctuation, and the "hydatid thrill"; (iii.) an exploratory puncture draws off clear, opalescent, non-albuminous fluid of low specific gravity (§ 254).

2. Cancer of the spleen is extremely rare as a primary disease, and comparatively rare as a secondary deposit. It is known by (i.) the hardness and irregularity of the tumour ; (ii.) the severe pain ; (iii.) cachexia, and cancer elsewhere.

Enlargement of the spleen in children *is a fairly common occurrence, and it may be due to* Tubercle, Constitutional Syphilis, Rickets, *or* Splenic Anæmia.

X. Chronic Infections.—1. Tubercle sometimes affects the spleen, but the enlargement is not great, and it is not of much clinical importance, as there is generally more advanced tubercle in other organs. 2. Constitutional or Hereditary Syphilis may produce three histological changes in the spleen—*fibrous infiltration*, or simple hypertrophy as it is sometimes termed, *lardaceous disease* (*vide supra*) ; and *gumma* (which are comparatively rare). Syphilitic fibrous infiltration is differentiated by (i.) it only occurs in children who are the subjects of hereditary syphilis ; (ii.) it is usually accompanied by snuffles, rash, and other syphilitic manifestations ; (iii.) it subsides under anti-syphilitic treatment. Gumma is sometimes met with under the same conditions ; occasionally also in adults.

XI. Rickets causes an enlargement of the spleen, and this form is recognised by (i.) its occurrence in early childhood ; (ii.) the accompanying swelling of the epiphyses, deformity of the chest and head ; and (iii.) there is often enlargement of the liver as well.

XII. In Splenic Anæmia of Infants the spleen is greatly enlarged, but anæmia is the chief symptom. There are leucocytosis and diminution of the red corpuscles, many of which are nucleated ; and myelocytes are usually found (§ 414).

The *Treatment* and *Prognosis* of splenic enlargement depend, for the most part, on the primary condition. The treatment of lardaceous disease and of hydatid is given under Hepatic Disorders (§§ 253, 254). The treatment of "Ague Cake" consists of (i.) removal to a non-malarious district, and the administration of quinine and tonics, with free saline purgation ; (ii.) unguentum hydrargyri iodidi dilutum, rubbed over the splenic area, was a remedy advocated by Murchison. (iii.) Violent movement must be forbidden, as the spleen may rupture.

§ 263. **Wandering Spleen** (Floating, Dropped, or Dislocated Spleen, Splenoptosis) may be readily mistaken for enlargement of that organ when met with in the lesser degrees of displacement. But when the dislocation is, as generally happens, considerable, it is more often taken for a floating kidney. However, the presence of the notch, the fact that it can be made to recede upwards and that it comes down in front of the colon, aid in the diagnosis. The condition is mostly met with in multiparæ with pendulous abdomens. It may be accompanied by nervous symptoms, though less constantly than in dislocation of some of the other viscera. If troublesome, the condition may be relieved by removal of the organ, an operation which has been performed several times with good results (Bland-Sutton, see footnote, p. 382).

§ 264. Atrophy of the Spleen is, as a rule, unattended by symptoms. It is, as Bristowe said, a condition not infrequently met with. It may be congenital, but its commonest causes are: I. CIRRHOSIS of the spleen, due to an increase in the interstitial tissue, the result, as in cirrhosis of the liver, of alcohol; and II. CONTRACTION OF THE FIBROUS CAPSULE, usually of syphilitic origin. The syphilitic deposits in the capsule of the spleen sometimes take on a cartilaginous change, and form plates of cartilage. I have come upon them several times in the dead-house, but they had been unattended by symptoms during life. I have also met with some five cases of marked atrophic condition of the spleen, without symptoms during life, death having occurred from independent causes.

CHAPTER XIII

THE URINE

The intricate subject of renal diseases is rendered more comprehensible by a brief consideration of their history. In 1812 Wells[1] found that albumen in the urine was associated with certain forms of dropsy. It was not, however, until 1836 that Bright[2] went a step farther and discovered that dropsy and albuminuria, when associated together (in the absence of heart disease), were indicative of disease of the kidneys. The term "Bright's disease" has thus come to be synonymous with disease of the kidney. We now know that there are many different disorders of the kidney which present dropsy and albuminuria at some stage of their course. Thus, we have *acute* inflammation of the renal epithelium, and this is known as Acute Bright's Disease. Similarly, the term "Chronic Bright's disease" should, in the author's judgment, connote *chronic* renal disease, of which there are at least two distinct clinical varieties—chronic tubal nephritis and chronic interstitial nephritis. But the term "Chronic Bright's Disease" has, in later years, sometimes been used to indicate only the last-mentioned variety. Now chronic interstitial nephritis—*i.e.*, the granular or gouty kidney, is very generally associated with a widespread cardio-vascular change in which the high blood-pressure and its general symptoms are more pronounced than the urinary symptoms. By degrees the term Chronic Bright's Disease came to be applied to cases in which the chief and perhaps the only symptoms were cardio-vascular. Even since it was shown that high arterial tension and cardio-vascular changes may occur without renal disease,[3] the term Chronic Bright's Disease is still sometimes loosely and incorrectly applied to high arterial tension and its associated symptoms.

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[1] Wells, Transactions of the Society for the Improvement of Medical and Surgical Knowledge, iii., 194, London, 1812.

[2] Bright, Guy's Hosp. Rep., I., London, 1836.

[3] The author's investigations into the large series of renal cases on the one hand and cardio-vascular cases on the other which presented themselves at the Paddington Infirmary revealed this (Trans. Med. Soc., London, 1897-1898; the *Lancet*, 1897, vol. i., pp. 882 and 1235; *Brit. Med. Journ.*, January 23, 1897; and Trans. Path. Soc., London, 1904).

Renal diseases are for the most part chronic, and often obscure; but, with a knowledge of these historical data, their study would be comparatively simple were it not for another confusion which has arisen owing to the numerous terms applied by pathologists to diseases which differ anatomically, though not always clinically. In what follows we shall be concerned only with the clinical aspect of renal disease.

It is not always possible in practice to separate kidney diseases proper from disorders of other parts of the urinary tract, because changes in the urine are common to them all. It will be necessary, therefore, to refer to disorders of the bladder, prostate, and urethra for diagnostic purposes, though their treatment comes mainly within the province of the surgeon.

PART A. SYMPTOMATOLOGY.

The chief function of the kidneys is the elimination of nitrogenous waste. When this is interfered with by structural or functional disease a toxic condition results, which, when it reaches a certain stage, is known as uræmia.

As a consequence of the deep-seated position of these organs, the local symptoms referable to the kidney are, except in cases of Tumour or displacement, of subordinate importance. The most constant and CARDINAL SYMPTOM of kidney disorders is some **Alteration in the Urine,** which, as an indication of renal disease, corresponds to the physical signs in other organs, and is dealt with in PART B. of this chapter. The cardinal symptoms next in order of importance are **Pallor of the Surface** and **Dropsy. General symptoms,** due to the toxic state which results from the retention of the nitrogenous waste, also accompany these diseases.

§ **265. Pallor of the Surface and Malaise** are very constant features of all organic kidney diseases. To the experienced eye the pallor differs from that of anæmia in a manner somewhat difficult to describe. The skin has a " waxy " hue, a simile which is still further exemplified when dropsy is present. It affects the whole body, but is always most evident in the face. In chronic interstitial nephritis the pallor has a greyish hue. Other causes of pallor and their diagnosis will be given in Chapter XVI.

§ **266. Renal Dropsy** is of *general* distribution, in which respect it differs from cardiac dropsy, which starts in the *legs* or most dependent parts, and from hepatic dropsy, which starts in the *abdomen.* It is, however, most evident in the loose cellular tissue—*e.g.*, around the eyelids, where it is most marked on first rising in the morning. Towards evening the ankles become œdematous, or, as the patient may express it, a " ridge is present around the top of the boot." In severe cases (*e.g.*, in acute nephritis) the eyes may be almost closed by the swollen

lids, and at the same time there may be signs of dropsy in the serous cavities—the pleura, peritoneum, and pericardium. Œdema of the solid organs also occurs in severe cases, and death may be produced by pulmonary œdema. Œdema glottidis is another serious though less frequent complication.

Dropsy is by no means an equally constant feature in all diseases of the kidney. In *acute and chronic parenchymatous nephritis* (*i.e.*, diseases in which the renal epithelium is primarily affected) dropsy is almost invariably present. But in *chronic interstitial nephritis* and *lardaceous kidney* it is comparatively rare ; in the former it may occur late in the course of the disease, when it is generally due either to cardiac failure or to secondary inflammation of the renal epithelium. In uncomplicated *pyelitis* and *neoplasms* dropsy is not present.

§ **267.** A large number of **General Symptoms** occur as the result of the retention of the nitrogenous waste products : Cardio-vascular changes, hæmorrhages, breathlessness, affections of the nervous system, ocular changes, gastro-intestinal disorders, and secondary inflammations.

Cardio-vascular Changes frequently accompany renal disease. In acute and chronic renal disease there is usually high arterial tension, and often dilatation of the heart. The accentuated second aortic sound which accompanies this high tension is a useful indication in some cases for bleeding, or other measures for the reduction of arterial tension. In chronic interstitial nephritis the high tension is apt to be followed by a thickening of the arteries due to hypertrophy of the muscular coat.[1] The left ventricle becomes hypertrophied and, in the later stages, signs of cardiac failure may ensue.

Hæmorrhages sometimes occur in chronic Bright's disease, a consequence of the high tension, combined in most cases with a diseased state of the bloodvessels. Epistaxis, for instance, may be the first symptom which leads to the discovery of chronic Bright's disease. Bleeding from the stomach and intestines, and purpura, sometimes occur. *Cerebral hæmorrhage is a frequent cause of death* in this disease.

Breathlessness, apart from that due to pulmonary œdema, is a common accompaniment of renal disease. A paroxysmal dyspnœa, coming on during the night in a person of advanced life, should lead us to suspect the existence of chronic Bright's disease, even although the patient may continue his occupation. *Cheyne-Stokes respiration* may develop towards the end, with or without other symptoms of uræmia.

Nervous Symptoms are not infrequent, apart from the cerebral hæmorrhage just referred to. Thus, *headache* is a symptom which accompanies all renal diseases, particularly those forms which terminate in uræmia. Experience among the aged shows that chronic interstitial nephritis is one of the most frequent causes of headache in advanced life. The patient may continue his work, and present no other symptom, but an examination of the urine may reveal the existence of chronic renal disease. *Vertigo*, *tinnitus*, and various *neuralgias* may also be complained of. *Insomnia* in the aged is another common symptom of chronic renal disease.

[1] As Dickinson and Rolleston have shown in the *Lancet*, July 20, 1895. See also Arterial Hypermyotrophy, § 68. With certain methods of preservation and hardening, or with insufficient staining, the middle coat presents precisely the appearance of fibrous tissue ; but well-stained logwood preparations always reveal the rod-shaped nuclei, and acid orcein will always reveal the elastic tissue boundaries of the tunica media.

The patient complains that he readily drops off to sleep, but as readily awakes, and that he may do so a dozen times every night. As the uræmic condition increases, however, *drowsiness* supervenes, which may pass into *coma*, with or without muttering *delirium*. Sometimes *convulsions* occur before death.

Ocular Changes frequently accompany renal disease attended by albuminuria; and so characteristic are the changes that albuminuria may be diagnosed by their presence. Albuminuric retinitis comprises three alterations in both fundi—papillitis, flame-shaped hæmorrhages into the retina, and white spots of fatty degeneration.

Gastro-intestinal symptoms attend some renal diseases. Thus dyspepsia and irregularity of the bowels are common. *Vomiting*, when persistent, is a symptom of considerable gravity, because it is usually of toxic—*i.e.*, uræmic origin.

§ **268.** The **Complications** and **Secondary Inflammations** in renal cases are very apt to affect the serous membranes, the mucous membranes, and the skin—in a word, the limiting or "surface" structures of the body. The *serous membranes* often become inflamed insidiously, especially the pleura and pericardium. The effusion may sometimes come on very suddenly, but the symptoms may be quite latent; therefore the occurrence of severe dyspnœa in renal cases should lead us to suspect the sudden supervention of a serious pleural effusion (§ 21). In addition to the *pulmonary œdema* already mentioned, a low form of pneumonia or bronchitis is a common complication of nephritis. Endocarditis is relatively rare. Within the last few years it has been recognised that various *skin affections* may complicate renal disease besides dropsy and the cellulitis which is liable to affect dropsical limbs.[1] Amongst these may be mentioned eczema, urticaria, and various forms of erythema. Undoubtedly the most fatal is an epidemic form of exfoliative dermatitis described by the author in 1891.[2] All the cases of renal disease complicated by the epidemic exfoliative dermatitis, which the author has since met with, have ended fatally.

§ **269. Pain in the Kidney.**—Many serious diseases of the renal substance are unaccompanied by any pain or local symptoms. A sense of dull aching in the loins may be present at the onset of acute nephritis. In pyelitis, lumbar pain generally accompanies the appearance of pus in acid urine. The pain is very severe when the pyuria (pus in the urine) is due to a renal calculus (Renal Colic, § 301). Various tumours of the kidney are accompanied by pain, and perinephric abscesses are associated with lumbar pain and tenderness. A dull, dragging pain or weight in the lumbar region, relieved by rest in the recumbent posture, occurs with movable kidney; it is usually on the affected side, and is liable to acute exacerbations resembling renal colic. The lumbar pain of renal disease must not be mistaken for the backache due to congestion of the female generative organs, nor for lumbago, in which the pain is usually of sudden onset, is not confined to one side, and may be accompanied by other rheumatic evidences. Less frequent causes of lumbar pain are aneurysm, cancer, and caries of the vertebræ.

§ **270. Uræmia** is a term used to describe the group of symptoms which arise from retention within the body of those nitrogenous constituents which, under normal circumstances, are elaborated into urea

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[1] Trans. Roy. Med. Chir. Soc. and Trans. Clin. Soc., London, 1899-1900.
[2] Trans. Med. Soc., London, 1891-1892; and *British Journal of Dermatology*, 1892.

and eliminated by the kidney. The exact nature of these retained substances is not yet known. It used to be thought that it was urea which was retained (hence the name), but this is not so, though there is generally a diminution in its secretion. The term uræmia is generally used for the intense *acutely* toxic condition which closes most renal cases ; but it may also be applied to the incipient or *chronic* condition which precedes this, and warns the observant physician of the gravity of the situation. It is the evidence of retention in the blood and the tissues of those substances which form a chain of compounds between the proteid food substances and the nitrogenous disintegration on the one hand, and the nitrogenous output on the other. Thus, uræmia may arise in many hepatic diseases (defective elaboration, as mentioned in the introduction to Chapter XII.), and in many renal disorders (defective excretion).

Uræmia, more or less severe, may occur in almost any disease of the kidney. In renal fibrosis (granular kidney) it occurs in a typically chronic form ; in acute, subacute, and chronic tubal nephritis it is the usual mode of death ; in tuberculous, calculous, and cystic disease, in hydronephrosis and consecutive nephritis, in active or passive congestion, and in lardaceous disease (rarely), mentioned in order of frequency, it is also apt to supervene. Moreover, complete suppression of urine may produce death associated with symptoms of what is called *latent uræmia* (§ 311), in those relatively rare cases of blocking of the ureters.

Symptoms.—Various forms (nervous, gastro-intestinal, dyspnœic, etc.) of uræmia are sometimes described, but it is more convenient and not more artificial to describe the symptoms under *incipient* and *advanced* chronic uræmia, and *acute* uræmia.

1. In *incipient chronic* uræmia, such as occurs typically in chronic interstitial nephritis, the symptoms are vague, and start insidiously. The patient remains at work, but complains of malaise, loss of mental and bodily vigour, general wasting of muscular and subcutaneous tissues, impaired memory, and sometimes sleeplessness after the first few hours of the night. These and the urinary changes may be the only indications of the condition.

2. Symptoms of *advanced chronic* uræmia may succeed the foregoing, or may come on abruptly in a person apparently in good health. They consist of (i.) restlessness and muscular tremors (which constitute one of the most constant symptoms) ; (ii.) persistent headache ; (iii.) drowsiness during the day, with sleeplessness or " cat-sleeps " (dropping off for a few minutes at a time) at night ; (iv.) vomiting, without obvious dietetic irregularity or gastric disturbance, and sometimes diarrhœa ; and (v.) dyspnœa on slight exertion (which is often the first symptom to be noticed), or coming on in paroxysms, especially at night. Uræmic Dyspnœa may be : (i.) *Paroxysmal ;* the attacks coming on

chiefly at night, and resembling cardiac asthma (§ 20*a*). The patient sits up in bed gasping for breath, but there is no cyanosis, and the mind is clear. The breathing is often noisy, with a characteristic hissing quality (Addison). (ii.) *Continuous*, or continuous alternating with paroxysmal. (iii.) *Cheyne-Stokes Respiration* may last for weeks. The pulse slows in the apnœa period, and there is alternate contraction and dilatation of the pupil, the contraction occurring during the period of apnœa.

3. *Acute* or *fulminating* uræmia may supervene at any stage of the foregoing, being ushered in by an increase of the headache, vomiting, or restlessness; or it may come on abruptly in an apparently healthy person. Its leading symptoms are three: (i.) Low muttering delirium; (ii.) stupor, passing into coma, with or without (iii.) convulsions. In some cases of chronic Bright's disease convulsions of coma may constitute the first manifestation of the disease.[1] Sometimes blindness (uræmic amaurosis), without appreciable ophthalmoscopic changes, follows the convulsions, and may last for several days. Deafness or local paralyses may ensue. There is often a urinous odour in the breath.

Diagnosis.—Uræmia is known by the combination of these symptoms, and the presence of a cause, which can be made out on a careful examination of the urine. The diagnosis of uræmic coma is dealt with in Chapter XIX., § 530.

The *Treatment* of uræmia is given under Chronic Bright's Disease (Contracted Granular Kidney) (§ 297), in which malady both chronic and acute uræmia typically occur.

PART B. PHYSICAL EXAMINATION.

The **Examination of the Urine** corresponds, in renal diseases, to the physical examination of other organs. We examine it by (*a*) observing its *physical characters* (§ 271)—viz., its appearance (*i.e.*, its colour, and whether it is clear or cloudy)—its odour, reaction, specific gravity; the presence and characters of any deposit; and its diurnal quantity. (*b*) Then by *chemical analysis* (§ 276) we ascertain the presence or absence of albumen, the presence or absence of sugar, and other substances, according to circumstances. (*c*) Finally, a *microscopic examination* (§ 289) has to be made of any deposit which may be present. It is important in all cases—not only in cases of suspected renal disease—to observe *and to note* the condition of the urine when the patient is first seen, even when the symptoms do not suggest renal disease.

[1] This is explained by a sudden congestion of a chronically diseased kidney, and such cases (coma or convulsions occurring suddenly in an *apparently* healthy person) usually occurred during the winter in the Infirmary.

(*a*) *Physical Characters of the Urine.*

§ 271. **Appearance.**—The colour of the urine depends upon the proportion of pigments present. The chief pigments are urobilin and urochrome, whose antecedents are the blood and bile pigments ; but there are many others.

The urine varies from a pale yellow to a deep amber, according to the DEGREE OF DILUTION of the pigments ; and, as the latter are fairly constant in quantity, a *dark urine* is associated with a smaller diurnal quantity and a higher specific gravity than a pale urine. The urine is dark in excessive perspiration, acute nephritis, and pyrexial states generally. On the other hand, in certain diseases with *polyuria* the urine is *pale*, as in chronic Bright's disease, and in diabetes. In diabetes insipidus and hysteria the urine may be as colourless as water.

The colour of the urine may be altered by MORBID PRODUCTS—*e.g.*, *a dark orange colour to brown*, having a greenish tint on the surface with reflected light, is due to the presence of bile, and will vary in depth of tint according to the amount of bile present. A *red* colour, which may be a dark red or porter colour or only a mere " smokiness," is due to the presence of blood. In diseases in which there is destruction of the red blood-corpuscles the urine is darkened, and this may be a means of distinguishing pernicious anæmia from chlorosis. *Blackish-brown* colour may be due to melanin and certain oxyacids, which cause the urine to darken on exposure. *Milky* urine is due to chyluria. Various DRUGS affect the colour of the urine. A *dark olive-green* or *black colour* may be due to the absorption of carbolic acid—as, for example, when this substance is used for dressings ; or it may appear after the administration of creosote, salol, tar, resorcin, or naphthol. The colour is explained by the presence of hydrochinon, which turns crimson on the addition of ferric chloride. A *reddish-brown colour* may be due to rhubarb, senna, or chrysophanic acid when taken internally, and a *bright yellow colour* follows the administration of santonin. All these turn red on the addition of an alkali. A *colourless* urine is said to result from tannin taken by the mouth, and a *reddish* hue from logwood. Coloured sweets and cakes may cause a coloured urine, from the presence of eosin, methylene blue, or other dye.

Urinary Deposit and Cloudiness will be described in § 288.

§ 272. **Reaction.**—The urine should be tested immediately or soon after being passed. In normal urine an acid reaction is found, turning blue litmus paper red, from the presence of acid phosphate of sodium. On standing for a time decomposition takes place, the urea being transformed into ammonium carbonate $(NH_2)_2CO + 2H_2O = (NH_4)_2CO_3$, and hence the reaction is alkaline. The same change takes place even within the bladder, in cases of chronic catarrh of that organ. Alkalinity due to a *fixed* alkali occurs even in normal urine after meals, or when a patient is undergoing alkaline treatment. A *neutral* reaction may occur under the same conditions. It is sometimes important to distinguish between the alkalinity due to a fixed alkali (*e.g.*, soda or potash salts), and that due to decomposition, which depends upon a volatile alkali (ammonia). This is done by holding over a flame the red litmus paper which has been turned blue ; if due to a volatile alkali, the red colour will return (as the volatile alkali is driven off) ; if to a fixed alkali, the blue colour remains.

§ 273. **Specific Gravity.**—The average specific gravity of the urine varies between 1015 and 1025. It depends chiefly upon two substances normally present : urea and salts (especially chlorides) ; and the simple rule of doubling the last two figures gives roughly a little less than the total quantity of solids present in parts per thousand. Extractives and pigment play only a small part ; and practically—since the salts are fairly constant—the specific gravity, *in the absence of sugar*, gives us a fair measure of the urea present in a given sample. The specific gravity must be considered in relation to the quantity of urine passed ; and to be able to draw accurate inferences from the specific gravity, the urine of a whole day should be collected, and a sample thereof tested (§ 275). The instrument used to test the specific gravity is called a urinometer (Fig. 76). It is convenient to have a metal one with a flanged foot, as shown. The instrument must not touch the

sides of the vessel, and the graduated stem should be read along the surface of the fluid, not at the place where it is raised along the stem by capillarity. These instruments are graduated for a temperature of 60° F. If the temperature of the urine is lower than this, the true specific gravity is a trifle lower than the actual reading.

When enough urine is not obtainable, and a glass bead urinometer is not accessible, mix the urine with one, two, or three times its own bulk of water and multiply the last two figures of the specific gravity by two, three, or four respectively. For example, a mixture of one ounce of urine with *three* ounces of distilled water gives a specific gravity of 1005; the specific gravity of the urine was 1020 (0·005 × 4 = 0·020).

Fig. 76. — URINOMETER, made of metal, and with flanged foot (as supplied by Hicks, of Hatton Garden, E.C.).—The flanges steady it while in the urine, and form a stand when not in use.

§ **274.** The normal **odour** of freshly-passed urine is described as "aromatic"; it is very different from the ammoniacal odour of decomposing urine. The resinous portions of copaiba, cubebs, and other balsams are excreted by the urine, and impart their characteristic odour to it. Turpentine gives to urine an odour said to resemble violets. It may smell of volatile sulphides due to the presence of some microbes, notably *B. coli communis.*

§ **275. The Diurnal Quantity** varies considerably within the range of health. Normally, 40 to 50 ounces (1½ litres) are passed per diem, but the quantity depends upon the amount of fluid drunk, the action of the skin, and the activity of the renal circulation. In order to estimate the quantity of urea, and for some other purposes, it is necessary to collect the whole of the urine that is passed in twenty-four hours—say, for example, from 8 a.m. Monday to 8 a.m. Tuesday. The patient should pass water *at 8 a.m. on Monday morning,* and this should be thrown away. Then all that is passed after that hour, together with what is passed *at 8 a.m. on Tuesday,* should be collected in one clean vessel, which must be carefully preserved from accident or interference. During the whole of that time it is necessary to pass water *before* going to stool, and to add this to the total collected. At 8 a.m. on Tuesday, after passing water and adding it to that previously passed, the whole should be stirred and measured. A specimen from this should then be put into a clean bottle (say 10 ounces), and this should be labelled with the name of the patient, the date, and the total quantity passed in twenty-four hours, and sent for examination immediately.

(*b*) *Chemical Examination of the Urine.*

Normally the urine consists of water containing about 4 per cent. of solids by weight, of which urea, the most important, comprises from 2·5 to 3 per cent. of the total urine, amounting to about 30 grammes per diem.

In disease the three most important substances for which the urine has to be tested chemically are albumen, sugar, and urea.

§ **276. Albumen** is the most frequent of the pathological constituents of the urine. The variety of albumen usually present is serum albumen. (The other forms are given below.)

The chief tests for albumen are: (1) Cold nitric acid; (2) Boiling; (3) Picric acid.

1. *The Cold Nitric Acid Test*[1] is the most delicate, accurate, and convenient test for small quantities of albumen in the urine. Pour some strong nitric acid into the bottom of the test-tube, hold the tube in a very sloping position, and let the urine gently flow upon the top ; a haze of precipitated albumen will appear at the line of junction. It is necessary to wait a few seconds for the haze to appear, when the albumen is very small in quantity ; and the tube should be gently heated at the junction.

The *Fallacies* of this test are not serious. (i.) Mucin, or urates, may form a precipitate, but it occurs *above* the line of junction ; (ii.) in a concentrated urine, a haze of tiny crystals of nitrate of urea may form, but this may readily be dissolved by heat ; (iii.) copaiba and other resins give haze in a similar position, but the odour is characteristic ; (iv.) the haze due to the presence of albumoses disappears on heating, and reappears on cooling ; (v.) both pus and blood contain albumen, and if present in the urine, give this reaction, apart from the presence of free albumen.

2. *Boiling.*—After testing with litmus, boil the urine, and afterwards add a drop or two or acetic acid. A generalised white precipitate forms on boiling if albumen is present, and is not dissolved by acetic acid. It is often convenient to boil the upper part of a column of urine so as to compare it with the lower.

The *Fallacies* of this test are : (i.) *Phosphates* may be precipitated by heat alone if the urine be faintly acid, neutral, or alkaline, but the acetic acid dissolves these and increases the albuminous precipitate. (ii.) Excess of acid may redissolve the albumen ; undue natural acidity may have the same effect, all of which prove the usefulness of test-papers. (iii.) In acid urines a cloud sometimes appears, not on boiling only, as albumen would do, but *after* the acid has been added, due to mucus. (iv.) Copaiba and other resins may give a precipitate insoluble in acid, but their odour is characteristic. (v.) If the urine is not quite clear, it may be necessary to filter it, if boiling the upper part of the tube gives us no information. If turbid from bacteria, add a trace of NaOH, and a deposit of phosphates occurs which carries the bacteria down with it.

3. *Picric Acid Test.*—Float carefully a saturated solution of picric acid on the urine by a pipette. A precipitate forming at the line of junction of the fluids indicates the presence of albumen. Urates, alkaloids, and albumoses may also be precipitated, but disappear on heating.

The *quantitative estimation* of albumen may be roughly determined by boiling as above and setting aside the test-tube for twenty-four hours, and reading off the proportion. It may be more precisely calculated by means of Esbach's albuminometer, a tube graduated for measuring the percentage of albumen. Urine taken from twenty-four hours' collection is poured into the tube up to the mark U, and the reagent[2] is added up to the mark R. The tube is then set aside for twenty-four hours, and the precipitate falls to the bottom. The level to which this reaches is then noted, and the number on the glass indicates the grammes per litre of albumen present. *Fallacies.*—(1) This method is not reliable if the specific gravity of the urine is over 1010. The urine should be diluted to 1010, and a calculation made afterwards by multiplying the result by the number of times of dilution. (2) If the patient is taking alkaline salts, crystals are liable to appear

[1] If HNO_3 is not handy, a saturated solution of common salt, carefully poured down the side of a test-tube containing albuminous urine, gives a haze at the line of junction. But cold nitric acid was decided by a Committee of the Clinical Society of London to be the best all-round test for albuminous urine.

[2] Picric acid, 1 part ; citric acid, 2 parts ; water, 100 parts.

after adding the reagent, and these must be allowed for in reading off the quantity of albumen. Another method is precipitation by boiling, washing the precipitates and weighing.

§ 277. **Nucleo-proteid** occurs sometimes in febrile disorders and in association with destruction of the kidney cells. It gives most of the tests for albumen, but is precipitated on the addition of acetic acid. From mucin it is distinguished by the fact that it is soluble in a large excess of acetic acid, whilst mucin is not.

§ 278. **Mucin** is precipitated, as above mentioned, by most of the same reagents as albumen, but it may be detected by taking a saturated solution of citric acid in a test-tube and trickling the urine down the sloped side of the tube, when a cloud forms above the junction of the fluids. Excess of mucus indicates irritation of the bladder or genito-urinary tract, or a vaginal or uterine discharge.

§ 279. **Sugar (Glucose)** is not a normal constituent of the urine, but it may occur as a permanent or temporary pathological product. The chief cause of *permanent* glycosuria (sugar in the urine) is Diabetes Mellitus (§ 309). It should be remembered that the sugar may disappear from the urine in this disease for some days, and reappear again as abundantly as before. *Transient* glycosuria is found with errors of diet, excess of carbohydrates, usually in gouty people. Its numerous other causes are referred to in § 308.

Tests for Glucose.—(1) *Trommer's Test* constitutes one of the readiest for discovering sugar. To an inch of urine in a test-tube add one-eighth its volume of caustic potash and a few drops of a solution of copper sulphate. On boiling, a red precipitate denotes the presence of glucose.

(2) *Fehling's Test.*—Fehling's solution consists of an alkaline solution of potassio-tartrate of copper, so prepared that 10 c.c. is reduced by 0·05 gramme of glucose. As it is apt to alter on keeping, it should be boiled before using, to make certain that no precipitate forms before adding the urine. It is better to keep the copper solution and the alkali solution in separate bottles, mixing them just before using. Add to it a few drops of urine and boil again ; and then continue adding till equal quantities of urine and Fehling are used. If on further boiling the solution is still clear, no noteworthy quantity of sugar is present. The Fehling's solution must always be in excess, and the boiling must not be too prolonged. This test depends upon the fact that glucose has the property of reducing cupric salts when heated in the presence of a free alkali. $CuSO_4$ added to NaOH causes a pale blue precipitate of hydrated cupric oxide. If a tartrate is present, the cupric hydrate is held in solution (Fehling's solution). If glucose or some other readily oxidisable substance is added, this blue cupric hydrate on gently heating is reduced, and falls as a *red* or *yellow* precipitate of cuprous hydrate (Cu_2O, H_2O), which on longer boiling becomes red or purple cuprous oxide (Cu_2O).

Fallacies.—(i.) The urine to be tested must be freed from albumen, and (ii.) it must not be ammoniacal. (iii.) Other reducing agents may occasionally give the reaction. After the administration of chloroform, chloral, morphia, curare, and some other drugs, a reaction is obtained resembling that due to sugar, but is due probably to the presence of glycuronic acid. Lactose, uric acid, and urates, ammonium chloride, and other ammonium salts, hippuric acid, kreatinine, oxyacids and the products of certain drugs, such as carbolic or benzoic acids, may occasionally be sources of fallacy. To avoid these it is best to control by the Fermentation Test, or to filter a few drachms of the urine through a charcoal filter seven or eight times, by which means all reducing substances other than sugar are removed.[1]

Quantitative Estimation by Fehling's Solution.—The urine should be a sample taken from the total collection of twenty-four hours. Fill a burette with urine diluted to 1 in 20, and have 10 c.c. Fehling's solution in a porcelain dish, diluted with water. Boil the solution, and while boiling allow drops of urine to mix with

[1] Saundby, *Brit. Med. Journ.*, April 14, 1900.

it, stirring all the while. Urine must be run in from the burette till the fluid is colourless ; this is difficult to decide unless the dish be tilted so that it shows against the white background apart from the red precipitate which collects at the bottom. Read off the amount of urine required for complete reduction and calculate. Supposing we find that 60 c.c. *diluted* urine from the burette are required to decolourize the 10 c.c. Fehling (representing 0·05 gramme glucose), then $\frac{60}{20}=3$ c.c. urine contain 0·05 gramme glucose. Then from this, as we know the number of c.c. urine passed by patient in twenty-four hours, it is easy to calculate the percentage of sugar excreted in that time. Carwardine's Saccharimeter (Fig. 77) may be employed in this process if an ordinary burette, as used in the laboratory, is not accessible.

(2*a*) *The Ammoniated Cupric Solution* (Pavy's Test) is a modification of Fehling's solution. It contains free ammonia, and this keeps the oxide in solution, which would otherwise be precipitated by the addition of diabetic urine. Hence the *blue colour* of the fluid is discharged *without the formation of any precipitate*, and it

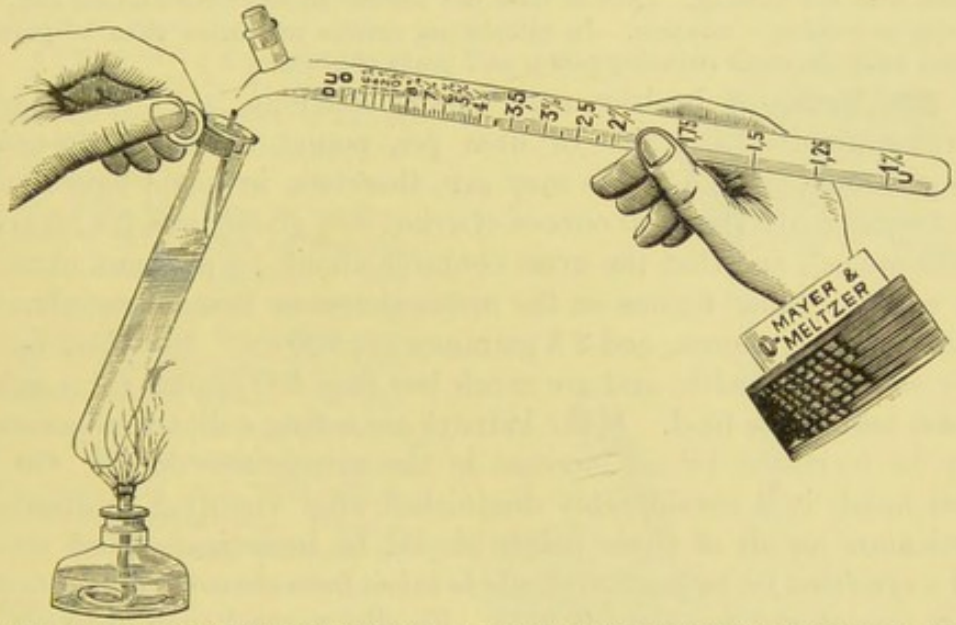

Fig. 77.—Carwardine's Saccharimeter.—A sample of the twenty-four hours' collection of urine is used to fill the burette (on right of figure) up to the letter U. Dilute it by adding water to D U, and mix thoroughly. Fill the measure supplied with the apparatus up to F with Fehling's solution, and dilute it by adding water to D F. Pour this diluted Fehling into the test-tube shown in figure and boil it. While it is gently boiling add the diluted urine drop by drop from the burette until all the blue colour has gone from the supernatant fluid. This may take some little time, as it is necessary after each boiling to wait a minute for the precipitate to subside a little. For calculation see text.

is thus easier to determine the exact point when the whole of the cupric salt is reduced than is the case when Fehling's solution is employed. Pavy's solution (10 c.c. of which represent 0·005 gramme of sugar) is therefore very useful as a quantitative test. It is usually applied by means of a special apparatus.

(3) The *Fermentation Test* constitutes the ultimate test in all cases of doubt, since sugar is the only known substance fermented by yeast. After seeing that the urine is acid, fill a test-tube with it, and insert a piece of German yeast ; then invert the tube over a saucer of water (or mercury) and place them in a warm place. Have a control tube beside it with normal urine or plain water. If sugar is present, bubbles of CO_2 will form and collect at the top of the tube.

This test can also be applied for the *quantitative estimation* of sugar, by Robert's differential density test. *Method.*—Have two 12 ounce bottles with a slit cut in the side of the corks for the gas to escape, and put in each 4 ounces of the urine taken from a twenty-four hours' sample. Add a piece of German yeast, the size of a walnut, to one of them, and set them aside in a slightly warm place for about

twenty-four hours. Then take the specific gravity of the two samples, and the difference between them gives the measure of the sugar in grains per ounce. The percentage is found by multiplying this difference by 0·23. Thus, supposing the specific gravity of the two samples is 1050 and 1005 respectively, there were 45 grains of sugar per ounce, and $45 \times 0{\cdot}23 = 10{\cdot}35$ per cent. It is important to wait until all fermentation has ceased, and to see that no decomposition of urea has taken place in the control bottle.

(4) *Picric Acid Test.*—Boil a few drops of liq. potassæ with a saturated solution of picric acid. Add urine and boil ; a dark claret-red colour denotes glucose.

(5) *Phenyl-Hydrazine Test.*—To about a drachm of urine in a test-tube add 4 grains phenyl-hydrazine hydrochlorate and 2 grains of sodium acetate ; boil it in a water-bath for about half an hour. Allow it to cool by placing the tube in cold water. A yellow deposit forms, which under the microscope shows fine yellow needle-shaped crystals in sheaves.

Lactosuria.—Lactose may be present in the urine in considerable quantity in women who are nursing. Lactose does not answer to the fermentation test, but it reduces Fehling's solution. In calculating results remember than 10 parts of lactose have the same reducing power as 7 parts of glucose.

§ **280. Urea.**—A healthy male adult, weighing, say, 140 pounds, excretes about *3 5* grains of urea per pound of his body-weight (0·5 gramme per kilo). We may say, therefore, in round figures, that he excretes daily about *50* ounces of urine, *500* grains urea (or *10* grains to the ounce), and that the urine contains about *2 3* per cent. of urea,[1] the corresponding figures on the metrical system being approximately 1,320 c.c., 33 grammes, and 2·3 grammes per 100 c.c. But these figures vary widely in health, and are much less (say 300 grains) for a lighter person taking less food. If the kidneys are acting well, the urea output may be increased by an increase in the nitrogenous food.[2] On the other hand, it is considerably diminished after vomiting or diarrhœa. Particulars on all of these points should be investigated and noted ; and a *specimen for estimation should be taken from the urine of twenty-four hours, mixed and measured* (§ 275). Finally, several such observations should be made before concluding that there is really deficient nitrogenous elimination. There is a deficient elimination of urea sooner or later in nearly all renal diseases (the accompanying effect being uræmia, § 270), in certain hepatic diseases, in myxœdema, Addison's disease, and melancholia.

It is now considered that the total quantity of salts in the urine is a better gauge of the kidney efficiency than the amount of urea (see § 285).

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[1] It is useful to know that the number of *grains per ounce* multiplied by 0·23 gives the *percentage.*

[2] The diurnal quantity of urea depends partly upon the destruction of nitrogenous tissues in the body, but chiefly upon the amount of proteid ingested. The urea dependent upon the latter for its source is usually accompanied by a parallel increase in the sodium chloride in the urine, because most proteid foods are rich in sodium chloride. If, however, the urea, which depends on tissue-destruction for its source, be increased, it is unaccompanied by a parallel increase in the sodium chloride. It is sometimes in practice a little difficult to determine whether deficiency of urea depends upon deficient tissue-destruction or deficient intake of proteids. But if the physiological facts just named can be relied upon, we have in the estimation of the chlorides an answer to the question.

Estimation of Urea.—The specific gravity of the urine gives us (in the absence of sugar) a very fair idea of the quantity of urea being excreted ; indeed, that is the chief reason why we habitually use the urinometer (§ 273). The rapid crystallization of nitrate of urea in a test-tube when an equal bulk of strong nitric acid is added to the urine and the mixture cooled, suggests excess ; but for accurate results it is necessary to determine the *total nitrogen* in the urine (the greater portion of this being in the form of urea) by volumetric analysis. This has been now rendered available for clinical practice by the simple apparatus described below. Albumen, if present, should be separated by boiling and filtration before beginning the estimation of urea, and the specimen of urine should be from the twenty-four hours' collection mixed.

Doremus's Ureameter (Fig. 78) is so easily used that it can be employed for estimation in one's consulting room. There are two stages in the process : (1) To fill the vertical U tube with a solution of hypobromite of sodium. This must be freshly prepared immediately before use, and it is best to keep it in two solutions, potash in one, bromine in the other, to be mixed in equal parts just before using. First, holding the tube vertically, the operator pours the solution into the bulb until it is about half full, then he inclines the apparatus horizontally so that the fluid passes up into the long or vertical limb of the U tube ; then he restores it to the vertical position, and repeats the process until the *vertical limb is quite full*, and the bulb is one-third full or thereabouts. The tube now contains about 35 c.c. (2) The second stage requires considerable exactitude. The accuracy of results depends upon the care with which the pipette is manipulated. Having drawn up the urine *very precisely* to the 1 c.c. mark on the pipette, wipe the outside of the pipette rapidly with a towel and introduce it as shown in the figure just beyond the bend of the U tube. Now comes the most difficult part of the operation. The rubber top is gently squeezed so as to SLOWLY and CONTINUOUSLY press out the urine. *Watch the tip of the pipette carefully* as the urine slowly passes out, so as to prevent (i.) the hypobromite from coming back into the pipette ; and (ii.) the air from the pipette going out into the hypobromite. If either happens, the whole must be done again. The nitrogen, which is rapidly liberated and collects in the upper end of the vertical limb of the U tube, is the exact measure of the urea contained in the 1 c.c. of urine used. Set the tube carefully for an hour until the bubbles and heat evolved have subsided, then read off the percentage of urea, or grains per ounce, as marked on the side of the vertical limb. The urine must be examined afresh, *and if it contains albumen this must be removed by boiling and filtration*. The U tube should be rested on a table or stand. This process estimates the uric acid and the other products of nitrogenous disintegration as well as the urea, but this does not invalidate the process for clinical purposes.

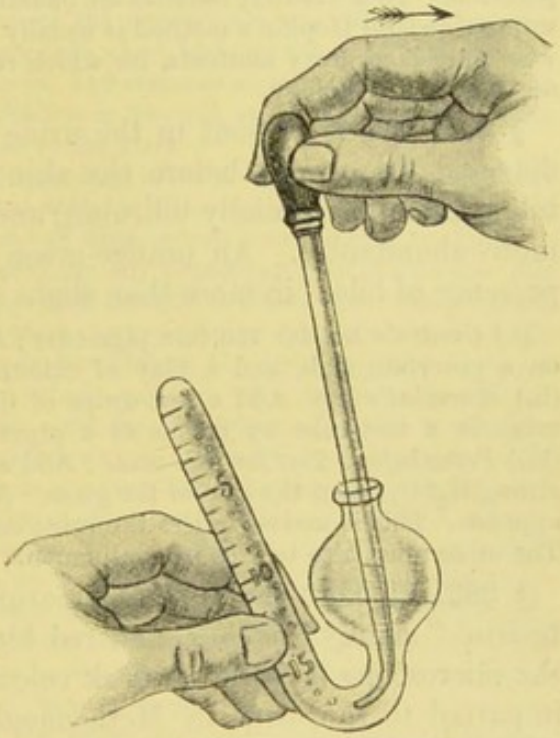

Fig. 78.—Doremus's Ureameter.

§ **281. Uric** (*i.e.*, **Lithic**) **Acid,** either free or combined in the form of **urates,** is normally present in a sample from a day's collection to the extent of 0·04 per cent., or about 8 or 9 grains per diem. According

to Dr. Alexander Haig, it bears in health a fairly constant proportion to the amount of urea, 1 grain of uric acid per day (per 10 pounds body-weight) to 35 grains of urea per day (per 10 pounds body-weight); the two rising and falling together. Others (*e.g.*, Dr. A. P. Luff) say the proportion is about 1 to 50. Uric acid and urates when in excess are best detected as a cloudiness or deposit (§§ 288 and 289). Their chemical quantitative estimation is a matter of some delicacy and difficulty.

The murexide chemical *test* for uric acid, free or in combination as urates, is performed by adding nitric acid to the suspected deposit in a porcelain dish, heating to dryness, and placing a drop of ammonia on another part of the dish. Where the two join, a characteristic purple coloration appears. If a drop of caustic potash be placed on another part of the dish, a blue coloration appears at the junction. To accurately *estimate the quantity* of free or combined uric acid in the urine, Gowland Hopkin's method is usually employed, or that of Haycraft, both of which are laboratory methods, for which reference should be made to a textbook on chemical pathology.

§ **282. Bile** is present in the urine in cases of jaundice, and can be detected there even before the skin assumes a yellow colour. Both bile pigments (especially bilirubin) and bile acids are present, the former more abundantly. An orange-green colour of the urine betrays the presence of bile if in more than slight amount.

(i.) *Gmelin's test* for the bile pigments: Add a drop of nitric acid to the urine on a porcelain slab, and a play of colours will be seen where the fluids meet. (ii.) *Marechal's test:* Add a few drops of tincture of iodine to the surface of the urine in a test-tube by means of a pipette, and a green reaction is obtained. (iii.) *Pettenkofer's Test for Bile-acids:* Add a solution of cane-sugar to urine; pour strong H_2SO_4 down the side of the glass. At the junction line a cherry-red colour appears. This is useless unless the urine contains a considerable quantity of bile. The urine must also be free from albumen.

§ **283. Blood** in the urine (Hæmaturia) imparts to the urine a characteristic "smoky" colour, and red blood-cells may be identified under the microscope (§ 290). A dark colour of different shades may also be imparted to the urine by Methæmoglobinuria, Hæmatoporphyrinuria, Alcaptonuria (all of which are referred to below), and Carbolic Acid. The most delicate test for hæmoglobin, either free or combined in the corpuscles, is the spectroscope (see Chapter XX.).

Chemical Test for Blood.—Add a few drops of freshly-prepared tr. guaiaci to the urine and shake, then add excess of ozonic ether. A blue line appears at the junction of the fluids. The same reaction may be obtained by using filter- or blotting-paper. Allow a drop of each of the reagents to fall on the paper beside a drop of the urine, noticing the colour at the junction of the three drops. *Fallacies.*—Saliva gives the same reaction, and so do iodides, in patients taking these salts. Pus gives a blue colour with guaiacum alone. It is very important to have the tincture of guaiacum freshly prepared, and to this end it is best to dissolve a little of the resin in rectified spirit at the time when it is used.

Hæmoglobinuria is always present with hæmaturia, because the corpuscles break up. Its presence *alone* is rare, and can only be proved by examining the centrifugalised deposit of absolutely fresh urine under the microscope and finding *no red cells*, although blood is present.

Methæmoglobinuria.—The characteristic smoky colour of the urine in hæmaturia

of renal origin depends largely on methæmoglobin, a substance formed from hæmoglobin by the action of acid urine. It is this pigment also which is found in Paroxysmal Hæmoglobinuria. It is recognised by the spectroscope.

Hæmatoporphyrinuria (Iron-free Hæmatin in the Urine).—The urine has a dark cherry-red colour like port-wine, but gives no guaiacum reaction. It is found after excessive drugging with sulphonal, and is an indication for at once stopping the drug and giving alkalies freely. It is known by its spectroscopic bands. If these cannot be detected in the urine, the hæmatoporphyrin should be extracted by shaking with acetic ether or amylic alcohol, after adding a few drops of acetic acid ; the extract so obtained will give the four characteristic bands.[1]

§ **284. Pus** in the urine is best detected by the microscope (§ 290). When in considerable quantity it may be detected chemically by the addition of an equal quantity of liq. potassæ to the deposit. A ropy gelatinous mass is formed, which pours from one test-tube to another like a fluid jelly. This test is only applicable when a fair quantity of pus is present. In small quantities it is best to make a microscopic examination of the deposit for pus cells. When pus comes from the *kidney* the urine is, at any rate when first passed, acid, and the pus is *uniformly disseminated* through the urine, and remains so for some time. When it comes from the *bladder* the urine is alkaline or neutral, and the pus very rapidly collects into a *creamy layer* at the bottom of the glass.

§ **285. Salts in the Urine.**—The total quantity of salts in the urine is of great importance, for it indicates the functional activity of the kidneys. In this respect it has usurped the position of the amount of urea, which was thought formerly to give the best indication. It is estimated by (*a*) *Cryoscopy*, (*b*) *Hæmolysis* (Sir A. E. Wright's method). (*a*) *Cryoscopy.*—This method depends upon the principle that the freezing-point of a solution of salts is proportional to the amount of salt present. If the total quantity of salts is diminished, the freezing-point is correspondingly higher. The method is one which requires very careful adjustment, and is therefore a laboratory procedure. (*b*) *By Hæmolysis.*—Sir A. E. Wright has devised a plan whereby the kidney efficiency is measured by hæmolysis. The hæmolytic power of a solution of salts is proportionate to the quantity of salts present. First we estimate the quantity of decinormal saline solution necessary to lake a given amount of blood. Secondly, the quantity of urine required to lake the same amount of blood. From this we know the amount of salts in the urine, for the amount required contains as much as the decinormal saline. Thirdly we may in the same way estimate the proportion of salts in the patient's serum. The ratio of the amount of salts in the urine to the amount in the serum is a measure of the renal adequacy, and is called the " kidney coefficient."[2]

Chlorides.—The chlorides found in the urine are principally salts of sodium, and vary in *health*, according to the food taken, from about 11 to 15 grammes daily. In *disease*, the chlorides are increased during convalescence from fevers, during the stage of absorption of œdema or other forms of serous exudations, and in diabetes insipidus. They are diminished in acute fevers, especially pneumonia (reappearing at the crisis), in renal diseases with albuminuria, in gastric disease, such as cancer or dilatation, where the digestive power is diminished, in anæmic conditions, and, it is said, in melancholia, idiocy, and dementia.

Test.—Add a few drops of HNO_3 to the urine, and an equal bulk of 3 per cent. solution of $AgNO_3$. A curdy precipitate follows if the chlorides are normal in quantity ; if the urine only becomes milky, they are diminished.

Quantitative Estimation of Chlorides (Mohr's Method).—After the urine has been freed from albumen, take 10 c.c. and mix with it 50 c.c. of distilled water ; then add a pinch of calcic carbonate and 3 drops of a neutral chromate of potassium solution (1 in 20). The calcic carbonate neutralises any free acid which may be

[1] Readers who are interested in the subject of proteids and pigments in the urine should consult the writings of Dr. Archibald Garrod (*Journ. of Physiology*, 1894, xvii., p. 349 ; and in the Bradshaw Lectures, the *Lancet*, November 10, 1900).

[2] For further details see the *Lancet*, October 21, 1905.

present. To this a standard solution of silver nitrate is slowly added from a burette, stirring constantly. The white precipitate of chloride of silver separates first, but the silver nitrate solution must be added drop by drop until the faintest tinge of pink appears. The pink colour is an indication that chromate of silver is now being formed, all the chlorides having first united with the silver. *Calculation.*—Take the total number of c.c. of silver nitrate used, and deduct 1 c.c. to account for other substances present in urine which unite with the silver ; then every remaining c.c. of the solution used represents 10 milligrammes of sodium chloride.

Phosphates.—Phosphates in excess may be attended by a group of somewhat vague symptoms, which are elsewhere described (§ 314).

Tests.—In an *alkaline* or neutral urine, phosphates spontaneously form a cloudy precipitate, which is increased on boiling, but which disappears on acidifying the urine. If present in a urine that is already alkaline, the deposit is distinguished from pus by the fact that it is dissolved by acetic acid. The microscope enables us to distinguish between pus and phosphates with certainty. This instrument is indispensable when, as often happens, the two deposits occur together. If the urine be *acid*, it is necessary first to add some caustic potash ; and if it be then heated the phosphates are precipitated.

Sulphates are also normally present in the urine, and there is a total increase with increase of diet or fever. They exist in two forms : (*a*) as potassium or sodium sulphate (*inorganic sulphates*) ; (*b*) as combinations of cresol, phenol, indol, skatol, etc. (*organic or ethereal sulphates*). A relative increase of the latter group is of considerable importance. It occurs when phenol or allied substances are given as drugs, and as the result of the action of putrefactive organisms on intestinal contents or abscesses. The exact determination of the total sulphates or of the proportion of inorganic to organic is too complicated for ordinary clinical work. To gauge roughly the proportion of organic sulphates, add to the urine an equal volume of alkaline barium chloride solution. This precipitates the inorganic sulphates and phosphates. Filter ; add hydrochloric acid until the filtrate is strongly acid, and heat. The organic sulphates are thus rendered inorganic, and are precipitated. They should normally form a white cloud only, and if the precipitate is dense the proportion of organic sulphates is abnormally high.

§ 286. Proteids in the Urine.—Besides serum albumen, hæmoglobin, methæmoglobin, hæmatoporphyrin, and mucin and nucleo-albumen, the only proteid which in the present state of our knowledge has any clinical significance is albumose. For further details the reader is referred to larger works on the subject.[1]

Albumosuria was formerly known as **Peptonuria**, but it is now supposed that true peptones never appear in the urine. Albumosuria occurs where there is great destruction of white corpuscles, and therefore whenever there is a large collection of pus in the body—*e.g.*, in empyema and any abscess formation. It is useful in deciding the character of an effusion, pleural, peritoneal, or meningeal. Albumosuria also occurs whenever tissue destruction takes place under the action of micro-organisms. It appears in the resolution stage of pneumonia, and has been described in connection with osteomalacia, with certain liver diseases, such as acute yellow atrophy, with ulceration of the intestine, with dyspepsia, sometimes when excess of animal food is consumed, and with some cases of nephritis (together, of course, with albuminuria). When occurring with multiple myelomata, it constitutes a rare disease known as myelopathic albumosuria or Kahler's disease.[2]

Test.—Primary and secondary albumoses are found, the latter being more nearly related to the peptones—but they have the same clinical significance. (1) Presuming the urine to be free from ordinary albumen, add HNO_3 drop by drop to the urine ; if a precipitate is formed, which disappears on heating and reappears on cooling, *primary* albumoses (? peptones) are present. Both forms of albumose react to the next test. (2) Acidify the urine strongly with acetic acid, add an equal

[1] See an interesting discussion on this subject at the Roy. Med. and Chir. Soc. of London, the *Lancet*, 1900, vol. i.

[2] Parkes Weber, Med. Chir. Soc. Trans., vol. lxxxvi.

bulk of saturated salt solution till a cloud forms ; if it disappears on heating and reappears on cooling it is due to albumose.

§ 287. **Other Rare Constituents** in the urine are acetone, diacetic acid, dioxyphenyl-acetic acid, and indican.

Acetonuria.—An infinitesimal trace of acetone is always present in the urine, and this may be increased by a highly albuminous diet. Acetone is also increased in some febrile states, in cancerous cachexia, and other conditions of inanition. It occurs principally in diabetes, and it was formerly believed that the presence in definite quantity in the urine heralded an attack of diabetic coma, but the evidence of this is not very satisfactory. It is found in abundance in delayed anæsthetic poisoning, in " cyclical vomiting " of childhood, and in poisoning by certain drugs, especially by morphia and salicylates,[1] in all of which it is associated with diacetic acid, and in diabetes, cyclical vomiting, and delayed anæsthetic poisoning with β-oxybutyric acid also.

Test.—Add to a few inches of urine in a test-tube a drop or two of 10 per cent. solution of sodium nitroprusside, and then pour gently down the side of the tube strong ammonia solution. A plum-coloured ring forms at the junction of the fluids on standing.

Diacetic Acid is found in the same conditions as acetonuria. Its presence is detected by adding a few drops of a strong solution of ferric chloride, when a Burgundy-red colour appears.

Alcaptonuria is a condition where the urine forms a pellicle on the surface and darkens from the surface downwards on standing exposed to the air, due to the presence of dioxyphenyl acetic acid. It is an inborn error of metabolism,[2] and has no known clinical significance. Its only importance lies in the fact that it reduces Fehling's solution and leads to difficulty in life insurance.

Indicanuria.—Indican (indoxyl sulphate of potassium) is found : (1) where there is undue intestinal putrefaction ; hence it is present also in cases of gastric disorder with deficient HCl, HCl being an antiseptic agent. (2) In peritonitis, and some other diseases where the peristalsis of the small intestines is impeded. Some maintain that in simple chronic constipation without intestinal obstruction there is no indicanuria, but von Jaksch and others affirm its presence in constipation and all gastric disorders. (3) With empyema, putrid bronchitis, etc. (4) Indican is also said to be present, in small quantity, in decomposing urine.

Test.—Add an equal bulk of strong fuming HCl and a few drops of a solution of hypochlorite of lime (bleaching powder) to the urine. This, on standing for a few minutes, produces a blue colour due to indican, which may be extracted by shaking up with about one-third its volume of chloroform.

(c) *The Urinary Deposit.*

§ **288. Cloudiness of the Urine** (naked-eye examination). In healthy urine there is no deposit, but most of the normal constituents, if in excess, and some abnormal substances, become evident as a sediment or turbidity. (1) A bulky pinkish turbidity and deposit in an acid urine, which forms when the urine cools, indicates the presence of *lithates*—*i.e.*, urates. It is the commonest of urinary deposits, and its appearance when the urine gets cold is typical. (2) *Uric Acid* is evident to the naked eye as a sandy deposit resembling red cayenne pepper. (3) A white flocculent turbidity in an alkaline or neutral urine indicates the presence of *phosphates*, which are cleared at once by the addition of a few drops of acetic acid. (4) *Calcium oxalate* gives a typical

[1] Langmead, *Brit. Med. Journ.*, vol. ii., 1907.
[2] A. E. Garrod, *Lancet*, vol. ii., 1908.

"powdered-wig" deposit of fine white points seen on the surface of a mucous cloud. (5) A fine cloud of *vesical mucus* is normally present in the urine, although it is only visible when the entangled debris and epithelial cells are sufficiently plentiful. (6) *Pus* forms a deposit which resembles phosphates to the naked eye, but it is readily distinguished under the microscope. (7) Urine is sometimes cloudy from the presence of *bacteria*, and this cloudiness cannot be cleared by boiling or the addition of acids.

§ 289. **Specimens** of the deposit must always be examined **microscopically** in cases of suspected renal disease. The urinary deposit is best examined after the urine has stood for some hours in a conical glass, or after the specimen has been centrifugalised.[1] Take a pipette, close it at the top with the right forefinger, pass it to the bottom of the glass, allow a small quantity of the sediment to enter, withdraw the pipette, wipe its exterior with a cloth, place the point on a slide, then surround the pipette with the palm of the left hand, the warmth of which will cause a drop to exude. Cover the drop with a cover-glass, and examine first under a $\frac{1}{3}$ or $\frac{1}{2}$ inch objective, then under a $\frac{1}{6}$ or higher. The deposit normally contains foreign substances, such as cotton and woollen fibres, etc., and a few bladder (and in women nearly always a few vaginal) epithelial cells, which are recognised by their large and nucleated appearance. Inquiry should always be made as to the sex of the patient, and in women if any leucorrhœa is present. If so, it is very desirable to draw off a specimen of urine by the catheter.

The urinary deposit may contain ORGANISED SUBSTANCES (§ 290), or CRYSTALLINE or unorganised substances (§ 291).

§ 290. The **Organised Constituents** of the urinary sediment are of far more serious import than the crystalline substances. They comprise TUBE-CASTS (which are the most important), EPITHELIAL CELLS, PUS CELLS, BLOOD CELLS, spermatozoa, and certain rarer structures such as microbes, fat cells, etc.

Tube-casts and renal **Epithelial Cells** are present in all renal maladies attended by shedding or destruction of the renal epithelium. When tube-casts are abundant in the urine microscopic examination of the sediment permits of their ready detection. But if, on the other hand, they are present only in small numbers, they may be easily overlooked, and this all the more so when, as in chronic interstitial nephritis and in amyloid disease, the urine is abundant and of low specific gravity, so that any suspended matter it contains is deposited only slowly and incompletely. Moreover, these are the exact instances in which the casts are apt to be of the hyaline variety, and their almost transparent character renders them inconspicuous objects in the microscopic field. Hence the search for tube-casts must be conducted with great care if the risk of a false conclusion is to be avoided. One of the best methods, after settlement or centrifugalisation of the deposit, is to examine it with a moderately low power of the microscope, using a narrow diaphragm and shading the light so as to have the field only feebly illu-

[1] For centrifugalisation a special apparatus is necessary, the specimen being placed in a tube on the edge of a rapidly rotating wheel. It is accurate, convenient, and saves time.

minated. Any suspicious-looking object can be brought into the centre of the field and examined with a stronger lens. In this way casts may be detected which in a strong light would readily be missed, and if several slides have been prepared and examined in this manner the detection of any casts present in the urine is rendered fairly certain. But the examination should be repeated on several occasions in any urine containing albumen before a negative conclusion is finally arrived at. There is often a special degree of difficulty in finding casts in alkaline urine, and in decomposing urine they undergo disintegration.

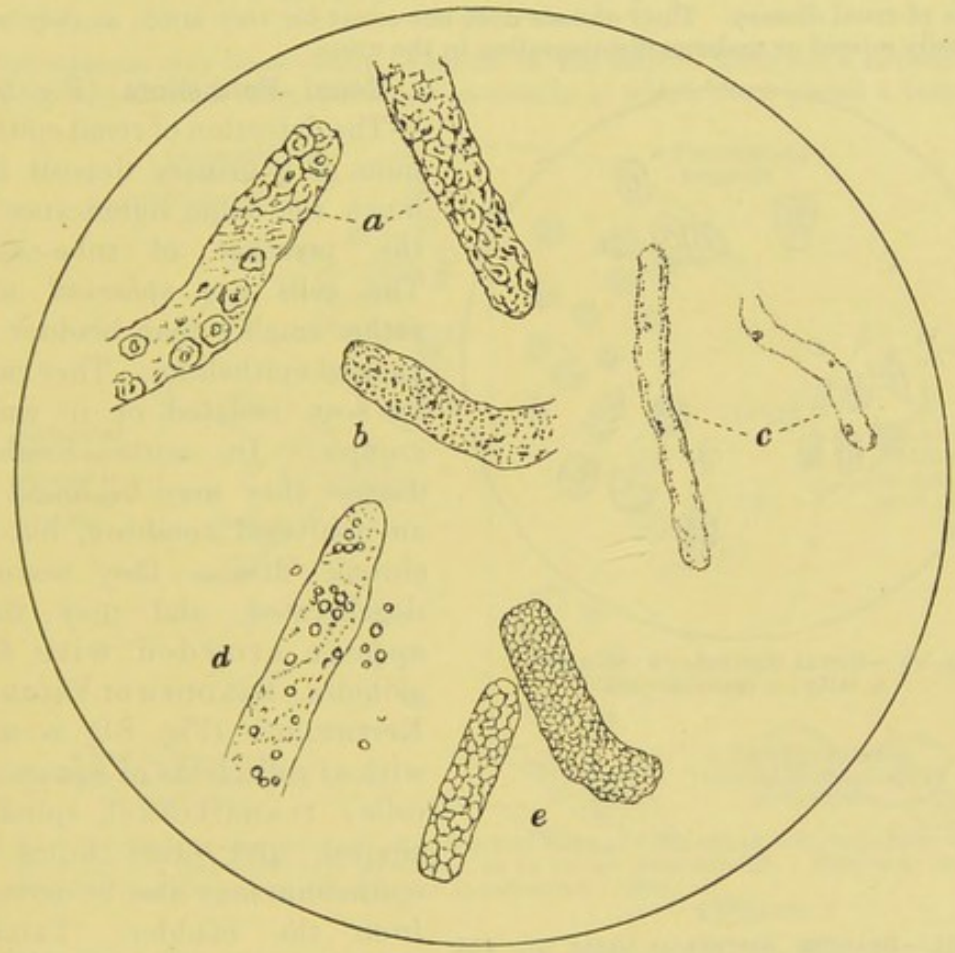

Fig. 79.—Renal Tube-casts—*a*, epithelial casts ; *b*, granular casts ; *c*, hyaline casts ; *d*, fatty cast ; *e*, blood casts.

The clinical importance of tube-casts in the urine is that, with but few exceptions, they definitely indicate disease of the renal epithelium. Thus, when found in a urine containing albumen they add great weight to the opinion that the albuminuria is a result of some structural change of the kidney. Similarly in cases of pyuria and hæmaturia the detection of tube-casts not only suggests that the pus and blood are of renal origin, but also that the kidney is becoming affected. It must be remembered that more than one part of the urinary tract may be diseased at one and the same time. In the urine of patients who are jaundiced, tube-casts

may often be found without, either at the time or subsequently, any evidence of renal disease.

The different kinds of casts (Fig. 79) are intermingled in most cases. But, in general terms, *epithelial casts* and *blood casts* are indicative of the earlier and more acute stages of parenchymatous nephritis. *Waxy casts* are not peculiar to lardaceous kidney, but occur in other forms of long standing renal disease. These and *fatty casts* indicate that the inflammatory process is passing to a degenerative stage. *Granular casts* are more abundant in chronic renal disease, both tubal and interstitial. *Hyaline casts*, which must not be confused with waxy casts, occur in all forms of nephritis, both acute and chronic, and also in health after middle age. Tube-casts in abundance always form a serious symptom, but a single cast may occur in normal urine. They are more abundant in the acute than the chronic forms of renal disease. Their *absence* does not count for very much, as they may be easily missed or undergo disintegration in the urine.

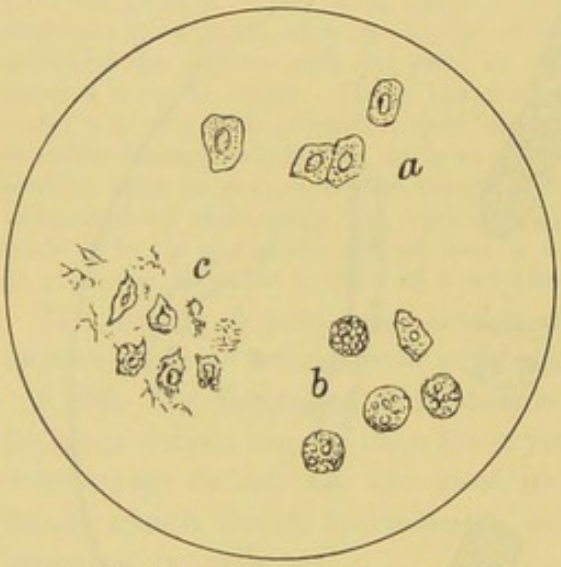

Fig. 80.—Renal Epithelium—*a*, normal; *b*, fatty; *c*, disintegrating.

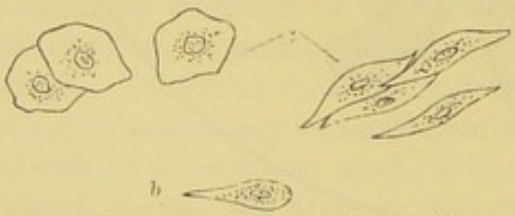

Fig. 81.—Bladder Epithelial Cells (*a*); and Tailed Epithelium (*b*) from the pelvis of the kidney.

Renal Epithelium (Fig. 80).—The detection of renal epithelium in a urinary deposit has much the same significance as the presence of tube-casts. The cells are *spherical* and rather smaller than bladder or vaginal epithelium. They may be seen isolated or in small groups. In acute Bright's disease they may be found in an unaltered conditon, but in chronic disease they become degenerated, and may thus appear crowded with fat globules. Bladder or Vaginal Epithelium (Fig. 81) is met with as collections of squamous cells; transitional, spindle-shaped, and other forms of epithelium may also be derived from the bladder. Tailed Epithelium may be derived from the pelvis of the kidney, and the presence of cells having this elongated character would greatly aid the diagnosis in a case of suspected pyelitis. It must be remembered, however, that the male urethra and the prostate gland yield epithelium practically identical with the above. A deposit from this source is not uncommon in cases of chronic prostatitis, the result of a former gonorrhœa.

Pus Corpuscles, under the microscope, are of globular form with a diameter about one-third larger than that of a red blood-cell. The corpuscles are opaque and granular, but when treated with acetic acid

they clear up, and a nucleus, often partially split into two or more divisions, is seen (Fig. 82, *a* and *b*).

Red Blood-Corpuscles.—The detection of red blood-corpuscles in a urinary deposit is, of course, conclusive evidence of the presence of blood. In most fresh urines they are readily distinguished, as they retain their bi-concave form and the outline shows a double contour (Fig. 82, *c*). But sometimes the corpuscles become much changed. Thus in a very dilute urine they are apt to become distended by imbibition, and then are seen as circles having sharp delicate outlines (*d*). In other instances they become crenate, shrunken, and deformed (*e* and *f*).

Spermatozoa may occasionally be found in the urine. Each has a minute oval or pear-shaped head, from the larger extremity of which there passes a long and

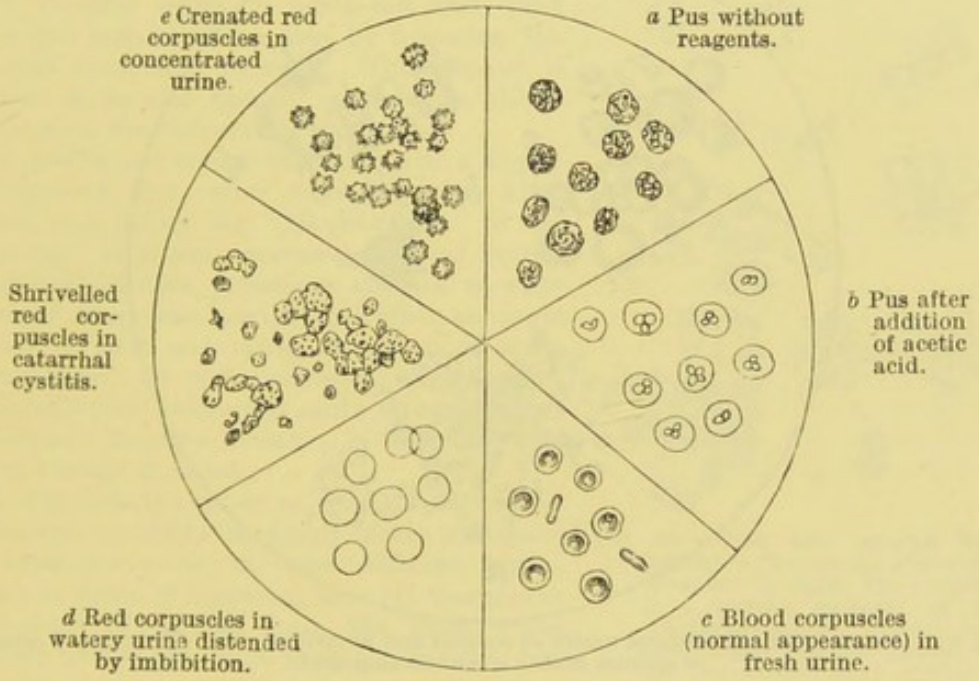

Fig. 82.—Various appearances of RED BLOOD CORPUSCLES and PUS CELLS.—In very pale, watery urine the red corpuscles may be so pale as to escape detection (*d*). They may then be revealed by adding a solution of iodine in potassium iodide.

delicate tail. The total measurement of the spermatozoon is about $\frac{1}{500}$ inch in length.

Microbes.—Numerous microbes are found in the urine, especially when decomposition has occurred either within the bladder or subsequently. The most constant are the *Bacillus ureæ*, *Vibriones*, and the *Hay bacillus* (bacillus subtilis), which have no special clinical significance apart from putrefactive changes. *Gonococcus* is found in cases of gonorrhœa, and the *typhoid bacillus* is very abundant in cases of enteric fever.

The TUBERCLE BACILLUS may be found in tuberculous disease of the bladder or pelvis of the kidney, and is therefore a sign of great value. In appearance under the microscope it resembles the smegma-bacillus and other organisms. Its special staining reaction will be given in Chapter XX., § 627 It is difficult to find in the urine early in the disease, and in obscure cases the experimental test upon guinea-pigs should be employed, the urine for inoculation being collected through a sterilised catheter into a sterilised bottle.

The B. Coli Communis is sometimes found in the urine in pure culture, and may produce an Ascending Pyelo-nephritis (§ 305). The bacilli of the urine and their clinical and pathological relations would well repay study.

§ **291. Crystalline and Inorganic Deposits** in a urinary deposit are usually of less serious import than the organised substances above noted.

In acid urines we meet chiefly with urates, uric acid, oxalates, and—among the rarer substances—stellar phosphates, cystin, xanthin, hippuric acid, tyrosin, and leucin.

In neutral or alkaline urines we meet chiefly with triple phosphates (occasionally urate of ammonium and calcium carbonate).

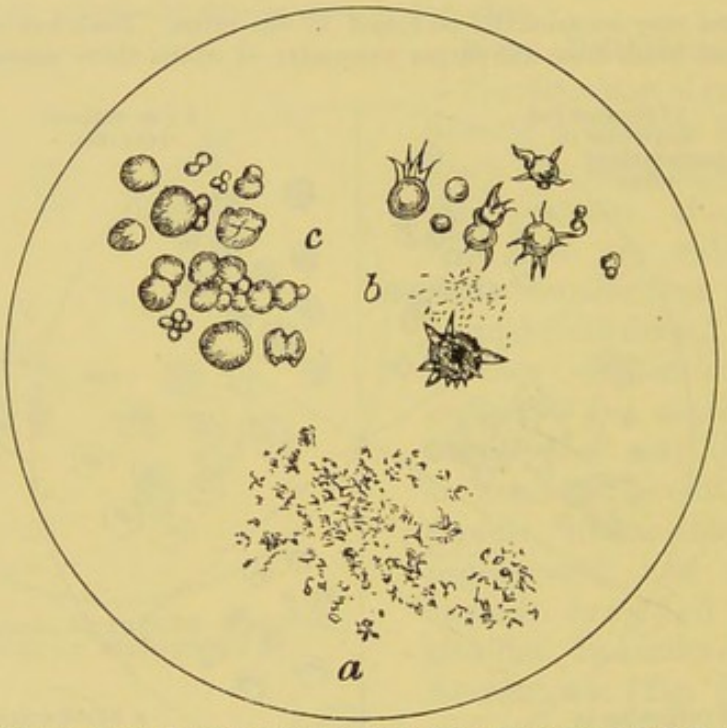

Fig. 83.—Urates.—*a*, Amorphous urates of sodium and potassium; *b*, "Hedgehog" crystals of sodium urate; *c*, Ammonium urate.

Amorphous deposits of urate of potash or ammonia, and phosphates and carbonates of the alkaline earths may be met with in urines of either reaction.

1. Urates (*i.e.*, Lithates), chiefly of sodium, potassium, or ammonium, when in excess are deposited as an amorphous deposit after the urine (warm when first passed) has become cold. A deposit having these characters, and disappearing when heated in a test-tube, is sufficiently characteristic for the detection of lithates. The deposit is dissolved on the addition of caustic potash; a test which also distinguishes urates from phosphates. *Urates of Soda and Potash*, under the microscope, appear as amorphous orange of *pale brown* granules (Fig. 83, *a*). *Urate of Soda* may occasionally appear as "hedgehog" crystals, globular masses covered with spikes (Fig. 83, *b*). *Urate of Ammonium* occurs as globular masses, sometimes spiked, very like sodium urate, but known from such by being found in alkaline urines (together with phosphates) and by being dissolved by acids (Fig. 83, *c*).

Clinically, urates and uric acid are important only when they occur *constantly*, in fresh urine, or in urine that has stood a few hours only. Gouty and other symptoms are apt to arise in such cases (see Lithæmia, § 249), and calculus might

be expected to form in the bladder or kidney. An occasional deposit of urates, or a deposit occurring in urine that has stood over six hours, is of but little importance. In all concentrated urines, on cooling, large deposits of urates normally occur.

2. Free Uric Acid is deposited when the urine is very acid or poor in salts and in pigment, and is therefore found chiefly in dilute pale urines with deficiency of salts. The red deposit of uric acid closely resembles cayenne pepper to the naked eye, and by patients is sometimes mistaken for blood, but the difference is at once apparent under the microscope. It may be detected in the urinary deposit under the microscope by the *colour* and shape of the crystals. It occurs in the form of *red-brown crystals* (the only coloured crystals commonly found in the urine), mostly lozenge-shaped (Fig. 84). Uric acid assumes many different shapes, owing to the presence of the colloid substances in the urine, but they are all derivatives from the rhombic prism or parallelogram, in which form uric acid crystallises from pure water. The more pigment, mucus, and other colloids there are in the urine the more spherical do the crystals become. Some of these are shown in the accompanying illustration, and the gradual transition from rhombic prism to dumb-bell and other spherical forms will be seen by following the crystals from left to right. This deposit is soluble in caustic potash, insoluble in dilute acetic acid, the converse of phosphates.

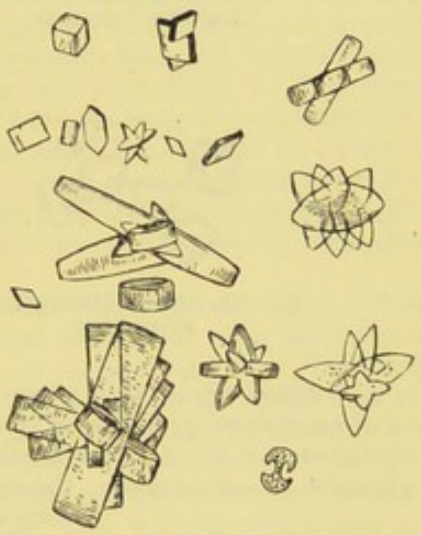

Fig. 84.—Uric acid crystals (red-brown).—The two top rows show, from left to right, the evolution in a colloid medium of the "lozenge-shaped" crystal from the primary rhombic prism. In the lower right-hand corner is the "dumb-bell" form occasionally met with.

In health uric acid is increased with a highly nitrogenous diet, after much exercise, after meals, and during the "alkaline tide" of the morning. It is also increased in most fevers, in splenic diseases, pernicious anæmia, in some cases of dyspepsia, during and after an acute attack of gout, and during an attack of acute rheumatism. It is diminished in chronic gout, especially just before the acute exacerbations; in chronic Bright's disease; in chlorosis and other chronic diseases.

3. Phosphates occur as a white deposit or flocculent turbidity in feebly acid, neutral, or alkaline urine, in three different forms, which in order of frequency are: (1) *Amorphous phosphates of calcium* form the thick white deposit that is apt to be mistaken for pus, but which is more readily shaken up in the urine. These and all other phosphates are soluble in acetic acid, and precipitated by ammonia. The latter test decomposes neutral phosphates. (2) *Triple phosphate* of ammonium and magnesium (Fig. 85), is found in urine which has undergone alkaline fermentation. The crystals are large colourless three-sided prisms like "house-tops," occurringly singly, or as snow-flakes or other irregular forms. In markedly ammoniacal urine "feathery phosphates" are found. (3) *Basic magnesium phosphate* occurs in large rhombic plates, not grouped, but scattered (Fig. 86). (4) *Neutral or dicalcium phosphate* occurs in neutral or alkaline urines as clear, refractile, pointed or wedge-shaped prisms arranged in stellate groups—"stellar phosphates" (Fig. 87). The constant presence of phosphatic deposits may be associated with symptoms (§ 314), or suggest the presence of a stone. *Monocalcium phosphate* occurs chiefly in acid urines.

4. Oxalates are chiefly met with as *oxalate of calcium*. This occurs as a scanty crystalline deposit of colourless transparent octohedra, appearing, under the microscope, like tiny envelopes, hence the name "envelope crystals" (Fig. 88). They sometimes rest like fine powder above a cloud of mucus, and have been described therefore as the "powdered wig" deposit. They are soluble in hydro-

chloric acid, insoluble in acetic acid or caustic potash. Oxalate of calcium may also occasionally appear as dumb-bell shaped crystals. The presence of crystals of oxalate of calcium in the urine is indicative of an excess (Oxaluria, § 314); their presence may also suggest the nature of a calculus.

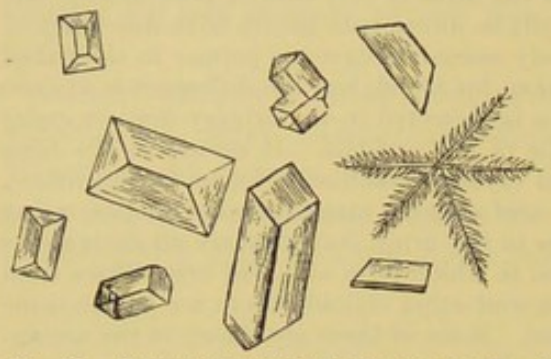

Fig. 85.—Triple phosphate—"house-top" and "feathery" crystals.

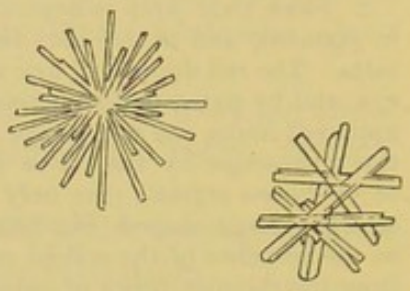

Fig. 87.—Neutral or "stellar" phosphate.

Fig. 86.—Basic Magnesium phosphate.

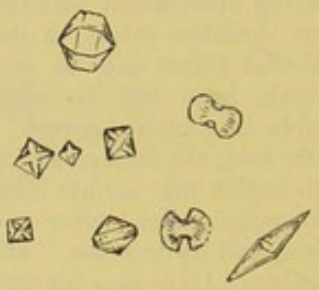

Fig. 88.—Calcium oxalate—"envelope" and "dumb-bell" crystals.

5. *Calcium Carbonate* is a rare deposit, consisting of tiny spheres and dumb-bells, or of amorphous granules, effervescing and dissolving in acetic acid (Fig. 89). The *Carbonates of the Alkaline Earths* are very occasionally found as tiny amorphous granules or concretions. Calcium sulphate and carbonate may take part in the formation of vesical calculi, especially in the aged, but otherwise they are of no clinical significance. Their presence only points to the existence of a calculus, and indicates its composition.

Fig. 89.—Calcium Carbonate.

When a patient is taking crystalline drugs, such as acetate of potash and phosphate of soda, or even liquor ammoniæ, various crystals which have no pathological significance sometimes appear in the urine. Moreover, after a reagent has been added to urine (*e.g.*, Esbach's solution for the estimation of albumen), and it has been set aside, crystals may also appear which have no clinical value.

6. *Certain rare and less important deposits*, which occur chiefly in acid urines, are as follows: *Hippuric Acid* is an antecedent of uric acid in the nitrogenous metamorphoses of the tissues. It occurs as four-sided prisms, either scattered or in groups. It is present after the ingestion of benzoic acid in large doses, cranberries, and other fruits. *Calcium Sulphate* occurs either as amorphous granules, or, very rarely, as long colourless needles or elongated tables with truncated ends. It is detected by being insoluble in ammonia and acids. *Leucin* occurs as laminated spheroids, and *Tyrosin* as bundles of acicular crystals (Fig. 73). Both occur in the urine in phosphorus poisoning and acute yellow atrophy of the liver. *Cholesterin* (Fig. 72) is only occasionally found among urinary deposits. It forms laminated plates with longitudinal striæ, and a notch at one end. *Cystin* occurs as hexagonal plates soluble in ammonia (Fig. 73, p. 362).

PHYSICAL EXAMINATION OF THE KIDNEYS.

§ **292.** A dull "sickening pain" is usually felt on firmly compressing the kidney with both hands, but there is no tenderness in a healthy organ. Tenderness may be elicited in cases of calculous and other causes of pyelitis, perinephric inflammation, abscess, or tumour of the organ. Kidney tumours tend to grow forwards, where there is least resistance, pushing the resonant colon *in front* of them. When, therefore, the palpating hand encounters resistance and swelling in the lumbar region *posteriorly*, it is probably due to a peri- or extra-renal, rather than to a renal condition (see Fig. 37, p. 135). The diagnosis of renal swellings from other abdominal tumours has been given in § 188.

In the majority of renal disorders the physical examination of the kidneys is of secondary importance to the examination of the urine. The kidneys are situated on either side of the spine, about 3 inches from the middle line; the right is slightly lower than the left, owing to the position of the liver just above it. The upper end of the right kidney reaches to the *lower* edge of the eleventh rib; the left kidney reaches as high as the *upper* edge of the eleventh rib. The kidneys lie partly in the hypochondriac and partly in the lumbar regions, and are therefore much higher than is commonly supposed, with reference to the anterior abdominal wall. The lower end of the right kidney is 1 inch and that of the left kidney 1½ inch *above* the level of the umbilicus.

Palpation.—Even in normal conditions the lower border of the right kidney may be palpable in thin people. In those whose abdominal walls are lax—in women who have borne children, for instance—is is surprising how frequently the right kidney can be palpated. The patient should lie on the back, with the abdominal muscles relaxed. The physician, standing on the right of the patient, should place his left hand beneath the patient's back, close under the ribs, just external to the quadratus lumborum. The right hand is laid flat over the anterior surface of the abdomen, in the mid-clavicular line, with the fingers pointing upwards, just below the liver. Pressure backwards, as if to meet the left hand, is made by the right hand. The patient should then be asked to draw a deep breath, and as he does so the rounded lower edge of the kidney is felt to slip between the opposing hands. When the ligaments of the kidney are relaxed—*movable kidney*—the fingers of the right hand may be able to palpate the upper border of the organ, and to retain it during expiration. A kidney is said to be "*floating*" when it cannot only be readily palpated, but can be pushed below the umbilicus or freely moved about in the abdominal cavity.

Percussion does not enable us to define the margins of the kidney, for the organ is too deeply seated. The feature of primary importance in this connection is its relation to the colon, which, as just mentioned, is pushed forward by enlargements or tumours of the kidney. Consequently the anterior surface of such growths is always resonant, there being dulness at the side which is continuous with that at the back; whereas with enlargements of the spleen or gall-bladder there is dulness anteriorly, and resonance at the side.

Other methods of examination of the kidneys are now open to us. In cases of doubtful renal calculus a **radiogram** will usually settle the diagnosis. Examination of the ureteral orifices by means of a **cystoscope** may demonstrate which kidney is affected, for the normal flow of urine may be absent or visibly altered, and the orifice itself may be the seat of infiltration or ulceration. The previous administration of methylene blue or other harmless pigment may make the differences of the flow from the orifices more obvious (**chromo-cystoscopy**). Lastly, the ureters may be **catheterised**, and a specimen of urine obtained in this way from each kidney.

PART C. URINARY DISORDERS, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **293. Routine Procedure and Classification.**—*First*, having ascertained that the patient's LEADING SYMPTOM refers to the urinary apparatus; and, *secondly*, the data of his ILLNESS, particularly as to whether it is of an ACUTE or CHRONIC nature; we proceed, *thirdly*, to examine the urine. The ROUTINE EXAMINATION of the URINE in every-day practice consists of Inspection, Reaction, Specific Gravity, Tests for Albumen and for Sugar. The subsequent and more detailed examination depends upon circumstances. As above stated, the examination of the urine stands in relation to renal disease, as the local signs do to diseases of other organs. There are very few diseases, certainly no common disorders of the kidneys, which are not attended by some change in the urine. On the other hand, the LOCAL EXAMINATION of the kidney, by palpation and percussion (§ 292), is difficult and relatively much less certain and instructive. On this account it comes last in our scheme of examination, but it should never be omitted in any case which is at all obscure.

Classification.—We will deal with urinary disorders under their respective cardinal symptoms as follows:

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§ **294. Albuminuria.**—The numerous morbid conditions which may give rise to albuminuria may be divided into three great anatomical (and clinical) groups: A. *Acute Inflammation* of the Epithelium (Acute Nephritis or Acute Bright's Disease); B. *Chronic Inflammations* and *Degenerations;* C. *Renal Congestions*, either active or passive, which include many cases of albuminuria, independent of structural disease of the kidney.

If, therefore, the illness came on recently, and is of an **Acute** character, turn first to § 295 (Acute Nephritis), and then to § 299 (Renal Congestions).

If, on the other hand, the illness is of some duration, and evidently of a **Chronic** kind, turn first to § 296 (Chronic Tubal Nephritis), and then the succeeding sections.

When the albumen is in small quantity, and there is also BLOOD or PUS in the urine turn to § 300 (Hæmaturia), or § 303 (Pyuria), respectively.

The illness came on recently, and is **acute** ; *the urine is diminished, and contains a considerable quantity of* ALBUMEN *and* TUBE-CASTS ; *it is or has been* " SMOKY " *from the presence of blood ;* ANASARCA *is present ; and there is a tendency to uræmia.* The disease is ACUTE NEPHRITIS.

§ **295. Acute Nephritis** (Acute Bright's Disease).—In this disease the inflammation begins and predominates in the epithelium or parenchyma of the organ. The condition usually lasts from five to six weeks, and may terminate in recovery or pass on to a chronic condition.

Symptoms.—(1) The albumen is often in considerable quantity, and the urine may even go solid on boiling. (2) The other characters of the urine are : (i.) It is scanty, sometimes only 10 or 20 ounces a day, or less. Consequently, the specific gravity is high, although the diurnal quantity of urea is diminished. (ii.) It is turbid or " smoky " from the presence of blood in the early stages. (iii.) Epithelial, hyaline, and blood casts, free renal epithelium, and red blood-corpuscles are present. (3) Dropsy is general from the commencement, although it is first noticed in the face in the loose areolar tissue below the eyes and in the genitals. There may also be collections of dropsical fluid in the serous cavities. (4) There is a waxy pallor of the skin. (5) A degree of malaise, with discomfort and even pain in the loins, may be present, but there is only a slight elevation of temperature for about four or five days. (6) Uræmic symptoms may come on early—*e.g.*, (i.) occasional vomiting, (ii.) headache, (iii.) drowsiness. (7) If the disease goes on for any time the pulse becomes of high tension, and the second aortic sound is accentuated.

Causes.—Acute nephritis is only very rarely a primary malady. (1) Ninety per cent. of the cases supervene on an acute specific fever, and by far the most common of these is scarlet fever. (2) Chill, especially a sudden chill when the skin is perspiring. (3) Traumatism—*i.e.*, a blow on the kidney is an occasional cause. (4) The persistent use of certain drugs, such as cantharides and turpentine. (5) Inflammation secondary to disease of the urinary tract below the kidney (see Ascending Pyelo-nephritis). (6) Pregnancy is a marked predisposing, and sometimes exciting, cause.

Prognosis.—Acute Nephritis may terminate in (1) complete recovery, the usual result when the treatment and hygienic surroundings are good. (2) Partial recovery. If the disease lasts longer than three months it usually develops into the condition known as large white kidney (Chronic Parenchymatous Nephritis, § 296). (3) Death may occur from uræmia, from dropsy into the serous cavities, or from other complications. The chief *complications* are : (*a*) Uræmia ; (*b*) inflammations of the *serous* membranes, such as pleurisy, pericarditis, or peritonitis, which are usually latent—*i.e.*, attended by little or no pain ; and (*c*) inflammations of the *mucous* membranes, such as bronchitis, gastritis, enteritis (causing diarrhœa) ; (*d*) œdema of the lungs or of the glottis ; (*e*) cardiac dilatation and failure ; (*f*) erysipelas, cellulitis, and

various other *skin diseases* are very prone to attack patients with acute nephritis.

The prognosis, therefore, of acute nephritis is grave in proportion to (i.) the diminution of urine ; (ii.) the development of uræmic symptoms ; (iii.) the amount of dropsy present ; and (iv.) the nature and severity of the complications.

Treatment.—The indications are to relieve the kidney by giving only bland non-irritating food—*e.g.*, milk ; to increase the action of the skin and bowels ; and to lessen local congestion. (1) To obviate the great liability there is to chill, the patient should be kept in bed ; and for the same reason all cases of scarlet fever should be kept in bed during convalescence, because they are so apt to develop this disease. (2) Diaphoretics, such as liquor ammoniæ acetatis, antimonium tartrate, jaborandi, or pilocarpine, warm baths, wet packs, and hot-air baths. This treatment may be applied by means of a wicker cage placed upon the bed, and connected with a spirit-lamp through an iron chimney at the foot. (3) Purgatives, such as pulv. jalapæ co. (30 grs. to ʒi.), are indicated. Saline purgatives are especially useful when there is much dropsy. (4) There is some difference of opinion about diuretics ; some say that they irritate the kidney, others that they relieve the symptoms, and especially the dropsy. It is agreed that during the acute stage copious libations of water, but no other diuretic, should be used. Saline diuretics employed are potassium bicarbonate, citrate, acetate, and bitartrate. Scoparium and digitalis are given with caution if the heart is feeble. (5) Local depletion by wet or dry cupping is especially indicated when the urine contains much blood. Counter-irritation over the kidneys, with poultices or leeches, has a similar effect. (6) During *convalescence* tonics, especially iron, must be given. An admirable prescription is liq. ferri perchloridi, ℥ 15 ; liq. ammoniæ acetatis, ʒi. ; acidi acetici, ℥ 5 (to prevent decomposition). Animal food should be forbidden so long as albuminuria continues. In the treatment of renal disease three drugs are contra-indicated—opium, cantharides, and turpentine. Mercury is generally added to these, but I have never seen any harm arise from its administration. For the treatment of Uræmia, see § 297.

Chronic Albuminuria.—There are three anatomical varieties of chronic renal disease attended with more or less albuminuria, which, when occurring in their typical forms, present well-marked clinical distinctions, as shown in a tabular form below. In Chronic Tubal Nephritis (including large white kidney), the renal *epithelium* is primarily and throughout the disease chiefly involved. In Chronic Interstitial Nephritis (Gouty Kidney) the *interstitial tissue* shows evidence of increase, and throughout the disease this is the most marked change ; the arteries also, however, show hyperplasia of their middle coat.[1]

[1] *Cf.* Introduction to this chapter.

This arterial change also occurs throughout the body, and is attended by a corresponding hypertrophy of the left ventricle. In the Amyloid (or Waxy) Kidney the *vessels* are primarily involved, the lardaceous degeneration beginning in the middle coat. Pathologists make many sub-divisions, but these represent the three clinically recognisable groups of chronic renal changes attended by albuminuria.

TABLE XX.—DR. MURCHISON'S TABLE OF CHRONIC ALBUMINURIA.

	Quantity of Albumen.	*Tendency to Uræmia.*	*Quantity of Urine.*	*Tendency to Dropsy.*
Chronic Tubal Nephritis.	Large.	Moderate.	Diminished or normal.	Great.
Chronic Interstitial Nephritis.	Very small.	Great.	Increased.	Very slight.
Waxy Kidney.	Very great.	Slight.	Greatly increased.	Slight.

If the albumen is CONSIDERABLE, turn to Chronic Tubal Nephritis, Amyloid Kidney, or Chronic Renal Congestions. If there is only a TRACE of albumen, and the urinary signs appear to be slight in proportion to the debility and other symptoms, turn to Chronic Interstitial Nephritis, § 297.

The illness is **chronic,** *and the general symptoms of renal disease pronounced; generalised* DROPSY *is marked; the urine is scanty, and* ALBUMEN *and* CASTS *are abundant.* The disease is CHRONIC PARENCHYMATOUS NEPHRITIS.

§ **296. Chronic Tubal Nephritis** (Synonyms: Large White or Pale Kidney, Chronic Parenchymatous Nephritis, Chronic Desquamative or Catarrhal Nephritis, Fatty Kidney) may follow on acute nephritis, or may develop insidiously. In the latter stages the connective tissue is increased, and if the patient lives long enough the kidney becomes a *Contracted Fatty Kidney*, or *Small White Kidney.*

Symptoms.—(1) The albuminuria is considerable, $\frac{1}{4}$ to $\frac{1}{3}$ on standing; (2) the other characters of the urine are: (i.) the diurnal quantity is slightly diminished at first, but towards the end, when the kidney contracts, the quantity may be greater than normal; (ii.) the specific gravity is not much altered in the early stages, but the urea is deficient throughout; (iii.) it is turbid, often with lithates; and recurrent hæmaturia may occur, especially if the condition has followed acute nephritis; (iv.) all forms of casts are met with (§ 290). (3) There is generalised dropsy, but most marked in the face. It may disappear towards the end, when the diurnal quantity of urine increases. (4) There are pallor, emaciation, weakness, and digestive disorder; and (5) cardio-vascular symptoms (§ 267) ensue.

Etiology.—(1) Chronic tubal nephritis frequently follows acute nephritis, or (2) it may result from prolonged mechanical congestion

of the kidney (as in cardiac disease). (3) Sometimes it comes on insidiously, without apparent cause. (4) Alcohol in excess predisposes.

Diagnosis.—When the insidious form occurs in young women it is often mistaken for *anæmia*. In all cases of anæmia resisting iron, examine the urine for albumen. In the later stages it may be mistaken for *chronic interstitial nephritis;* but in that disease the patient is usually older, and see Table XX. In certain cases which present *both renal and cardiac* symptoms it may be very difficult to say *which condition is the primary* one. In such cases it is important to note the following points : (i.) If there is a *history* of rheumatic fever and previous attacks of dropsy, it is probable that the cardiac condition is primary. (ii.) If *other than mitral* systolic murmurs are present it points to cardiac disease ; a mitral regurgitation murmur *alone* might be due to the cardiac failure following renal disease. (iii.) The *urine*, when there is any difficulty in diagnosis, is in both cases scanty and albuminous. Many tube-casts point to renal disease ; the rapid clearing up of the urine after a short period of rest in bed points to heart disease. (iv.) A *hard pulse* favours kidney disease, but an irregular soft pulse is found with cardiac failure secondary both to renal and to cardiac disease.

Prognosis.—When once established the disease can never be cured, and even with careful diet and treatment the patient rarely lives more than a few (two to five) years. Death occurs as a consequence of dropsy, uræmia, or complications (as in acute nephritis). The prognosis is grave in proportion to (1) the amount of dropsy and albuminuria ; (2) the diminution of urine ; and (3) the presence of uræmic symptoms. If the patient survive for several years the prognosis improves ; because, when the stage of contraction sets in life may, with care, be somewhat prolonged.

The *Treatment* is much the same as that of acute nephritis (*q.v.*) ; but two points demand constant attention : (1) The avoidance of chill, by the wearing of flannel and resort to equable climates ; (2) careful dieting, with the object of reducing the nitrogenous intake to a minimum. The latter is best accomplished by making the patient live entirely on milk, 2 to 4 pints per diem, and as much fluid as he can drink. If meat be taken, it should not exceed 2 or 3 ounces once a day. Stimulants, meat extracts, and animal soups should be avoided, unless evidences of a fatty heart are present. Purgatives should be administered so that the bowels act twice a day. Tonics, and especially iron, are the best drugs. The best tonic is that prescribed for convalescent acute nephritis (*q.v.*) ; it may be combined with a diuretic.

The patient complains of lassitude, and other symptoms of INCIPIENT URÆMIA *mentioned in* § 270. *There are only* TRACES OF ALBUMEN, *the diurnal quantity of urine is increased, dropsy is absent.* The disease is probably CHRONIC INTERSTITIAL NEPHRITIS.

§ 297. **Chronic Interstitial Nephritis** (Synonyms : Contracted, Granular, or Gouty Kidney ; Cirrhotic Kidney ; Renal Fibrosis ; Small Red Kidney ; Chronic Non-Desquamative Nephritis).—It is accompanied by widespread cardio-vascular changes, as mentioned in the introduction to this chapter, consequent on recurrent high blood pressure.

Symptoms.—(1) The albuminuria in this disease is small in amount, and many samples of the urine may be examined without finding any. In cold weather, however, when there is deficient skin action, there is generally a trace, especially after a chill or any cause which produces renal congestion. The other characters of the urine are : (ii.) The diurnal quantity is greatly increased (maybe to 100 ounces). The patient often consults us because he has to get up at night several times to pass water. (iii.) The specific gravity is very low (1005 to 1012), owing partly to the deficiency in urea, but chiefly to the increased quantity of urine. The deficiency in total urea is not very great, and may not be sufficient to suggest the onset of uræmia ; the total amount of salts in the urine is diminished, and affords a more reliable indication (see § 285). (iv.) The urine is clear, pale, and contains but few casts, and these are chiefly hyaline or granular (Fig. 79). (2) Dropsy is usually absent. If dropsy occur it is due to (i.) secondary cardiac failure, or (ii.) the supervention of acute nephritis. Sir George Johnson found a history of dropsy in only fourteen out of thirty-three cases. (3) The patient may look robust, but sometimes he has a greyish pallor. (4) The pulse indicates persistent high tension, and is often associated with hypertrophy of the left ventricle, an accentuated aortic second sound, sometimes with a systolic apical murmur, and always sooner or later with a thickened condition of all the arteries. Later the heart may dilate, with consequent dropsy and albuminuria, and it may be hard to diagnose whether the kidney or the heart condition is primary or secondary (§ 296, *Diagnosis*). (5) There is throughout a condition of chronic or incipient uræmia (§ 270), due to the deficient nitrogenous metamorphosis in the body, and the retention in the blood and tissues of the antecedents of urea, owing to deficient renal function. These symptoms are indefinite, but in order of importance they are : (i.) Insomnia and *headache*, symptoms which, occurring in the aged, should always lead us to suspect granular kidney ; (ii.) gradual impairment of the mental and bodily vigour ; (iii.) tremors and twitching of the muscles; (iv.) digestive disorders; (v.) dyspnœa, often paroxysmal.

Course and Complications.—Apart from the existence of slight and intermittent albuminuria and persistent *high arterial tension*, non-urinary symptoms are the earliest, and often for prolonged periods the only evidences of this disease. In many cases the high arterial tension first reveals the disease to the physician ; in other cases it is the *ophthalmoscopic changes* (Renal Retinitis, § 267), changes which may or may not be attended by failure of vision. Apart from the progressive

enfeeblement, the disease generally first manifests itself by the occurrence of one of its numerous complications. The most frequent and most serious of these is *cerebral hæmorrhage*, resulting from the prolonged high tension and consequent arterial degeneration. *Hæmorrhages* of various kinds may occur in other directions, such as epistaxis, or melæna. Epistaxis constitutes a kind of safety-valve, relieving the vascular system from more serious internal hæmorrhages, consequently it should not be checked. The *mucous membranes* are often affected, and intractable bronchitis or gastro-enteritis in an elderly person may be the condition which brings the patient under our notice ; the *serous membranes* less often, though a latent form of pleurisy or pericarditis is not uncommon. *Skin diseases* are often very troublesome. The earliest symptom noticed in many cases is the itching of the skin: Urticaria, eczema, erythematous, desquamative, and hæmorrhagic eruptions are apt to occur. The patient is liable, on exposure to cold, to attacks of *congestion of the kidney*, when the albuminuria and all the other symptoms are aggravated.

The *Diagnosis* from other forms of chronic renal disease is given in Table XX., p. 417. However, the diagnosis of this form of chronic renal disease from the other conditions which give rise to lassitude and DEBILITY is often a question of much greater difficulty (Chapter XVI.).

Etiology.—(i.) I have assisted at an autopsy in a well-marked case of granular kidney in a child of nine[1]; but the disease almost invariably occurs in *persons of middle age or advanced life.* Out of 376 cases admitted into the Paddington Infirmary 317 were over forty years of age, 251 were over fifty, and 203 over sixty. (ii.) *Gout* and a gouty habit of body is the most important causal factor. In many cases there is a long history of persistent lithuria, and in a large proportion of cases of granular kidney the joints and ears show evidences of gouty deposit ; hence the name "gouty kidney." (iii.) An *indolent* life, and (iv.) *Chronic lead poisoning* are undoubtedly causes, not only of gout, but of granular kidney. (v.) Various other forms of *toxæmia which produce recurrent or constant high blood-pressure* (*q.v.*) may also be followed by chronic interstitial nephritis. Many of the symptoms usually attributed to chronic interstitial nephritis (renal fibrosis) are really those of high blood-pressure, and in the author's opinion[2] renal fibrosis is an accidental occurrence in certain cases of toxæmic high tension—not in all.

Prognosis.—The course of the disease, as already mentioned, is prolonged. With care and attention to diet the patient may live for five, ten, or more years, but the disease can never be cured. The amount of albumen is no criterion as regards prognosis in chronic interstitial, as

[1] In childhood the leading symptoms are general pigmentation, polyuria, and headache. The first is explained by the possible invasion of the adrenals. The headache is often worse on rising in the morning—*i.e.*, like a high tension headache.

[2] "On Senile Epilepsy," the *Lancet*, July, 1909 ; and elsewhere. See also p. 389.

it is in chronic parenchymatous, nephritis. The prognosis is grave in proportion (1) to the duration of the disease; (2) to the evidences of uræmia present and their degree; (3) the degree of cardiac failure; and (4) the presence and severity of the complications (*vide supra*). Life is frequently terminated by cerebral hæmorrhage or some other complication; a large number of these cases die of acute uræmia (§ 270), as the records of the Paddington Infirmary show. Older authors described this as death by "serous apoplexy," thinking that the serum which replaced the atrophy of the brain was the cause of pressure upon that organ.

Treatment.—Diet is of chief importance. The amount of proteid should be reduced to an absolute minimum, and alcohol forbidden. All chances of chill should be avoided by clothing in flannel, and living in equable climates; and the action of the skin should be maintained. The arterial tension should be reduced (§ 61); this takes the strain off the heart, and will cure symptoms such as headache and insomnia due to high pressure. Purgatives, such as mistura alba, will do a great deal for this, at the same time enabling the bowels to drain off the poisonous substances which ought to be eliminated by the kidneys. Tonics are useful, such as nux vomica, and digitalis for heart failure. The action of the latter must be carefully watched (special heed being given to the pulse), lest it produce apoplexy or other hæmorrhages. Iron is not of much use, and may do harm by leading to constipation. *Symptomatic Treatment.*—(i.) Liquor trinitrini, ℥ i., b.d. will cure headache when due to high tension. (ii.) Potassium iodide relieves tension, and may prevent further arterio-sclerosis. (iii.) For the attacks of "renal asthma" amyl nitrite, chloroform, or venesection may be necessary. (iv.) For restlessness, chloral and bromides are useful.

The treatment very often resolves itself into *the treatment of uræmia*, which is as follows, the indications being (1) to eliminate the poison as rapidly as possible; and (2) to alleviate the symptoms. In *chronic* uræmia a daily dose of Epsom salts may be taken; the skin must be encouraged to act; diuretics such as pot. bicarb. and acet., sp. æth. nit. and scoparium administered; and digitalis if the heart is dilated. Large quantities of water should be taken. To relieve tension and headache nitroglycerine is valuable (see also § 61).

For *acute* uræmia—muttering delirium, convulsions, coma (diagnosis of uræmic coma, § 530)—a brisk hydragogue purgative must be given at once, such as pulv. elat. co., pulv. jalapæ co., or a concentrated solution of magnesium sulphate. The skin must be made to act by means of hot packs, hot air or vapour baths, or pilocarpine ($\frac{1}{8}$ to $\frac{1}{4}$ grain hypodermically). Venesection (10 to 20 ounces) did a great deal of good in many of my infirmary cases, and undoubtedly averted a fatal issue. Transfusion of normal saline solution (0·75 per cent. NaCl) compensates for the loss of fluid by bleeding or purgation, and may with advantage be adopted after venesection. Chloroform relieves the convulsions.

There is **abundant albumen** *with the passage of* LARGE QUANTITIES *of urine, but little tendency to dropsy and uræmia; the patient has a history of prolonged* SUPPURATION, *or of* SYPHILIS; *and there may be evidences of lardaceous disease elsewhere.* The disease is LARDACEOUS KIDNEY.

§ **298. Amyloid Kidney** (Waxy or Lardaceous Kidney) is generally part of a widespread lardaceous disease involving the liver (enlargement), spleen (enlargement), and intestines (diarrhœa). With more efficient modern surgical methods amyloid degeneration is becoming a rare condition.

Symptoms.—(1) The albumen, though it may be small in quantity in the early stage, is very abundant, amounting to three-fourths or more when the condition is established. The other characters of the urine are: (i.) The diurnal quantity is greatly increased, even to 150 ounces; (ii.) the specific gravity is very low, but the urea is not diminished till the later stages; (iii.) the colour is pale and clear; (iv.) all varieties of casts may be found, including amyloid and fatty casts. (2) There is great pallor of the surface and anæmia, but there may be no dropsy, till quite the end of the disease. In cases with great cachexia dropsy *may* occur early. (3) Evidence of lardaceous disease in other organs is present—liver, spleen and intestines, consequently hæmorrhages may occur from different parts. The amyloid disease of the bowel gives rise to very intractable diarrhœa, a symptom which often accompanies amyloid kidney.

It is important for the *diagnosis* to ascertain the history of a *cause*—namely, (*a*) prolonged suppuration, either from a chronic abscess, chronic phthisis, or caries. Dr. Murchison used to be of the opinion that caries of the vertebræ, even without definite formation of an abscess, could give rise to lardaceous disease of the viscera, especially the kidney. (*b*) Syphilis is the second of the two great causes which bring about lardaceous disease.

Prognosis.—The course of the disease is protracted. The patient may live for several years, dying by exhaustion from diarrhœa, or other complications; very rarely from uræmia due to the supervention of acute nephritis. With careful treatment patients may live for many years, or even recover if the disease is seen in a very early stage; but the prognosis is bad in proportion to (1) the amount of albuminuria, and (2) the extent of the involvement of the other organs.

Treatment.—Alkalies have been reputed not only to prevent, but also to improve, the lardaceous process—*e.g.*, liquor potassæ (5 minims); the tartrates and citrates of the alkalies are also administered. Iodine, especially in the form of iodide of potassium or iodide of iron, should be given, particularly in syphilitic cases. The most troublesome complication is diarrhœa. The only remedies which in my experience are of any use are liquor ferri pernitratis (15 minims); or pil. plumbi cum opio (5 grains) continued every four hours until the diarrhœa ceases. Opium may be administered in this form of renal disease when there is no tendency to uræmia. The *preventive treatment* of lardaceous disease consists in the adequate treatment of syphilis in its early stages; and in curing prolonged suppuration, especially when this occurs with chronic profusely discharging ulcers of the leg.

§ **299.** In **Renal Congestion** (Secondary Albuminuria) there is sometimes a very considerable amount of albumen in the urine; but the urinary and other symptoms do not conform to the foregoing types. Casts are generally absent, never abundant, and the constitutional disturbance, apart from the *primary* malady, is slight.

If the albuminuria is MARKED *and* CONSTANT, *and especially if the urinary symptoms are associated with symptoms referable to some other organ, it is probably* PASSIVE *renal congestion due to* I. CARDIAC DISEASE; II. ASCITES OR ABDOMINAL TUMOURS; OR III. PREGNANCY (?)

If the albumen is SLIGHT *in amount, and especially if it be* TRANSIENT, *it is probably* ACTIVE *renal congestion due to* IV. CHILL TO THE SURFACE;

V. Toxic Blood States, with or without Pyrexia; VI. Drugs; VII. Dyspepsia or Hepatic Derangement; VIII. Deranged Innervation; or IX. Functional Albuminuria.

I. Cardiac Disease (the Cardiac Kidney[1]) is the most frequent of the congestive causes of albuminuria. Albuminuria is a very common accompaniment of mitral valvular disease, and of the dilatation of the right heart which so frequently follows chronic bronchitis and emphysema. At first the kidney is only congested, but later the epithelium may become affected and the interstitial tissue increased. The diagnostic features of the albuminuria in such cases are: 1. The amount of the albumen is always considerable, and may be very great. 2. The urine is scanty, high-coloured, of high specific gravity, and there may be blood-cells, renal-cells, or even casts; nevertheless these latter may disappear when the heart is relieved. 3. There are evidences of the cardiac condition which has produced the renal disease. In some cases it is difficult to decide which of these was primary (§ 296, *Diagnosis*).

Cardiac disease may give rise to renal disease in three ways: (i.) In the manner just stated. (ii.) *Embolism of the kidney* is one of the consequences of endocarditis (acute or chronic). In this condition the albuminuria appears suddenly with hæmaturia and constitutional symptoms, and disappears equally suddenly in a few days. (iii.) Some causes of *aortic valvular disease* (the mitral being healthy) have been attended by temporary albuminuria. The explanation is not obvious. In these cases the compensatory hypertrophy and dilatation were great, and the arterial tension high; and it seems probable therefore that the albuminuria may have been due to an *active* renal congestion.

II. Ascites and Abdominal Tumours.—Here the albuminuria is due to pressure on the renal veins. This condition is recognised by: (1) The amount of albumen is generally moderate; (2) there is abdominal enlargement with the signs of fluid or tumour; (3) the albuminuria will disappear on removing the cause. There are two fallacies to be remembered before diagnosing albuminuria as due to ascites: (i.) Both albuminuria and ascites may be the product of some common cause—*e.g.*, heart disease; and (ii.) the ascites may be the result of a general dropsy due to renal disease.

III. Pregnancy is an undoubted cause of albuminuria, and according to Playfair it occurs in 20 per cent. of parturient women after the third month. It also seems certain that permanent and ineradicable renal disease may, in some cases, date from pregnancy. According to some, the albuminuria of pregnancy is due to pressure on the renal veins—a view that is supported by its more frequent occurrence in primiparæ, in whom the abdominal walls are more rigid. But, on the other hand, the albuminuria may occur before the uterus is large enough to cause pressure on the renal veins. These and other considerations

[1] It is well to bear in mind that when both cardiac and renal disease are present, they may be associated in three ways: (*a*) Cardiac disease may produce renal disease in one of the above-mentioned ways. (*b*) Renal disease may produce cardiac disease, as when acute nephritis or granular kidney lead to cardiac hypertrophy and failure. (*c*) They may both be the result of a common cause—*e.g.*, gout.

point to the conclusion that it is probably due to some blood change associated with the parturient state. The clinical features are: (1) The amount of albumen is not usually great, and the urine is otherwise normal or very much as in cardiac cases. (2) Ophthalmoscopic changes (§ 267) may be present; but (3) these and the urinary symptoms disappear within two or three weeks of labour unless permanent renal disease has been induced. The treatment is discussed below.

The remaining causes of albuminuria are probably due to ACTIVE congestion of the kidney.

IV. CHILL TO THE SURFACE.—Chill to the surface may result in albuminuria, but in such cases the kidney is rarely quite healthy. This condition is recognised by: (1) The amount of albuminuria is never very great, and it does not last for more than a few days; (2) the urine is otherwise normal, or may deposit lithates; (3) the patient, in other respects, is healthy, or complains only of slight bronchial catarrh or coryza.

V. TOXIC BLOOD STATES with or without PYREXIA.—This cause of albuminuria, is characterised by: (1) Tube-casts are absent unless there be active renal disease. (2) An elevated temperature; in hyperpyrexia albuminuria is invariably present. (3) Other evidences of the toxic blood state, namely: (i.) Various acute specific fevers—*e.g.*, diphtheria, where albuminuria may be present without high temperature. In scarlet fever albuminuria frequently comes on between the sixteenth and twenty-sixth day, at which time also acute nephritis may supervene, and, to avoid this risk, scarlet fever patients should be kept in bed three or four weeks. Transient albuminuria may occur in secondary syphilis, between the sixth and eighth weeks of the disease. If albuminuria occurs in the later stages of the disease, it may be due to lardaceous disease or gumma of the kidney. (ii.) Acute pneumonia is sometimes, and (iii.) acute gout is very frequently, accompanied by albuminuria. (iv.) Albuminuria may also occur in diabetes (in which it is a grave sign), in pernicious anæmia, leukæmia, and the reaction stage of cholera.

VI. Various DRUGS, such as morphia, quinine, phosphorus, arsenic, cantharides, cubebs, copaiba, turpentine, salicylic acid, mercury, and carbolic acid, may give rise to albuminuria. This cause is recognised by (i.) the presence of the drug in the urine; (ii.) there may be a history of the administration of the drug; and (iii.) the albuminuria disappears when the drug is stopped.

VII. DYSPEPSIA and LIVER DERANGEMENT are sometimes accompanied by albuminuria. The symptoms of hepatic congestion may be present, showing the intimate connection between the hepatic and renal functions (§ 238). Albuminuria is sometimes present with that form of dyspepsia which is accompanied by oxaluria. Certain articles of diet are known to have been attended by albuminuria. Thus, cases have been recorded in which albuminuria followed the ingestion of shell-fish, eggs in excess, cheese, and large quantities of alcohol. In many of these instances the condition is probably albumosuria (§ 286).

VIII. DERANGED INNERVATION may be attended by albumen in the urine, such as (1) burns and other causes of severe shock. (2) In exophthalmic goitre the albuminuria is usually a temporary condition, though it may last for months. It may vary in amount at different times on the same day, which tends to show that it is of vaso-motor origin. The urine in other respects is healthy. (3) Excessive study or other cause of nerve strain has been reported to have occasioned albuminuria. (4) Certain cases of cerebral tumour, and other conditions in which there is increased intracranial pressure, have been attended by albuminuria. (5) Albumen is found in the urine after epileptic fits.

IX. PHYSIOLOGICAL or FUNCTIONAL ALBUMINURIA.—A *cyclic* form of albuminuria, or the "albuminuria of adolescence," has been described. It appears regularly at some time each day, usually in the morning or after a cold bath. It is usually absent at night, or when the patient retains the horizontal position, and is possibly therefore of vaso-motor origin. So also is the form of *paroxysmal* albuminuria

which has been described, and which is probably closely related to paroxysmal hæmoglobinuria (below). It appears at intervals, without any apparent cause, and lasts for a few days or weeks at a time. Some of the reported cases were probably early stages of Raynaud's disease, others were perhaps associated with oxaluria. Sir A. E. Wright has shown that in functional albuminuria the renal adequacy is normal, and that the condition is allied to urticaria, in that it is a serous hæmorrhage due to deficient coagulability of the blood.

The *Prognosis* of albuminuria due to congestion is very much that of its cause. Before giving a prognosis it is important to thoroughly and repeatedly examine the urine, for casts in particular, so as to be satisfied that the kidneys are structurally healthy. When due to prolonged dyspepsia and liver derangement, interstitial nephritis may supervene in time if the diet is not properly modified.

Treatment.—The treatment must be directed to the cause. Rest in bed will do a good deal for the renal complication of cardiac disease. In the *albuminuria of pregnancy* careful investigations should be made, and the amount of urea watched. If (1) there is a clear history of renal disease prior to pregnancy, or (2) puerperal eclampsia has occurred in previous pregnancies, or (3) the renal disease, no matter of what kind it may be, is distinctly *progressive in its nature*, then premature labour should be induced. For the treatment of cyclic and paroxysmal albuminuria general hygienic and dietetic rules must be followed. The administration of calcium lactate, by increasing the coagulability of the blood, temporarily stops the albuminuria in some of the functional cases.

§ **300. Hæmaturia.**—When the patient is "passing blood" in the urine, an endeavour should be made to ascertain if the blood comes chiefly at the beginning of micturition, chiefly at the end, or whether it is intimately mixed with the urine and gives to it a "smoky" tint. For the tests for blood in the urine see § 283. The fallacy of menstrual blood must be avoided by using a catheter.

A. If the blood is bright crimson and comes chiefly AT THE COMMENCEMENT *of micturition, it is probably of* URETHRAL *or* PROSTATIC *origin.*

In these circumstances, which are mainly of surgical interest, there will probably be a history of injury or gonorrhœa. In congestion or abscess of the prostate there are local pains and tenderness and rectal irritation.

B. If the blood comes most freely AT THE END *of micturition, and especially if in clots, it is probably of* VESICAL *origin.*

The COMMONEST CAUSES of vesical hæmorrhage are :

I. ACUTE CYSTITIS, chiefly at its onset (see § 304).

II. CALCULUS, or stone, in the bladder. Here the hæmorrhage is worse after exercise, moderate in amount, and there is pain, which, like the bleeding, is worse at the end of micturition and after exercise, and is frequently referred to the point of the penis. The ensuing cystitis may complicate the symptoms and render the diagnosis of stone difficult, but its detection by the sound or cystoscope is conclusive.

III. TUMOURS of the bladder.—The hæmorrhage here, especially in *villous* tumours, is usually great in amount. Shreds of the growth may be passed, and cystitis may develop. In *cancerous* tumours the hæmorrhage is more or less intermittent and resists treatment; there are pain and cachexia, and sometimes the growth may be palpable above the pubes. The cystoscope is the best means we have of recognising the condition of the bladder.

Some of the LESS COMMON CAUSES of vesical hæmaturia are TUBERCULOUS DISEASE of the bladder (when the bacillus may always be found), VESICAL VARIX, certain constitutional diseases such as SCURVY and PURPURA, and BILHARZIA HÆMATOBIA.

Bilharzia Hæmatobia, "Endemic Hæmaturia," occurs in Egypt and South Africa. It is due to the presence of a parasitic trematode. The adult worm is only found in the portal vein and its radicals, but the ova and liberated embryos migrate into and block the veins of the bladder, ureters, or kidney pelvis, causing hæmorrhage and great changes in their mucous surfaces. It is not known how the parasite enters the body, but it is believed to enter the rectum (some say the bladder) while bathing. This cause of hæmaturia may be readily detected by the presence of the highly characteristic ova in the urine (Fig. 90). They are equipolar ovoid bodies with a spinous projection at one end, and can be easily seen under a ½-inch objective. Urotropin relieves the vesical symptoms, but the best treatment is large draughts of water containing benzoic acid (20 to 30 grains daily), and every fourth or fifth day methylene blue (4 grains t.d.).[1] The hæmorrhage may be very great, and severe anæmia result; death generally takes place by pneumonia.

Fig. 90.—Egg of Bilharzia Hæmatobia, magnified about 100.—Life history, see Table XVII., p. 322.

The *Diagnosis* and *Treatment* of these various vesical conditions (excepting the last-named) is mainly in the hands of the surgeon; but temporary relief generally attends rest and the administration of henbane.

C. If the blood is INTIMATELY MIXED *with the urine, causing it to assume a "smoky" tint, it is probably of* RENAL *origin. In these cases also the tests for blood should be carefully applied, and fallacies avoided* (§ 283).

The Causes of Renal Hæmorrhage may for convenience be grouped into: Inflammation (I.); calculus and other causes of pyelitis (II. to IV.); local conditions (V. to VII.); causes from distant parts (VIII. to XI.); paroxysmal hæmorrhage (XII.); and parasites (XIII.).

I. In acute nephritis the blood usually gives rise to the characteristic "smoky" urine, and the deposit contains casts (§ 295).

II. Renal calculus (see below).

III. Tuberculous disease of the kidney (§ 305).

IV. Any of the other causes of pyelitis (§ 305) may give rise to red corpuscles in the urine in larger or smaller amounts.

V. Malignant and other tumours of the kidney cause profuse and sometimes intermittent hæmorrhage (§ 315).

VI. Villous disease of the pelvis of the kidney.

VII. Injury to the kidney (below).

VIII. Passive congestion of the kidney—for example, in heart disease or chill.

IX. Embolism of the kidney (see Endocarditis, § 39).

X. Blood poisons—fevers, scurvy, purpura, etc.

XI. Drugs, such as cantharides or turpentine.

XII. Paroxysmal hæmoglobinuria (below) differs from all the foregoing in the absence of blood discs, though blood colouring matter is plentiful in the urine.

XIII. Parasites—*e.g.*, Bliharzia Hæmatobia (see above). Filaria sanguinis hominis usually causes chyluria, but hæmaturia also may occur.

§ **301. Renal Calculus and Renal Colic.**—Calculi may form either in the pelvis of the kidney or, more rarely, in its substance. Perhaps the commonest form consists of *uric acid* and urates mixed in varying proportions (for tests see § 281). These form stones of light brown colour, either round or branching, and are the commonest stones in subjects of

[1] Mr. P. S. Lebeau, R.A.M.C., Report of the Med. Soc., London, in the *Lancet*, May 10, 1902.

the gouty diathesis, and those whose urine habitually deposits lithates. The other variety, which is dark brown in colour, consists of *oxalate of calcium*, and gives rise to acuter symptoms, for each bristles with sharp-pointed crystals which wound the mucosa. Calculi are often multiple. Compound stones consisting of an oxalate nucleus, or alternate layers, are met with. Phosphates and cystine are only very rarely met with in renal calculi. Various *events* may happen. (1) A calculus may remain in the renal pelvis, giving rise to chronic pyelitis (§ 305) for years ; or (2) by its movement produce acute symptoms, RENAL COLIC. (3) It may obstruct the ureter and lead to hydro- or pyo-nephrosis (§ 315). (4) If the other kidney is not healthy sudden blocking may lead to obstructive suppression (§ 312). (5) It may pass into the bladder and result in cystitis. (6) Small stones may be voided through the urethra as "gravel." (7) In rare cases small calculi become encysted and quiescent. The typical clinical history of renal calculus consists of (*a*) *attacks of renal colic*, separated by (*b*) *intervals* in which the symptoms are those of calculous pyelitis (§ 305).

The *Symptoms of Renal Colic* consist of severe paroxysms of lancinating pain, starting in one loin, shooting down to the testicle or vulva on that side ; attended by vomiting, shivering, sweating, pallor, and a certain amount of collapse. These symptoms are in most cases followed by hæmaturia, the urine containing blood discs and pus cells, but usually no casts. Crystals are also present, and guide us as to the nature of the stone. It is with the oxalate calculus that most blood and pain occur. The diagnosis of renal from other forms of colic is given in the form of a table (XIII., p. 256). All the symptoms of renal colic may arise simply from the irritation of *fine crystals*. They may also be produced without alteration in the urine by *movable kidney ;* or by the passage of *clots* of blood or caseous material down the ureter. *Malignant* disease of the kidney may be mistaken for calculus, but in that case the blood is more copious and more constant, and the pain is less severe, but more continuous.

Treatment.—(1) Of the colic and (2) during the intervals. 1. The treatment of an attack of *renal colic* consists mainly in the relief of the symptoms—pain, vomiting, and collapse. Various forms of hypnotics may be tried (see Neuralgia), but usually nothing avails except morphia, and it may safely be given unless there is reason to fear the renal parenchyma is also diseased. Effervescing citrate of potassium with spiritus ammoniæ aromatici may be administered with advantage, and rest should be enjoined. 2. The treatment in the intervals resolves itself into (i.) the prevention or solution of the stone, and (ii.) treatment directed to the pyelitis. (i.) Dietetic treatment is of great use in some cases. If uric acid is being passed, the treatment is the same as that for lithæmia (§ 249). If oxalates are being passed, any dyspepsia should be carefully treated ; such articles of diet as rhubarb,

tomatoes, cabbages and onions, sweets and alcohol, should be avoided. The urine in all cases should be kept diluted by drinking plenty of fluid. The alkaline waters are very useful here, such as those of Vichy, Ems, and Contrexéville. In uric acid calculus, large doses of alkaline salts are certainly useful, especially the citrate and the acid tartrate of potassium. Begin with 50 grains of potassium citrate in 4 ounces of water every four hours until the urine is alkaline, and then give an effervescing drink, consisting of 1 drachm of sodium bicarbonate, and 40 grains of citric acid in 4 ounces of water, three times a day. This treatment should not be continued if the urine is or has become ammoniacal. (ii.) For the treatment of pyelitis see § 305. (iii.) Operative treatment is called for if repeated attacks of colic recur, and especially if the stone can be detected by radiographic examination.

Injury of the Kidney, laceration or rupture, is usually caused by a fall on the back or loin, or in " buffer accidents " on the railway during shunting operations. There may be no bruising or external signs, but a laceration of the kidney may be inferred from (1) the history of such an accident; (2) a tense swelling (due to extravasated blood) with increased area of dulness in the region of the kidney; and (3) copious hæmaturia. In a few cases there is no hæmaturia, and the other two evidences have to be relied on. Immediate operation is advised by Mr. Bland-Sutton and others,[1] the collapse being treated by saline injections.

§ **302.** In **Paroxysmal Hæmoglobinuria** (Paroxysmal Hæmatinuria, Roberts) porter-coloured urine is passed at intervals. An attack commences abruptly with (1) *a rigor* or " chilliness," nausea, and malaise; and (2) lumbar pain. (3) An hour or so later the patient passes dark, highly albuminous urine, showing the spectroscopic band of methæmoglobin, containing no red discs, but a quantity of amorphous granular matter. It has a specific gravity of 1020 to 1022, a slight excess of urea, and deposits crystals of oxalate of calcium. Each attack lasts a few hours, and passes off as suddenly as it came, but only to recur in a few hours' or days' time. In the intervals the general health is fair, but later the patient becomes anæmic and languid. Relapses recur for months or years without fresh exposure to " chill."

The *Causes* are obscure. The symptoms clearly indicate, as Sir William Roberts points out,[2] a sudden transitory congestion of the renal capillaries with transudation, but *no rupture*. In 90 per cent. of the cases (Roberts) the attacks are connected with chill to the surface, and this seems to determine a reflex vaso-motor dilatation of the renal vessels. The disease is sometimes associated with rheumatism, oxaluria (the sharp crystals of which were thought by some to produce the attacks), malaria, mental or physical over-exertion, and dyspepsia.

The *Treatment* consists of rest in bed during the attacks, with warmth, and hyoscyamus internally. Persons predisposed to such attacks should avoid exposure to cold, and take iron. In one case seen by the author bromide of ammonium had a marked effect in preventing the attacks.

Symptomatic Hæmoglobinuria may occasionally accompany Raynaud's disease, malaria, severe burns, and acute infective diseases.

Toxic Hæmoglobinuria may be produced by toxic doses of chlorate of potassium, naphthol, pyrogallic acid, carbolic acid, arseniuretted hydrogen, quinine, and carbonic oxide.

The patient complains of lassitude *and ill-health, which have come on gradually; the urine is found to contain* pus (§ 284)—*i.e., there is* Pyuria.

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[1] Discussion at the Clin. Soc., *Lancet*, November 17, 1899.
[2] Reynolds' " System of Medicine."

With few exceptions (see footnote 2, below), when the pus comes from the BLADDER *the urine is* ALKALINE, *and the pus remains diffused through the urine; but when it comes from the* KIDNEYS *or any other part of the urinary passages the urine is* ACID, *and the pus settles at the bottom. Pus cells produce a trace of albumen in the urine.*

§ **303. Pyuria.**—If we except the rupture of an abscess into the urinary passages, there are three sources of pus in the urine :

A. From the **Urethra** (*e.g.*, gonorrhœa).

B. From the **Bladder** (cystitis).

C. From the **Kidney** (pyelitis).—There are three chief forms of pyelitis : CALCULOUS, TUBERCULOUS, and ASCENDING.

Abscesses bursting into the Urinary Tract.—The abscesses most liable to burst into the urinary tract are : (*a*) prostatic abscess (above) ; (*b*) perineal abscess ; (*c*) pelvic cellulitis ; (*d*) psoas abscess ; (*e*) perinephric abscess ; and (*f*) abscess of the liver ; and there are also many other sources. (i.) The urine is usually acid ; (ii.) the pus is in large quantity and settles at the bottom ; (iii.) there is a clinical history of abscess prior to the appearance of pus in the urine ; and (iv.) localising signs of the abscess may be present.

It is believed by some observers that persons in health may pass a few leucocytes, but it is extremely probable that these are always derived from the generative organs (male or female), and that the occurrence of *any* pus cells in a properly collected catheter specimen is always pathological.[1]

When the presence of pus is suspected, the reaction should be tested immediately after it is passed, before decomposition can set in. Decomposition makes the urine ammoniacal, and therefore alkaline.

A. The **pus** *comes chiefly at the* BEGINNING OF MICTURITION, *and the urine is* ACID ; it comes from the URETHRA, and is usually caused by one of three conditions :

I. URETHRITIS.—There is pain, swelling, and redness of the meatus, scalding during micturition, and discharge of pus apart from micturition.

II. PROSTATIC ABSCESS is known by : (1) pain at the end of micturition ; (2) the finger in the rectum detects a tender, fluctuating swelling ; (3) the symptoms closely resemble those of vesical calculus with concurrent cystitis. It may be distinguished from this, however, by : (i.) a history of gonorrhœa, which is the chief cause of prostatic abscess ; (ii.) the signs on examination per rectum ; and (iii.) a discharge occurring in the intervals between micturition.

III. PERINÆAL ABSCESS is detected by the local signs.

B. The **pus** *comes chiefly at the* END OF MICTURITION, *or is intimately mixed with it ; the urine is* ALKALINE *when tested immediately after it is passed*[2] (*alkaline pyuria*). The pus comes from the BLADDER, and is indicative of CYSTITIS.

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[1] In some cases there is a history pointing to leucorrhœa or gleet, but the quickest way of settling this point is to draw off the urine by catheter.

[2] At the outset of acute cystitis the urine may be acid, and it may become acid again in the stage of recovery from chronic cystitis. It may also be acid in the early stage of tubercle and new growths of the bladder, and in cases of cystitis due to bacillus coli communis. In all other conditions in which the urine contains pus derived from the bladder the reaction is alkaline.

§ **304. Cystitis,** or inflammation of the bladder, occurs in two well-recognised forms—acute and chronic.

(*a*) In Acute Cystitis.—(1) In this condition the pus is in small amount, and in severe cases there may be considerable hæmaturia at the outset. At first the urine is acid, but it soon becomes alkaline, and ropy with pus and mucus. (2) There are pain and tenderness in the hypogastrium. (3) Micturition is frequent and painful ("scalding"). After micturition the pain is relieved for a short time, unless the cystitis is due to stone in the bladder, when the pain is severe *after* micturition, because the inflamed walls of the emptied bladder then come into contact with the stone. (4) There is generally marked constitutional disturbance, with pyrexia.

(*b*) In Chronic Cystitis (which may supervene upon the acute form, or the cystitis may be chronic from the outset), there is (1) a larger amount of pus. (2) The urine is markedly alkaline, directly it is passed, and contains a large amount of ropy mucus. (3) The pain and other symptoms are less severe than in acute cystitis.

Etiology.—(i.) Gonorrhœa causes the most severe and often fatal form of acute cystitis and pyelo-nephritis. Other causes are (ii.) stone or foreign bodies setting up irritation; (iii.) injury by instruments or foreign bodies introduced by the patient; (iv.) the use of catheters which have not been rendered thoroughly aseptic; (v.) cancer, villous disease, and other tumours of the bladder; (vi.) urine decomposing in the bladder, as in stricture urethræ, prostatic enlargement, and other causes of retention of the urine (§ 311); (vii.) various nerve complaints, producing paralysis and retention; (viii.) extension from a urethritis or inflammation from adjacent organs, as in pelvic cellulitis; (ix.) tubercle not infrequently affects the bladder, when bacilli are found in abundance; (x.) other microbes are now known to affect the bladder, notably the bacillus coli communis, which produces a mild cystitis, and which is very apt to ascend (see Infective Pyelo-nephritis); (xi.) various constitutional states, such as gout and diabetes, are said to predispose to cystitis; (xii.) drugs—*e.g.*, cantharides or turpentine. The diagnosis of these causes is mainly accomplished by the surgeon.

Differentiation.—(1) Cystitis due to Vesical Calculus.—In addition to the symptoms of simple cystitis, there is (i.) pain at the end of micturition, lasting for some time after, very severe, shooting down the urethra; (ii.) hæmaturia is common, though in some cases it may be so slight that it is detected only by the microscope; (iii.) a history of renal colic (§ 301).

(2) Cystitis due to New Growth in the Bladder, or Ulceration, is characterised by (i.) paroxysms of lancinating pain, quite independent of micturition and movement; (ii.) copious hæmorrhage at intervals, occurring without apparent cause; (iii.) the urine may contain cancer cells or tubercle bacilli; a tumour may be felt per rectum or through the abdominal wall. (iv.) Cystoscopic examination may settle the diagnosis.

Prognosis.—Cystitis is not dangerous to life unless the inflammation spreads upwards from the bladder to the kidneys and produces pyelo-

nephritis ; but, on the other hand, it is a very troublesome, painful complaint, and has a special liability to recur. When the cause is not removable—*e.g.*, in cystitis due to tumours of the bladder—the prognosis is very grave. When it is due to retention of urine (such as that caused by the atony of the bladder in old age), and when it is due to gonorrhœa, it tends to cause ascending pyelitis and pyelo-nephritis. When there is pre-existing hydronephrosis (§ 315), and acute cystitis develops, the inflammation is almost certain to extend upwards to the kidney, and so lead to pyonephrosis.

Treatment.—The cause must be sought for, and, if possible, removed. (*a*) In the *acute* form absolute rest in bed with milk diet is necessary. Copious libations of water, barley-water, and other bland fluids are called for. Alkalies allay the irritability of the bladder. Mild laxatives should be given, combined with hyoscyamus. Boric acid, 10 to 30 grains, thrice daily in large draughts of water is valuable. Soothing drugs—*e.g.*, buchu, triticum repens, and uva ursi—are useful. Hot sitz-baths and morphia suppositories are given to relieve the pain. It is useful to administer internal antiseptics, such as quinine and salol, and urotropin (7 grains three times a day). (*b*) For the *chronic* and *subacute* forms wash out the bladder with hot water and boric acid. Sir Henry Thompson recommended that it is better to use a strong solution of boric acid, not exceeding 2 ounces at each sitting, than to wash out with large quantities. Benzoic acid (gr. xv. t.d.s.) renders the urine acid. (*c*) Therapeutic vaccination is now widely employed, and has proved of great service in uncomplicated bacterial infection of the urinary tract, especially tuberculous and coli infections.

C. The **pus** *is associated with a urine which is* ACID *when freshly passed* (**acid pyuria**), *the pus cells are at first disseminated through the urine, but in a short time they settle down as a* SEDIMENT, *and there are* PAIN, *and perhaps* SWELLING, *of the kidney*—the pus comes from the kidney, and the disease is PYELITIS.

§ **305. Pyelitis,** or inflammation of the pelvis of the kidney, is indicated by the symptoms just mentioned. The urine, which is acid unless there be concurrent cystitis, contains, in addition to pus cells (Fig. 82), epithelial cells from the renal mucosa ; but, unless the renal parenchyma is involved, no casts and no albumen in excess of the quantity which would be accounted for by the pus are found, nor is there any dropsy. There is increased frequency of micturition. Renal pain (nephralgia) and tenderness are nearly always present, but they vary widely in degree and character in the three varieties about to be mentioned. *The kidney should always be carefully examined* (§ 292), because, in addition to the renal congestion, all forms of pyelitis are liable to result in partial or complete obstruction of the infundibula, and the gradual supervention of hydro- or pyo-nephrosis. A few pus cells in the urine may be found

in acute nephritis, after enteric and other fevers, and toxic doses of cantharides or turpentine. Apart from these there are three well-marked varieties or causes of acid pyuria.

I. Calculous Pyelitis is due to the irritation set up by the presence of a stone. The *Differential Symptoms* are : (i.) A history of renal colic (§ 301) is often obtainable. (ii.) *Pain on one, the diseased, side*, which varies with exercise, and (iii.) hæmaturia, also varying with exercise. (iv.) The quantity of pus often varies from day to day, and the patient may feel easier after a discharge of pus, as the retained pus causes pain, and sometimes swelling. (v.) Attacks of intermittent pyrexia from time to time. (vi.) *Crystals in the urine* aid the diagnosis considerably.

II. Tuberculous Pyelitis.—Tuberculous disease of the kidney may be primary or secondary to tubercle elsewhere. Very often both kidneys are diseased. This condition may be very difficult to diagnose from Calculous Pyelitis, but the *Differential Symptoms* are : (i.) No previous history of colic, but dull pain in the loins, liable to exacerbations from the passage of caseous masses ; (ii.) hæmaturia is not usually present[1] ; (iii.) the amount of pus in the urine does not vary but steadily increases ; (iv.) the urine contains amorphous granular matter and tubercle bacilli, but usually no crystals ; (v.) *pyrexia of a regularly intermitting type*, with increasing emaciation ; and (vi.) there are often evidences of tubercle in other parts of the body, as in the testes or lungs ; (vii.) Calmette's and Von Pirquet's reactions are present, and the opsonic index indicates tubercle (*vide* § 387).

III. Ascending Pyelitis or Pyelo-Nephritis arises from three groups of causes, which may conveniently be termed Obstruction, Extension, and Infection Pyelitis. (*a*) Some *obstruction in the urinary passages* below the kidney not infrequently causes retention and decomposition of the urine, and septic infection of the pelvis of one or both kidneys, which may go on to pyo-nephrosis (Fig. 91, p. 445). The diagnosis of this form, which used to be known as "Surgical Kidney," rests mainly on the history of the cause of retention—enlarged prostate, urethral stricture, uterine and other tumours pressing upon, or calculus impacted within, the ureter (see also Retention, § 311). Here, as in the next group, the urine may be alkaline from concurrent cystitis. (*b*) Ascending pyelitis may also result from the *extension of cystitis* without obstruction, and thus the numerous causes of the latter disease (§ 304) are brought into operation—*e.g.*, gonorrhœa, septic catheterisation, etc.

(*c*) Infective Pyelo-Nephritis is a condition which is gradually being recognised by the profession. Previous bladder symptoms may be slight, transient, or altogether absent. The nature of the microbic infection which infects the urine is not always apparent ; but that the b. coli communis is capable of so acting is beyond doubt. The *Symptoms* of coli infection may be wholly indistinguishable from calculous pyelitis on the one hand and tuberculous pyelitis on the other, unless one is aided by the detection of the respective microbes in the urine. There are, however, three features which in the author's experience are fairly characteristic of the coli infection : (i.) the occurrence of attacks of pyrexia at irregular intervals of a distinctly pyæmic type, attended by shivering, sweating, and vomiting ; (ii.) a distinctive smell of volatile sulphides in the urine ; and (iii.) the

[1] Occasionally hæmaturia is an early symptom of renal tuberculosis.—Dr. Newman, *Lancet*, vol. ii., 1899, p. 559.

fact that pure cultures of b. coli can be readily obtained from a specimen of the urine collected through a sterilised catheter. The disease runs a most indefinite course, but usually wears itself out sooner or later. It occurs chiefly in women and children, and especially during pregnancy. Vaccine treatment has been successful.

Prognosis.—(i.) The most serious form of pyelitis is that due to extension of inflammation upwards from the bladder. When originating in gonorrhœal cystitis, death usually occurs in seven to fourteen days. (ii.) In the tuberculous form there may be no general symptoms until the disease extends beyond the one kidney ; in other cases it may be fatal in twelve to eighteen months. (iii.) Calculous pyelitis may last indefinitely for years, though not without danger of uræmia and abscess of the kidney. (iv.) The course of ascending pyelitis depends very much upon the cause, the possibility of its removal, the age of the patient, and his general condition. *Pyonephrosis* is apt to ensue.

Treatment.—1. In *all forms of pyelitis* fluid diet, milk and warm drinks, rest and warmth are essential ; and cupping of the loins is sometimes useful. Sedative drugs, such as hyoscyamus and belladonna, may be administered, and antiseptics, such as boric acid, quinine, and creosote may relieve the condition. Buchu, pareira, urotropin, and salol are useful. Many of these cases call for nephrectomy or other surgical measures. The question is often raised (before or during operation) whether one or both kidneys are diseased. This is a difficult question to answer, and each case presents different data ; but in all cases *daily observations on the amount of urea* should be made (*cf.* § 280). 2. Of *calculous pyelitis.*—If due to uric acid calculi large doses of potassium citrate and bicarbonate may be employed ; if due to oxalates, nux vomica, and nitro-hydrochloric acid ; nephrolithotomy in nearly all cases. 3. Of *tuberculous pyelitis.*—Tonics such as iron, quinine, and cod-liver oil must be given. Excision of the kidney is to be advised if (i.) the other kidney is believed to be healthy ; and (ii.) there is no tuberculous disease elsewhere in the urinary tract, in the lungs, or intestines. Inoculation with an emulsion of dead bacilli, the dose of which is graduated, if necessary, by the estimation of the opsonic index, has given encouraging results. 4. For *ascending pyelitis*, our attention is best directed to the cause.

A **diminution in the specific gravity** *when marked and continuous, even in the absence of albumen, is suggestive of* CHRONIC INTERSTITIAL NEPHRITIS, *or more rarely* DIABETES INSIPIDUS. **A marked increase** *in the specific gravity is suggestive of* DIABETES MELLITUS.

§ **306.** The other **causes of altered specific gravity** are relatively less important, because they are identified mainly by other means. Nevertheless, the specific gravity of the urine is an extremely important feature, because, in the absence of sugar, it is a MEASURE OF THE NITRO-

GENOUS and SALINE EXCRETION, the specific gravity being higher in direct proportion to the amounts contained in a given sample of urine. Therefore, with certain reservations about to be mentioned, it is a very fair measure of the FUNCTIONAL ACTIVITY of the secreting substance of the two kidneys taken together. For example, when one kidney is known to be diseased or destroyed, it will give us a good idea of the condition of the other, and in Bright's disease we may learn something of the amount of renal epithelium undamaged. In such cases regular estimations of the urea secreted should be made (§ 280). The reservations just alluded to are four in number: (1) the specific gravity must always be considered in relation to the total diurnal quantity of the urine; (2) the total urea varies considerably with the body weight, being less in women and persons of slight build; (3) it varies *to some extent* also with the amount of proteid food ingested, and the work done by the body—thus it is rather less in a person lying in bed; (4) it is assumed that the liver is healthy because, as mentioned in the introduction to Chapter XII., the first stage in the manufacture of urea takes place there, only the concluding stage being performed by the kidney.

The variations in the *total output of urea* have been mentioned under the several diseases of the kidney.

The specific gravity is DIMINISHED in—

1. Chronic Interstitial Nephritis.
2. Polyuria, and all the diseases about to be mentioned under that heading, excepting Diabetes Mellitus.
3. Myxœdema and other conditions where the nitrogenous disintegration within the body is diminished.

The specific gravity is INCREASED in—

1. Diabetes Mellitus (owing to the sugar).
2. Some renal diseases where the quantity of water is considerably diminished, such as Acute Nephritis or the Cardiac Kidney.
3. Febrile and other conditions where the nitrogenous disintegration is excessive.
4. Whenever the urine becomes concentrated by profuse sweating, vomiting, or diarrhœa.

An **increase** (POLYURIA), *or* **diminution**, *in the* **quantity of urine** *is complained of by the patient in several important diseases.*

§ **307.** In **Polyuria** it is very desirable to measure the total diurnal quantity, since patients are very apt to mistake increased frequency for increased quantity, and *vice versa*.

There is INCREASED QUANTITY of urine secreted in—

1. *Diabetes mellitus*, which is known by the high specific gravity of the urine and persistent glycosuria.
2. *Diabetes insipidus*—low specific gravity and malaise, but no sugar.
3. *Chronic interstitial nephritis*, which is known by the low specific gravity of the urine, slight albuminuria, etc. (§ 297).
4. *Waxy kidney*, which is known by the low specific gravity of the urine and great albuminuria (§ 298).
5. *Hydronephrosis*, which is known by the passage of large quantities of urine

for a limited period of time, accompanied by the disappearance of a swelling from the loin. This is followed by a return to the normal both in quality and quantity of the urine, and then a gradual re-formation of the swelling (§ 315).

6. *Convalescence* after fevers.

7. *Temporary polyuria* occurs in hysteria, nervous excitement, and any other condition giving rise to reactionary or paralytic condition of the abdominal sympathetic.

8. During the administration of *diuretics*.

9. During the *absorption of exudations*, such as pleural effusion.

There is DIMINISHED QUANTITY of urine in—

1. Acute Nephritis.
2. Subacute and Chronic Tubal Nephritis (some stages).
3. Final stage of Chronic Interstitial Nephritis.
4. The Cardiac Kidney and some other Renal Congestions.
5. Febrile states.
6. Whenever there is profuse vomiting, diarrhœa, or perspiration, or but little fluid is taken.

The patient complains of **polyuria** ; *the urine is of* HIGH SPECIFIC GRAVITY, *and* CONSTANTLY *contains* SUGAR (**glycosuria**) ; *there are also thirst, and, in spite of a voracious appetite, gradual loss of flesh.* The disease is DIABETES MELLITUS.

§ **308. Temporary Glycosuria** may arise from several causes, many of which are due to active hepatic hyperæmia. Often it is of little or no consequence. (1) Dietetic errors (glycosuria only after a meal). (2) Gradual occlusion of the portal vein. (3) After large or prolonged doses of certain drugs—chloroform, chloral, morphia (the reaction here *may* be due to glycuronic acid). (4) After epileptic convulsions. (5) During the collapse of cholera. (6) During the paroxysms of ague. (7) In chronic Bright's disease with high tension. (8) Cardiac disease, asthma, pertussis, and some other cases of dyspnœa. (9) Injury to the liver. (10) Congestion of the liver in gouty people, and when much exercise is taken by those unaccustomed to it. (11) Intestinal irritation. (12) After concussion and compression of the brain, and tumour cerebri, especially if involving the floor of the fourth ventricle. (13) Violent mental and moral emotions. (14) During pregnancy and suckling. (15) Pancreatic disease. (16) After acute fevers such as cholera, malaria, influenza, or diphtheria.[1]

§ **309. Diabetes Mellitus** is a constitutional disease, characterised by the passage of large quantities of urine containing glucose, associated with progressive emaciation and voracious appetite. 1. There is usually increased frequency of micturition, and the patient passes large quantities (6 to 40 pints) of clear pale urine, which has a sweetish odour. If dropped upon the boot, this leaves a crystalline deposit, by which means the condition has occasionally been identified. The specific gravity is high—1,030 to 1,040 or more. The amount of sugar varies from 2 to 40 grains or more per ounce, and the total amount per day varies from 10 ounces to 2 pounds. In diabetes the sugar may occasionally disappear for several days, but in general terms it is permanent and persistent. The diurnal quantity of urea and phosphates is increased ; acetone may be present, and albuminuria sometimes

[1] Cases of non-diabetic glycosuria are reported by Dr. Saundby, *Brit. Med Journ.*, April 14, 1900.

especially towards the end. 2. Progressive weakness and emaciation are sometimes the first symptoms to attract notice. 3. At other times thirst or voracious appetite, accompanied by a raw beefy tongue and dry skin, are the first signs. 4. The complications (*vide infra*) not infrequently lead to our detecting the disease, for its earlier stages are often overlooked by the patient.

Varieties.—There are two well-marked varieties of diabetes : (*a*) The mild form, which is met with in corpulent middle-aged people, where the symptoms are moderate, and dietetic restriction removes the sugar from the urine. This is really a *transient glycosuria*. (*b*) The severe variety is met with in *acute* and *chronic* forms. The acute form usually occurs in children or young adults, and occasionally after head injuries. The chronic form is met with in older people, and is attributed sometimes to mental worry. It also occurs with tumour of the fourth ventricle and other causes of transient glycosuria which become chronic.

Causes.—Diabetes occurs in the proportion of three males to two females, and may be hereditary. Gout, insanity, or phthisis may be present in the family. Sedentary habits and brain overwork may predispose. The pathogenesis is not at present known, but it is certain that the pancreas is fibrotic in about 50 per cent. Physiological evidence also points in this direction. It is probable, also, that the glycogenic function of the liver is in some way interfered with, possibly indirectly through the pancreas.

The *Complications* of diabetes are numerous. In order of frequency they are : 1. *Phthisis*, which is one of the most frequent causes of death in the condition. 2. Various *skin conditions*, especially eczema, boils, pruritus, and xanthelasma, which appear early in the disease, and carbuncle and gangrene, which appear in the later stages. It is essential to examine the urine for glucose in all cases of boils, carbuncles, and pruritus vulvæ. 3. The *nervous* system is specially apt to be affected, and peripheral neuritis is now known to be frequently caused by diabetes. The knee-jerks are commonly lost in diabetes, sometimes without any other nerve symptom, or there may be tingling, numbness, perforating ulcer, or neuralgia. Restlessness is common, and this may go on to mania or melancholia. The sudden supervention of diabetic coma often terminates life. 4. *Ocular changes* are almost as common as the foregoing, and these may take the form of soft cataract, or defective accommodation leading to a rapidly increasing presbyopia. Retinitis, optic atrophy, and amblyopia also occur.

Diagnosis.—In any of the conditions mentioned under *Complications* the urine should be examined. This is the key to the diagnosis. In *diabetes insipidus*, *granular kidney*, *amyloid kidney*, and sometimes in *hysteria* the quantity of urine is excessive, but in none of these conditions is sugar present. Two golden rules will often enable us to

identify a case of diabetes which otherwise might be overlooked: 1. Always examine the urine of a patient suffering from boils or from eczema of the genitals; and 2. of a patient the subject of apparently causeless wasting. For the diagnosis of diabetes from *temporary* glycosuria *vide supra*.

Bremer found that diabetic blood has a special reaction with certain stains. Heat the diabetic blood-film, together with a control film or normal blood, at 130° C. in the oven for six to ten minutes. Stain two minutes in 1 per cent. solution of Congo red. Normal blood is stained red; diabetic blood is not stained. Williamson's chemical test for diabetic blood has been found useful. Make the following mixture: Blood, 20 centimetres, aqueous methylene blue, 1 c.c., liquor potassæ (specific gravity 1058), 40 c.c., water, 40 c.c. Stand the tube containing this mixture in boiling water for four minutes. If the blood is diabetic the mixture becomes yellow; normal blood does not.

Prognosis.—1. The mild form, which is met with chiefly in corpulent persons and others over thirty-five, may occasionally pass into the graver form, but generally with suitable diet the sugar disappears, and the condition warrants an excellent prognosis. 2. In the severer forms the prognosis chiefly turns upon the age of the patient. If the disease is established in a young adult, life rarely lasts more than two years at the outside. The effect of *diet* is a valuable aid to prognosis. If by this means the sugar can be reduced to 3 or 4 grains per ounce, the patient may live many years, especially if the disease did not come on until middle life was reached; if, on the other hand, 10 or 20 grains are constantly present, the case will go rapidly downhill. The presence of *complications* other than pneumonia or phthisis does not add very materially to the gravity of the situation. Death may ensue in three ways: (i.) By complications—a third of the cases die of phthisis; (ii.) by asthenia; and (iii.) about an equal number die with coma. Coma is heralded in most cases by certain symptoms which it is well to bear in mind. Such are a decrease in the amount of urine, the occurrence of albuminuria, a rapid increase in the urine of the fatty acid series, viz.: β-oxybutyric acid, diacetic acid, and acetone (§ 287), epigastric pain, increasing languor, an accelerated and sighing respiration ("air-hunger"), and drowsiness. In some cases the coma supervenes suddenly, after a period of excitement. A peculiar sweet odour in the breath, due to acetone, is often a valuable means of diagnosis of diabetic coma.

The chief *Treatment* is dietetic, and consists in the reduction of sugars and farinaceous foods. A strict dietary is given in § 212. The amount of sugar in the urine should first be estimated, and then the carbohydrates gradually diminished, until the sugar has been brought as low as possible. Milk and a little bread should be retained, unless they seriously affect the amount of sugar passed. The tendency is to be more lenient in the dietary than heretofore. Saxin is taken in place of sugar. Among therapeutic agents codeine, one of the alkaloids of opium, still takes the first place. It is given in increasing quantities

from ½ grain to 6 or 8 grains. Professor Ebstein recommends sodium salicylate. If diarrhœa is present, opium may be given, and very large doses are tolerated. Uranium nitrate is a remedy still on its trial. Arsenic, bromides, and antipyrin are used, especially when the nerve structures are affected. Among the symptomatic indications, thirst is best allayed by frequent doses of dilute phosphoric acid; the voracious appetite and dyspepsia may sometimes be allayed by bismuth. Coma has not hitherto been successfully treated by the ordinary means, but cases have been reported of recovery after large intravenous saline injections, to which 1 to 2 per cent. of sodium bicarbonate should be added. If coma is threatened, as shown by the presence of diacetic acid and increase in acetone, more carbohydrate must be allowed; levulose is useful in this respect, for, although a carbohydrate, it does not increase the amount of sugar in the urine. Large doses of alkali must also be administered by the mouth.

Pancreatic Diabetes.—It is not quite certain whether this exists as a separate variety, but some cases of Diabetes Mellitus have been observed with pancreatic lesions, such as chronic inflammation and fatty degeneration. They have been characterised by the presence of undigested fat in the fæces, and the ordinary symptoms of a severe diabetes running a very rapid course. In chronic pancreatitis and pancreatic calculi, glycosuria is often present. On the other hand, pancreatic disease may be unattended by glycosuria.

The patient complains of **polyuria** *and many of the other symptoms of Diabetes Mellitus, but the* SPECIFIC GRAVITY OF THE URINE IS LOW, *and there is* NO SUGAR. The disease is DIABETES INSIPIDUS.

§ 310. **Diabetes Insipidus** is characterised by great and persistent increase in the quantity of the urine, without glycosuria and albuminuria, attended by great thirst and emaciation. It is believed to be due to a dilatation of the renal vessels, though how this permanent dilatation occurs is uncertain. The fact that the condition occurs with tumours of the medulla or pons, or with lesions involving the thoracic or abdominal nerve ganglia, is strongly suggestive of a vaso-motor paralysis.

Symptoms.—(1) The amount of urine may be very great, from 10 to 20 pints per day. It is pale in colour, so that it may resemble clear water. The specific gravity averages 1002 to 1005. The diurnal amount of solid constituents is as a rule not very much increased, and no other abnormality may be present. Occasionally traces of albumen and sugar appear towards the end. (2) In the mild form of the disease polyuria and thirst are the only symptoms; but in the severer variety nearly all the symptoms mentioned under Diabetes Mellitus are also present—dry skin, emaciation, large appetite, and alternating constipation and diarrhœa. Indeed, it is distinguished from that condition only by the absence of glycosuria. Intercurrent attacks of pyrexia have been observed. (3) Obscure nervous symptoms, with irritability of temper, are common in this disease—such as disturbed sleep, occipital headache, neuralgic pains in the lumbar region, diminished reflexes, and muscular twitchings.

Diagnosis.—The disease is apt in its early stages to be mistaken for *chronic interstitial nephritis*, but the greater age of the patient, the presence of traces of albumen, and of cardio-vascular symptoms, and the absence of thirst and voracious appetite help to distinguish the latter condition. With *amyloid kidney* there is much albumen, and with both *hydronephrosis* and *cystic kidney* a tumour is generally palpable in the region of the kidney (*cf. Phosphatic Diabetes*, § 314).

Causes.—(i.) More males are affected (two or three to one female). Childhood and early middle age are the favourite ages. (ii.) It is said to occur in association with a nervous temperament. Certainly among the chief exciting causes are injuries to the nervous system, especially blows on the head. Intracranial tumour or inflammation or powerful emotions are also exciting causes. (iii.) Muscular exertion, exposure to cold, and intemperance are also mentioned.

Prognosis.—The milder varieties may last for a great many years, and exist rather as an inconvenience than as a malady. In the severer forms, especially those due to intracranial tumours, the course may be very rapid, and death ensue in the course of a month. When setting in acutely after injury to the head (which may be attended by some glycosuria at first) recovery may ensue after a year or so. In children with the tuberculous diathesis death usually occurs in the course of one or two years. In general terms, cases setting in acutely are more hopeful than those which start insidiously. Death may take place from gradual exhaustion, drowsiness passing into coma, with or without convulsions, or from complications such as phthisis or pneumonia.

Treatment.—Most reliance is placed upon hygienic treatment. Tea, coffee, and other substances which increase diuresis should be avoided, but the amount of fluid taken should not be reduced below that excreted. Of drugs the favourite is valerian, gr. v. of the powdered root, increased up to gr. xxx. Nitroglycerine has been used, and arsenic relieves the gastro-intestinal and skin symptoms. When there is disease of the bulb, electricity may be tried—the positive pole placed on the back of the neck, the negative pole passed through the nostril to rest on the cervical spine, ½ to 5 milliampères for five minutes every second day.

The patient complains that he **cannot pass water,** *and a* DISTENDED BLADDER *can be made out by percussion and palpation above the pubes, or by the passage of a catheter.* The condition is RETENTION OF URINE.

§ **311. Retention of Urine** come mainly within the province of the surgeon. Those of *sudden* onset are often due to urethral spasm or congestion; those of *gradual* onset are more numerous. The age and sex of the patient may aid us. Thus, in *childhood* we may suspect impacted calculus, phimosis, or a ligature round the penis; in *women*, tumours pressing on the neck of the bladder (*e.g.*, fibroid or retroverted uterus), hysteria, or reflex irritation after parturition; in young or middle-aged *adults*, stricture, gonorrhœa with congested mucous membrane, spasm after exposure to cold or a drinking bout, or tabes dorsalis; in *old men*, prostatic enlargement, or atony of the bladder. At all ages there may be calculus or tumour blocking the neck of the bladder, paralysis of the bladder from diseased or injured cord or brain, or reflex spasm after operations about the perinæum.

The *Treatment* is mainly surgical, but in cases of spasm a hot bath or hot fomentations to the abdomen will give relief. Hysterical and other nervous affections are referred to elsewhere. Atony and simple vesical paralysis may be treated by nux vomica, and the constant current, one pole being placed on the perinæum and the other just above the pubes.

The patient complains that he has **not passed any water** *for some time, but there are* NO EVIDENCES *of a* DISTENDED BLADDER, *and on passing a catheter it is found to be empty, or nearly so.* The condition is SUPPRESSION OF URINE.

§ 312. **Suppression of Urine** is a very grave condition. A catheter should always be passed before the diagnosis of suppression is made. There are two kinds: I. Obstructive suppression, which is due to some obstruction to the flow of urine through the ureters; and II. Non-obstructive suppression, which is due to the non-secretion of urine by the kidneys. This latter form is sometimes spoken of as true suppression.

I. Obstructive Suppression is due to partial blocking of one or both ureters (the kidneys being healthy) by (i.) renal calculus; (ii.) tumour at the base of the bladder; (iii.) congenital malformation of the ureters. The *Symptoms* are: (1) When only one ureter is completely blocked, the urine that passes is clear, of low specific gravity, and non-albuminous; and chronic uræmia ensues until the condition is relieved or the remaining kidney undergoes compensatory hypertrophy (see also Hydronephrosis, § 315). (2) When both ureters are blocked, a condition known as "*latent uræmia*" arises. The patient passes no urine for about a week, and may complain of nothing except slight drowsiness, but after eight or ten days he becomes restless, with contracted pupils, subnormal temperature, dry brown tongue, and muscular twitchings. In other cases vomiting may be so severe as to suggest the presence of intestinal obstruction. Death is usually sudden, after ten to fourteen days, the mind remaining clear to the end.

II. The causes of Non-Obstructive or True Suppression are: (i.) Acute nephritis, or the terminal stage of chronic nephritis (ten to twenty hours before death); (ii.) collapse (of which suppression is one of the symptoms)—*e.g.*, after abdominal operations or injuries, passage of a catheter, fevers, or local inflammations; (iii.) hysterical anuria; (iv.) acute lead, phosphorus, or turpentine poisoning; (v.) embolism or thrombosis of both renal arteries (very rare). Whichever of these causes is in operation, the *Symptoms* are: (1) any urine passed is high-coloured and concentrated (high specific gravity), and may contain albumen and casts (indicating that the suppression is due to renal disease); (2) there may be urgent vomiting, diarrhœa, and sweating. The other symptoms are those of acute uræmia (§ 270) and those of the cause.

Prognosis.—Suppression is a very serious symptom, though the gravity depends somewhat upon the cause. Of the *obstructive* forms, calculus blocking one ureter, the kidney of the opposite side being healthy, is perhaps the most favourable. If the obstruction affects both ureters and is not removed, death will occur in about eleven days after the obstruction began. In the *non-obstructive* forms death or partial recovery takes place in a few days.

Treatment.—Hot baths, pilocarpine, and other diaphoretics promote the action of the skin, and so relieve the toxæmia. Free purgation

promotes the excretion by another channel ; cupping, wet or dry, over the loins relieves the local congestion. For the treatment of obstructive suppression a surgeon should be called at once. Stone is usually impacted in the ureter at its entrance to the bladder.

The patient complains that his urine **dribbles away constantly**, *and on percussing over the pubes or passing a catheter, his bladder is found to be empty.* He has True Incontinence. *Or he complains that he has a frequent call to urination, and cannot always hold his water.* He has Active Incontinence.

§ **313. Incontinence of Urine** may be of two kinds, and it is best to speak of these as True Incontinence and Increased Frequency (Active Incontinence) respectively.

(*a*) True Incontinence, when the urine dribbles away involuntarily as fast as it is formed, must not be confused with *overflow* or *false incontinence*, which is due to the overflow of a distended bladder in *retention*. The latter is recognised by the percussion signs of a full bladder and by the relief afforded by the passage of a catheter. In true incontinence, which is relatively a rarer condition, the *Cause* is generally quite apparent, such as vesico-vaginal fistula, paralysis and dilatation of the sphincter after the operation of lithotrity, or the paralysis of the sphincter associated with various cerebro-spinal affections.

(*b*) Increased Frequency of Micturition, or, as it is (unfortunately) sometimes called, *Active Incontinence*, is a very common complaint. The patient can hold his water, but the calls to urinate are too frequent, and sometimes so urgent that a few drops dribble away before arrangements can be made. The normal time during which the urine can be retained differs in different individuals, and also according to the amount of fluid taken ; but four to five hours is a fair average. It is longer in the female than the male ; some women can retain the urine for ten or twelve hours. The habit is injurious, and is said to lead to flexions of the uterus.

Increased frequency is due to many *Causes*. The first point to determine is whether there is any marked increase in the diurnal quantity, as in diabetes or chronic granular kidney, because any of the causes of polyuria (§ 307) may be a cause of increased frequency of micturition. In young adults diabetes is perhaps the commonest, but in advancing years granular kidney and enlarged prostate are by far the most common causes. Indeed, our attention is often first drawn to the latter condition because the patient develops a habit of rising at night to pass water. It is not always easy to decide whether the quantity is increased or not, as the patient is apt to think that, because he passes water too often, he passes too much. But having as far as possible excluded polyuria, there remain three groups of causes to consider : 1. Some cause of *local irritation* is undoubtedly the most fre-

quent. The *urine* may be too acid. The *bladder* may be irritable, as from the presence of an enlarged prostate (the usual cause of abnormal frequency in old age), chronic cystitis, ulceration, tumour, stone (in the young) or pressure upon the viscus by a displaced uterus. Or the irritation may be in the *kidneys* from the presence of stone, tubercle, or other cause of pyelitis (§ 305). Or the irritation may be *reflex*, from disease in the vicinity of the bladder, worms, phimosis, or too long a prepuce (a very frequent cause of nocturnal incontinence in children), fissures, piles, prolapse or polypus of the rectum, vascular urethral caruncle (a cause frequently overlooked in women), pelvic inflammation, or varicocele. 2. *Constitutional* causes are occasionally associated with this condition, such as hysteria, sexual excesses, adenoid vegetations in the pharynx,[1] and other causes leading to deficient aeration of the blood. 3. A *congenital* want of development of the sphincter is sometimes present. True congenital cases are rare, and defective action of the sphincter is more frequently due, especially in women and children, to some of the reflex causes above mentioned, the habit persisting after the cause has been removed.

Nocturnal Incontinence in children is a troublesome condition often met with in private practice. In such cases we must first satisfy ourselves of the absence of any organic disease. Having done this it is well to remember in this condition that it may be associated with incipient insanity in childhood, general debility, stone in the bladder, and adenoid vegetations in the pharynx.[1] The last named, if severe, result in a deficient aeration of the blood and an unduly heavy sleep. The other causes mentioned above should also be remembered.

Both *Prognosis* and *Treatment* turn almost entirely upon the cause, and are hopeful in proportion as this is removable. The power of retention of the urine is a habit which can be cultivated in early life, and the relative frequency in different individuals varies a good deal with habits engendered in childhood. Careful local examination should always be made to exclude local causes. If the urine is acid, or the bladder irritable, much good may be done by the administration of alkalies and hyoscyamus. If the bladder is wanting in tone, belladonna and nux vomica are the two sovereign remedies. Tincture of rhus aromatica, ℥ v. to xv. has been found to be useful where no cause is obvious, and thyroid extract has been recently recommended. If there is irritability of the nervous system bromides are specially useful. Children of faulty habits may be treated by sleeping on hard mattresses, or by preventing them sleeping on the back by means of a reel of cotton fixed to the sacrum by plaster. They should be made to pass water before going to bed. Raising the foot of the bed and cold douching to the spine are recommended.

[1] Dr. Leonard Williams in the *Lancet*, May 1, 1909, has recorded a case made worse by removal of adenoids; he has moreover recorded twenty-three cases which were cured by the administration of thyroid extract (1½ grains thrice daily).

§ **314.** *The urine presents a* **cloudiness,** *due to some* CRYSTALLINE *or* OTHER DEPOSIT ; it may be URATES, URIC ACID, PHOSPHATES, OXALATES, or FAT, unless it be pus (§ 288), blood (§ 283), or bacteria (§ 288).

In **lithuria** *the urine,* CLEAR *when first passed, becomes cloudy, with a pinkish* AMORPHOUS DEPOSIT *when it gets cold ; the deposit dissolving again when heated in a tube.* The condition described as LITHÆMIA (the clinical condition associated with lithuria) is still by most believed to be due to functional derangement of the liver, and its symptoms are described in the disorders of that organ (§ 249). Various other conditions with which excess of urates and uric acid in the urine may be associated, as a more or less subordinate symptom, have already been referred to in § 293.

The clinical significance of uric acid and urates is still a subject of debate.

Phosphaturia *is usually indicated by cloudiness in a neutral or alkaline urine* (§§ 285 and 291). (1) Phosphates frequently occur in the urine in such quantity as to cause a turbidity even when *first passed.* They are apt to occur especially towards the end of micturition, not infrequently alarming the patient unnecessarily. Phosphates may be especially abundant in the "alkaline tide" of the early morning, or after dinner. There may be no symptoms, even when phosphates are passed in large quantities ; but more frequently phosphaturia is accompanied by chronic dyspepsia, or some condition in which the urine is alkaline. Phosphates are thought by some to be an evidence of excessive nerve waste ; I have seen several cases of phosphaturia in medical men who had recently undergone severe brain work and nerve strain.

(2) The name PHOSPHATIC DIABETES has been given to a condition where there are thirst, emaciation, aching pain in the loins and back, and an increase of phosphates in the urine, the diurnal quantity of which is greatly increased. The urine is alkaline or very feebly acid in reaction. In diabetes mellitus the phosphates vary inversely as the sugar. Phthisis may supervene, or the disease may pass into diabetes mellitus or diabetes insipidus, if unrelieved by treatment.

(3) There is an increase of phosphates in wasting diseases, in leukæmia, severe anæmias, and in convalescence from fevers.

(4) Phosphates are diminished in acute fevers, and in diseases of the kidney—*e.g.*, nephritis.

(5) *Stellar Phosphates* may indicate grave constitutional disturbance—*e.g.*, diabetes and cancer. *Triple Phosphates* found in freshly passed urine denote that decomposition is going on in the bladder, an indication of cystitis. It is liable to deposit within the bladder or to form stone.

The *Treatment* of "phosphatic diabetes" is by rest, warmth, and light nourishing food. Alcohol and coffee should be forbidden, as they promote diuresis. Codeia or opium should be administered until pain is abated ; when tonics, iron, quinine, nux vomica, and cod-liver oil should be freely given.

Oxaluria *is generally indicated by a "powdered wig" deposit on the top of the mucus which settles to the bottom* (§ 291). Transient oxaluria has no clinical significance except as indicating the *nature* of a stone, which has revealed its *presence* by other symptoms. It is also found after a diet of rhubarb, tomatoes, cabbage, or onions. But oxaluria is also connected with four other clinical conditions.

(i.) The *oxaluric diathesis* is a tendency which exists in some individuals to the excessive formation of oxalates in the urine. Certain cases have been recorded where the symptoms of rapid emaciation and pains in the loins and back were attended by an excess of oxalates in the urine.[1] (ii.) Other observers have con-

[1] Cantoni, "Oxalurie." German translation by Hahn ; Berlin, 1880 ; Begbie Schmidt's Jahrbh., lxvii., 52, 1850 ; and Jaksch, *op. cit.*, p. 307.

nected certain nervous symptoms, such as mental depression going on to neurasthenia and even melancholia, with this condition.[1] It is probable, however, that these symptoms are connected with the concurrent dyspepsia. (iii.) Oxaluria very often seems to be connected with dyspepsia. Urates are generally precipitated in the urine at the same time as the oxalates, and Sir Lauder Brunton has shown that the passage of sulphuretted hydrogen through a strong solution of urates gives rise to the formation of oxalates, by its reducing or deoxidising power. In intestinal dyspepsia a large quantity of this gas is formed in the intestines, and it seems probable that oxaluria in these cases may be caused by the deoxidising or reducing power of the H_2S upon the urates. (iv.) Oxalates are found in large excess in paroxysmal hæmoglobinuria (§ 302).

Fat may occur in the urine in chronic tubal nephritis attended by much fatty degeneration of the epithelium, and after fractures of the bones. It is found in great abundance in **Chyluria.** The presence of chyle in the urine gives to it a milky white appearance and the power of coagulating. Chyluria is not uncommon in the tropics, where it is due to the migration of the filaria sanguinis hominis from the lacteals into the urinary tract, the unnatural communications thus made leading to the paroxysmal appearance of chyle in the urine. The urine passed at night is the more completely white ; that passed by day may be mixed with blood. The embryos of this parasite are to be found in the urine with a few red and white blood-cells, albumen, fat and shreds of fibrin. However, other cases have been observed in persons who have never resided in the tropics and in whom no parasite can be found. The causation of such cases is obscure.

Prognosis.—The patient may live twenty years with but little impairment of health. In other cases, however, great debility and mental depression may be present.

Treatment.—Prevent the disease by boiling the drinking-water. Gallic acid is recommended. To meet the drain on the system give plenty of food.

§ 315. **Renal Tumours** may be of six kinds : (I.) HYDRONEPHROSIS ; (II.) PYONEPHROSIS ; (III.) PERINEPHRIC ABSCESS ; (IV.) MALIGNANT DISEASE ; (V.) CYSTIC DISEASE ; and (VI.) MOVABLE KIDNEY. The last-named comes under Abdominal Pain (§ 177), which is its chief symptom.

The *Physical Signs* common to all tumours of the kidney, and their diagnosis from other ABDOMINAL TUMOURS, are given in §§ 188 and 292.

I. **Hydronephrosis** is a term indicating a cystic tumour of the kidney, caused by the gradual obstruction of the urinary passages, and the consequent dilatation of the pelvis of the kidney.

The *Symptoms* by which this tumour is recognised are : (1) At intervals a large amount of urine passes, with concomitant reduction or even disappearance of the tumour. The urine is pale, clear, and of normal composition. (2) Constitutional and general symptoms may be absent. (3) Local pressure symptoms may arise, causing pain or disturbance of function of the neighbouring organs.

Etiology.—The causes of obstruction to the outflow of the urine may be (i.) *congenital* (contracted or twisted ureters) ; (ii.) *acquired* causes, which may occur (*a*) in the *urethra*, such as stricture or enlarged prostate (Fig. 91) ; (*b*) in the *ureter*, such as occur from stone or blood-clot ; pressure by pelvic or other tumours ; contraction after operation, injury, or disease of the ureter ; or kinking, as in movable kidney.

[1] Neidert, *Münchener Med. Wochenschr.*, xxxvii., 590, 1890.

These acquired causes give rise to a gradual obstruction, and when the obstruction is intermittent the tumour may become very large. It is then liable to be mistaken for an ovarian cyst, or even for ascites. In such cases a trocar may be introduced, and the fluid withdrawn would reveal an absence of the albumen which is always present in an ascitic fluid.

Prognosis.—If the condition is unilateral and intermittent it may cause little trouble, and may disappear after a duration of years. On the other hand, a double hydronephrosis is very serious, as it leads to uræmia. The complications are rupture into the peritoneum or pleura; the onset of suppuration in the pelvis of the kidney (pyonephrosis); or uræmia, due to atrophy of the substance of both kidneys.

Treatment.—If the tumour is intermittent, unilateral, and causing few symptoms, it is best to leave it alone. Osler recommends the use of a pad to retain the organ in place and prevent further dilatation.[1] If the tumour becomes very large, surgical treatment is advisable. In all cases the cause must be ascertained and, if possible, treated.

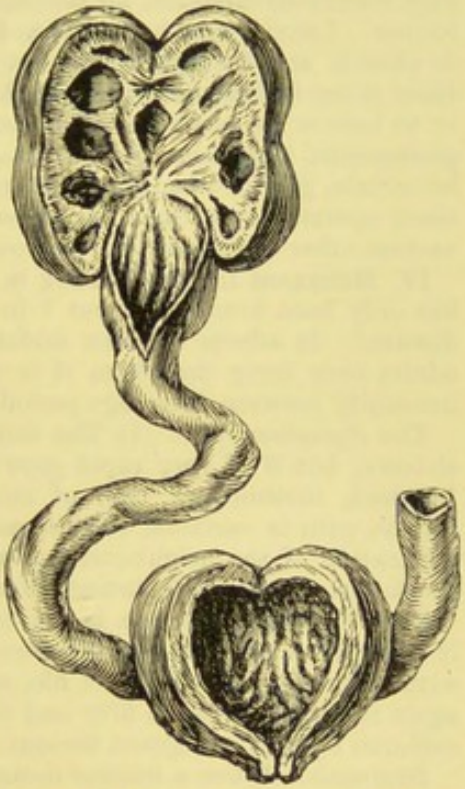


Fig. 91.—Results of URINARY OBSTRUCTION such as arise from enlarged prostate or stricture urethræ (Walsham's Surgery).

II. Pyonephrosis is a cystic tumour of the kidney due to distension of the pelvis and calices by fluid containing pus. It is consequent on obstruction to the free outlet of the urine in septic cases of pyelitis, or sepsis supervening on hydronephrosis.

The *Symptoms* are: (1) The tumour is tender to palpation; (2) symptoms of pyelitis are present—pyuria, intermittent pyrexia, sometimes rigors, and dull pain in the loin; (3) at intervals, when the obstruction is removed or diminished, the tumour may subside, coincident with the passage of a large quantity of pus in the urine.

The *Causes* are: (1) *pyelitis* (§ 305), with blocking, partial or complete, of the ureter; or (2) *hydronephrosis* (*vide* Causes of this above) becoming septic—*e.g.*, from extension upwards of cystitis.

Diagnosis.—(1) From *hydronephrosis*, which has no tenderness or fever; (2) from *perinephric abscess*, which has greater tenderness in the loin and a more superficial swelling, with local signs of abscess sooner or later.

Prognosis.—The condition is very grave. A tuberculous pyonephrosis may undergo cure by fibrosis; but in most cases the patient becomes worn out with long discharge, or develops amyloid disease, or a fatal issue is rapidly brought about by the tumour bursting into the abdomen or chest.

Treatment is mainly surgical, and nephrotomy is indicated. The cause must be treated medically.

[1] Professor William Osler, "Abdominal Tumours," 1894.

III. Perinephric Abscess is not very uncommon. It may arise by (i.) extension from kidney disease (pyelitis); (ii.) extension from a perityphlitic abscess; (iii.) extension from other organs—*e.g.*, abscess of the liver or Pott's disease of the spine; (iv.) after an injury. The *Symptoms* are: (1) dull, aching pain in the loin radiating down the leg; (2) deep-seated resistance in the hypochondrium in front, tender to pressure; (3) the temperature is continuous or pyæmic in acute cases with sudden onset, or intermittent in insidious cases; (4) the leg on the same side is kept flexed; (5) swelling, which appears late in the disorder, it is seen between the iliac crest and the last rib, and it may be fluctuant; (6) the urine may or may not be altered according to the cause, but traces of albumen are common. The *Diagnosis* is difficult in the early stage when pain alone is present, when it may readily be mistaken for *lumbago* or *spinal disease*, but there is no fever in the former. Later it may be mistaken for a *renal tumour*, but in a simple tumour fever is absent, and the leg would not be held constantly flexed. In *pyonephrosis* there is not such acute pain or tenderness. *Prognosis.*—The abscess tends to open or to burrow its way in various directions, into the alimentary or urinary canals, peritoneum, or pleura. *Treatment.*—In the early stages, before the diagnosis can be certain, give hot fomentations and opium for the pain; as soon as pus is recognised operative procedure is necessary. It may point in the lumbar region or various other directions, and burrow for a considerable distance.

IV. Malignant Disease starting in the Kidney is certainly a rare condition, as it has only been found in about 1 in 500 autopsies on persons dying of malignant disease.[1] It affects children under nine (in whom *sarcoma* chiefly occurs), and adults over forty (in whom it is usually *carcinoma*), there being a remarkable immunity between these age periods.[2]

The *Symptoms* are: (1) The tumour is rapidly growing, usually of firm consistence, but if of very rapid growth it may appear fluctuating; (2) hæmaturia, frequent, intermittent, and of moderate amount; (3) progressive emaciation; (4) the pain is variable, sometimes it is very severe, owing to pressure upon or infiltration of the neighbouring organs. Sometimes pain is entirely absent, and the tumour may have attained a very large size before any symptoms occur.

Causes.—Renal sarcoma is the commonest abdominal growth in children, and it is believed often to start before birth. According to Bland-Sutton, it is met with in the first five years of life, and then, after a period of immunity, is found again in people between fifty and sixty. In people over forty cancer is the most common form of malignant tumour.

Diagnosis.—When a tumour occurs in a movable kidney it is apt to be mistaken for *ovarian tumour* or *fibroid*, and vaginal examination is necessary to clear up the diagnosis. *Tuberculous* kidney in a child may present difficulty, but the pain is less, and pyuria is present rather than hæmaturia. *Pyonephrosis* is accompanied by fever, the swelling is fluctuant, and there is a history of pyuria. The diagnosis of malignant tumours is not usually difficult.

The *Prognosis* is very grave. If untreated death occurs in six to twelve months after detection of the growth, the cancer of adults being somewhat more slowly growing.

Treatment is usually too late; early excision gives the only chance of life. Medical treatment is entirely palliative.

V. Cystic Disease of the Kidneys is a rare condition, usually of congenital origin, in which both kidneys contain cysts of varying size and number.

Varieties.—(i.) The cystic kidney in its typical form is a mass of cysts, and is usually congenital; (ii.) cystic kidney may arise in connection with granular

[1] Discussion on Renal Tumours, Path. Soc. B. M. A., 1899.

[2] The solid tumours affecting the kidney consist of (*A*) *Connective tissue type*:—I. Simple or benign growths (fibroma, lipoma, angioma); II. Sarcoma, which is by far the commonest. (*B*) Growths of an *epithelial type*:—I. Adenomatous growths (simple adenoma, trabecular, and papilliform cystomata); II. True Carcinoma:—(1) glandular type; (2) malignant papilloma. (*C*) Adrenal inclusions. (*D*) Adrenal growths.—"Renal Growths," T. N. Kelynack, Manchester.

kidney ; in this variety the tumour is never so large as in the former ; (iii.) cystic formations may also be due to hydatid.

Symptoms.—(1) There is a swelling usually in both lumbar regions, of insidious growth, very hard at first, and later yielding. (2) The other symptoms are similar to those of chronic interstitial nephritis—the urine is abundant, pale, of low specific gravity, containing traces of albumen, and occasionally blood and casts. The heart becomes hypertrophied, and the pulse indicates high tension. The patient may have excellent health for many years, or may develop symptoms of chronic uræmia.

The *Diagnosis* may be difficult. When symptoms of granular kidney occur, together with a tumour in both renal regions, the condition may be diagnosed as Cystic Kidney. The tumours have to be diagnosed from other abdominal tumours (§ 188).

Causes.—The disease may occur in the fœtus. Patients with the above symptoms, however, are usually men over middle age. Out of twenty-one cases collected by Dr. W. H. Dickinson, eleven were over forty.

Treatment is similar to that of Bright's disease. Death may occur from uræmia or the same complications as those of interstitial nephritis.

CHAPTER XIV

DISEASES PECULIAR TO WOMEN

The symptoms and consequences which may arise from disorders of the female genito-urinary organs are very numerous and widespread. Indeed, there is hardly a physiological system which does not suffer when these organs become affected. It is perhaps on this account that they should receive more attention from the general physician than is the custom.

PART A. SYMPTOMATOLOGY.

§ **316.** The symptoms proper to these organs may be divided into *local* and *general*. The Local Symptoms are certain external conditions around the vaginal orifice, leucorrhœa (vaginal discharge), dysmenorrhœa (painful menstruation), menorrhagia (excessive menstruation), amenorrhœa (deficient menstruation), pain in and around the organs, various disorders of function (*e.g.*, dyspareunia and dysuria) and tumours of the uterus.

The General Symptoms consist of (1) malaise and general ill-health, which is often quite out of proportion to the amount of local mischief. A life of chronic invalidism not infrequently supervenes upon some chronic though slight derangement of the reproductive organs. This general weakness is specially apt to affect the nervous system, and one is sometimes tempted to credit the older authors who named hysteria on account of its supposed origin in the womb (ὑστερος). (2) "Dyspeptic" symptoms of a reflex kind are nearly always present, as in other disorders connected with the abdominal viscera. (3) Anæmia is another consequence, though this may be due in part to the confinement indoors, or to the "loss" in cases of excessive menstrual flow. (4) Various neuralgiæ and a general hypersensitiveness of the sensory and sensitive apparatus. A certain degree of this is normal during the menstrual periods, and as civilisation advances it seems as though this recurrent hypersensitiveness were increasing. But by degrees, especially in those who suffer from dysmenorrhœa, this undue generalised hyperæsthesia is prolonged into the intervals between the periods.

Case-taking in diseases of women differs somewhat from that given

in Chapter I. The following summary will form a guide to the principal questions to be answered as a matter of routine :

1. What is the leading symptom complained of by the patient ?

2. History—name, age, married or single. (*a*) If married, how long ? How many children ? Date of last confinement ? Any miscarriages ? Confinements easy or difficult ? How long in bed after the birth ?

(*b*) Menstruation—age at which it commenced ? (i.) Regular ? Twenty-eight day or thirty-day type ? Lasting—three, five, seven days ? (ii.) Blood coming in clots (means excess) ? (iii.) Painful or not ? Pain dated back to a particular time ? Pain in small of back, shooting down one or both legs, or in ovarian region ? Pain persistent or paroxsymal ? What relation to the flow ?

(*c*) Any intermenstrual discharge—duration ; quantity ; white, clear, or thick and yellow ; offensive ; or with débris and blood.

(*d*) Micturition—painful, dribbling, or too frequent. Condition of bowels—pain on defæcation ?

(*e*) Other physiological systems to be inquired into ; and whether general health has suffered.

PART B. PHYSICAL EXAMINATION.

§ 317. With the exception of certain circumstances, an abdominal and local examination should be a matter of routine in all gynæcological cases which are not on the surface obvious. There are four methods by which the female pelvic organs can be investigated.

(*a*) An External Examination of the abdomen—inspection, palpation, percussion, auscultation (§ 166).

(*b*) A Vulvo-vaginal Examination should not be undertaken without duly considering both the necessities of the case, and the feelings of the patient (p. 450). A right-handed couch is indispensable, so that the physician may be on his patient's right. The patient should lie on her left side, with left arm out behind her, and the legs both drawn up ; the light should come from the foot of the couch. Note by inspection the colour and condition of the vulva, hymen, urethral orifice, and the condition of the perineum, especially in women who have borne children, and then proceed to pass the finger gently. Some use the first, others the second finger ; it is useful to be able to use either hand, so that we may keep one hand for possibly septic cases alone. The finger-nails should be kept extremely short and smooth, both for the patient's comfort and for cleanliness.[1] As a lubricant for the finger some physicians like sanitas with vaseline (about 5 per cent.), others use carbolised glycerine (1 in 200). The finger is passed well in, and the condition of the vaginal walls noted ; the position and condition of the os, whether patulous and soft as in pregnancy, firm, granular, fissured, conical, etc. Note also any fixity of the uterus, and whether

[1] A story is told of the late Mr. Lawson Tait, whom a great many foreigners used to visit. One particularly insistent gentleman, who generally had long dirty nails, was always seeking to ascertain from him the secret of his success. Lawson Tait, who was not in the habit of measuring words, became somewhat annoyed at the insistence of his visitor, and one day in reply to the oft-repeated question, he said : "The secret of my success is that I keep my nails short and extremely clean."

there is an angle or dip between the cervix and body anteriorly or posteriorly such as occurs in flexions.

(*c*) It is very desirable to make a BIMANUAL EXAMINATION next in order. Instruct the patient to turn over on to her back, draw her right leg up, and, with the finger of the right hand still in the vagina, place the left hand firmly above the brim of the pelvis, so as to be able to manipulate the uterus between the two hands. In this way note the size, position, and mobility of the uterus, the presence or absence of tumours, displacements of the uterus, or pelvic swellings or exudations.

(*d*) Various INSTRUMENTS are of considerable aid.

1. The *Sound*—invented by the same brilliant genius that adapted chloroform for use in surgical operations (the late Professor Simpson, of Edinburgh)—is the most valuable of all instruments. Its use, however, is contra-indicated in (i.) pregnancy, (ii.) menstruation, (iii.) acute inflammation in the pelvis, (iv.) cancer, and (v.) it should never be passed before making a bimanual examination.

With the tip of the right forefinger against the os pass the sound along the palm of the right hand until it slides well into the cervix. Then by a gentle turn and by a very gentle pressure upwards the sound will pass upwards and forwards into the uterine cavity.

The uses of the sound are to discover: (1) the depth of the uterus, which is normally $2\frac{1}{2}$ inches, and the thickness of its wall; (2) the direction of the uterine cavity, whether retro- or ante-flexed, or pushed to one side by pelvic growths; (3) the state of the endometrium; (4) the size of the os; (5) the presence of tumours in the uterus.

2. *Vaginal Speculum.*—Before passing a vaginal speculum in single women the late Dr. Matthews Duncan advised that the consent of three persons should be obtained—(1) that of the patient; (2) that of her parent or guardian; and (3) that of the physician.

Many different specula are in use, as most gynæcologists of eminence invent a new one. Practically they are of three types. The Ferguson, which is a tube; the bivalve or trivalve, which consists of two or three limbs jointed together; and the duckbill, which consists of two separate pieces. The first is best for the examination of the os; the second for the examination of the walls of the vagina; and the third for operative measures. In passing it do not forget the vaginal canal is directed backwards and upwards, and less pain is produced by quick movements in the right direction than by slow bungling. Note the condition of the mucous membrane, and the character of any discharge. If it be desirable to make some application to the interior by means of a Playfair's probe, this should be done before withdrawing the speculum.

3. The *volsellum* is a hook for drawing down one or other lip of the os, which is desirable, for example, (i.) for the introduction of tents. It is also of use to examine (ii.) any catarrhal patch, (iii.) whether the uterus is freely movable, and (iv.) to palpate the posterior surface of the uterus. It is contra-indicated in those conditions in which the sound is contra-indicated, and also in tubal pregnancy.

DILATATION OF THE CERVIX may be done by two methods:

1. *Slow Method.*—Sea tangle, tupelo, or sponge tents are inserted into the os uteri, and left *in situ* for some hours. By the absorption of fluid they swell up and distend the cervical canal. This method is useful in nulliparous women or when the cervix is rigid.

2. *Rapid Method.*—Hegar's dilators are employed. There are heavy vulcanite or metal instruments of various sizes, which are useful in multipara when the cervix is soft—*e.g.*, after a confinement or abortion. Chloroform is desirable. Having inserted the duckbill speculum, fix the anterior lips of the cervix with the volsellum or ovum forceps, draw well down, and insert the dilators gradually one after the other until the cervix is large enough to examine the interior with the

finger. In this way one can (1) examine the state of the endometrium by the finger; (2) curette the interior if there is any granular endometritis; or (3) diagnose the nature of any growth present by a microscopic examination of the scraping (a procedure which should never be omitted), thus differentiating, for instance, between cancer and simple endometritis. Dilatation of the cervix is contra-indicated in tubal disease, possible pregnancy, or cancer of the cervix.

PART C. DISEASES OF WOMEN, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **318. Routine Procedure and Classification.**—Having ascertained the patient's principal or *Leading Symptom*, and the leading facts as to the *History*, acording to the scheme given in Part B, proceed, unless the nature of the case is not already apparent, to the *Physical Examination* (subject to the reservations mentioned in Part B).

Classification.—The diseases of the female reproductive organs may be arranged, like urinary disorders, under the various cardinal symptoms to which they give rise—viz. :

(*a*) Diseases of the vulva and external parts	§ 319
(*b*) Leucorrhœa	§ 320
(*c*) Dysmenorrhœa	§ 321
(*d*) Hæmorrhage	§ 322
(*e*) Amenorrhœa	§ 328
(*f*) Pelvic pain, acute (§ 331), chronic	§ 335
(*g*) Pelvic tumours	§ 336
(*h*) Painful sitting, dispareunia, dysuria, and other disorders of function	§ 340

§ **319. Diseases of the Vulva** are generally surgical, and can only be enumerated here.

(1) Vulvitis in children may be caused by the migration of round worms, by uncleanliness, debility, or bad habits. In adults it is generally accompanied by vaginitis (*q.v.*).

(2) Pruritus Vulvæ (itching) is sometimes a very troublesome condition. An examination should always be made to discover whether eczema, pediculi, or irritating discharges be present. If these be absent diabetes may be suspected.

(3) Eczema of the vulva is, in the author's experience, greatly on the increase, as a consequence, in his belief, of the modern fashion of wearing closed non-washable knickers.

(4) Caruncle is a minute red irritable papilloma situated usually just within the urethral orifice. It is a frequent cause of painful micturition, painful sitting, and painful coitus.

(5) Labial Thrombosis is readily recognised, and is a not infrequent condition in certain hyperinotic states.

(6) Abscess of the vulva sometimes follows the last named. Sometimes it occurs as an inflammation of Bartholin's gland.

(7) Noma, Diphtheria, Chancres, Condylomata, Ulcers (simple or malignant) also affect the part.

In the *Treatment* of vulval conditions cleanliness is essential, and on

the whole the lack of this is one of the most frequent causes of vulvitis. It is surprising what little attention is paid to this matter, as is shown by the immense quantities of epithelial cells which are habitually found in the urine. Any eczematous or local condition must be treated as elsewhere. Caruncle is best treated by strong nitric acid or Paquelin's cautery. Labial thrombosis requires rest. Pruritus vulvæ may in my experience often be cured by large doses of calcium chloride. Cases which have long resisted other treatment have yielded to this.[1] Locally, lotio calaminæ co. in weak carbolic acid solution, liq. carbonis detergens, and sodium bicarbonate and borax solution are employed in varying conditions. In cases where the itching is very intense, a solution of nitrate of silver (20 grains to the ounce) may be painted on, the parts having been first anæsthetised by the application of cocaine solution. At the same time internal remedies, such as arsenic, quinine, bitter tonics, and calcium chloride should be persisted in.

§ **320. Leucorrhœa** is any white or whitish discharge from the vulval orifice. It is colloquially known as the "whites." There are three pathological causes of leucorrhœa—vaginitis, endocervicitis, and endometritis. When it comes from the *vagina* it is a thick, opaque white fluid, containing flocculi. If it comes from the *cervix* it has a more glairy and tenacious character, but is fairly constant, resembling in that respect the vaginal discharge. But if it come from the body of the *uterus* it is "curdy," and comes in gushes when the patient sits up or moves about. Examination by the speculum settles its origin, and is generally necessary for correct diagnosis.

(*A*) Leucorrhœa of Vaginal Origin arises when there is vaginitis from any cause, either acute or chronic.

(*a*) In Acute Vaginitis the discharge is profuse, yellow or greenish, and sometimes blood-stained, attended by dysuria and local signs of inflammation. The chief *Causes* of acute vaginitis are: (1) Traumatism, due to pins, peas, and worms in children, or in the adult an irritant pessary, or other foreign body (such as a letter), too powerful injections, or excessive coitus; (2) gonorrhœa, which is hard to diagnose from non-specific acute vaginitis except by its severity and history; and (3) spread from adjacent parts. A severe acute vaginitis is probably of gonorrhœal origin,[2] and the danger of this rests in the liability to endometritis, pyosalpinx, peri- or para-metritis, cystitis, and ascending pyelitis. The *Treatment* consists of rest, saline purges with hyoscyamus to allay the pain, copious warm drinks, hot hip-baths, and douches of weak carbolic, potassium permanganate, 10 grains to the pint), or corrosive sublimate, and after a few days some astringent lotion such as sulphocarbolate of zinc (2 drachms to the pint), glycerine of

[1] The *Lancet*, August 1, 1896.

[2] The b. coli, and possibly other pyogenic infections, may also cause acute vaginitis.

subacetate of lead (4 drachms to the pint), alum, or tannic acid (1 drachm to the pint).

β. In Chronic Vaginitis there is a thick continuous *opaque white* discharge, with local signs of inflammation. The *Causes* are (1) antecedent acute vaginitis ; (2) various constitutional conditions, such as general debility, strumous (*i.e.*, tuberculous) diathesis, diabetes, old age, alcoholism, anæmia, syphilis, rheumatism, and convalescence from fevers ; (3) new growths in the vaginal walls, such as epithelioma ; (4) irritant foreign bodies and other causes mentioned under acute vaginitis. The *Treatment* consists of tonics and other remedies for any constitutional disease present, combined with warm douches (100° F.), containing sulphate of zinc or sulphate of copper, or weak chloride of zinc, or the remedies mentioned under Acute Vaginitis. Local applications are made with Ferguson's speculum, and a cotton swab dipped in silver nitrate solution (5 per cent.) or corrosive sublimate (1 to 1,000), especially in gonorrhœal cases.

(*B*) Leucorrhœa of Uterine Origin may be due to endocervicitis, endometritis, subinvolution, fibroids or polypi of the uterus, cancer of the uterus (see Hæmorrhage), concurrent peri- or para-metritis (see Pelvic Pain), and, lastly, to various constitutional causes such as the gout, rheumatism, anæmia, syphilis, or tuberculosis.

I. In Endocervicitis (Chronic Cervical Catarrh), or inflammation of the cervix, the discharge is more or less constant, and consists of *glairy material* like white of egg. The other symptoms are : (1) The cervix is swollen, hard, and may present retention cysts ; (2) perhaps presenting catarrhal patches which bleed readily on touching ; and (3) menorrhagia or dysmenorrhœa is frequently present. Endocervicitis may have to be *diagnosed* from *cancer* of the cervix. Here the age is not much guide, as cancer of the cervix may come on in a patient as young as twenty-six. In cancer there is a shorter history, and it soon breaks down, is friable to the touch, and there is often a slightly blood-stained discharge. Microscopic examination of scrapings will aid the diagnosis. When fixity of the uterus and cachexia appear in due course, the diagnosis is simple. *Causes* and *Treatment* below.

II. In Endometritis, or inflammation of the body of the uterus, the discharge comes in gushes when the patient rises or walks about ; and it has usually a *curdy character*. Endometritis is generally accompanied by both menorrhagia and dysmenorrhœa, and is often associated with displacements of the uterus. Bimanually the uterus is slightly enlarged and tender, and on passing the sound the interior is tender to touch ; it readily bleeds, and irregularities of the surface may be felt. Sometimes there is a history of recurring abortions or of sterility.

Causes of endocervicitis and endometritis : (1) Cold during menstruation ; (2) extension upwards of vaginitis ; (3) retained fœtal products after labour or abortion ; (4) subinvolution ; (5) flexions and

versions; (6) instruments causing local injury or sepsis; (7) intra-uterine growths; (8) acute specific fevers; and finally it may come on insidiously without any definite cause, especially in old age (senile endometritis.)

Diagnosis.—Occurring in middle or advanced life endometritis and endocervicitis may have to be diagnosed from *cancer of the body* of the uterus by its more prolonged history, by there being less cachexia, and by the relative absence of those three pathognomonic features of cancer —pain, profuse hæmorrhages, and offensive discharge. Of all the local signs of cancer, fixity of the uterus is the most certain, and the diagnosis may be aided by curetting and examining with the microscope.

Treatment.—Both diseases call for a certain amount of hygienic and general treatment, especially if there has been much menorrhagia. Local douches and astringents are of very little use, and there are thus two methods of local treatment: (1) The application of strong carbolic or other corrosive to the interior by means of Playfair's probes; (2) dilating the cervix and curetting the interior. Endocervicitis is treated by applications of silver nitrate or copper sulphate (gr. xl.—℈i.) applied by a Playfair's probe, passed through a Ferguson speculum to protect the vaginal wall; or by cataphoresis (see Dr. S. Sloan, the *Lancet*, July, 1909). Hot douches are used twice or thrice daily (never less than a quart at a time); and tampons of ichthyol (10 per cent. in glycerine) are inserted after the douches or applications. Constipation must be avoided.

§ **321. Dysmenorrhœa** is pain during the menstrual period. There are three varieties: (I.) Neuralgic or Spasmodic, in which the pain is paroxysmal, situated chiefly in the hypogastrium, begins a few hours before the flow, and lasts for one to two days; (II.) Inflammatory, in which the pain is dull, aching, persistent, situated sometimes in the small of the back and down the legs, begins several days before the flow and is relieved by the flow, especially when it is profuse; and (III.) Membranous, in which the pain is severe, paroxysmal, and relieved as soon as the membrane is passed. In the *first* named, local examination reveals nothing wrong in the uterus or its appendages, and the menstrual flow is usually natural. In the *second* variety, examination generally reveals some abnormality in the uterus or its appendages—*e.g.*, endometritis, fibroids, adhesive bands, ovaritis; and it not infrequently dates from a confinement or abortion. The *third* is diagnosed by the passage of a membrane, and is distinguished from abortion in that it is passed every month.

Causes.—The causes of neuralgic dysmenorrhœa are of a general character, like those which lead to neuralgia in other parts. The causes of varieties II. and III. are such as lead to inflammation of the uterus or its appendages. All three varieties have been variously ascribed to

obstruction of the flow by flexions of the uterus, or by constriction of the cervix, or to the undue excitation of uterine contractions.

Treatment.—I. The neuralgic form usually calls for general treatment—hygienic, dietetic, and tonic. Treatment directed to the diathesis, as in rheumatic persons, may effect a cure. It is very important in this and in the other varieties to avoid constipation. Warm baths, and especially Turkish baths, are very valuable in my experience. Remedial treatment at the time of the period consists of hot bottles to the hypogastrium, hot drinks, feet in hot water, cannabis indica, belladonna, camphor, sal volatile, bromides, castor, antipyrin, and morphia (with great caution). Dr. Champneys recommends tr. castoreum (℥ xx.). In obstinate cases dilatation of the cervix often lessens the severity of the attacks.

II. The inflammatory form admits of the same symptomatic treatment as the foregoing. The remedial treatment should be directed to the inflammatory lesion which is the causal agent. Depletory methods, such as a glycerine tampon, leeches, or scarification of the cervix, combined with a saline purge, are indicated. Very hot douches (110° F. for ten minutes) should be given twice daily. In severe cases removal of the appendages has been adopted.

III. For the membranous form the symptomatic treatment is as above, with the subsequent dilatation and curetting of the interior during the interval.

§ 322. **Hæmorrhage.**—*Menorrhagia* indicates an excessive flow at the monthly period ; *Metrorrhagia* indicates irregular hæmorrhage from the uterus, irrespective of the period. It is difficult to separate these two symptoms, as their causes are more or less identical, and they very often occur together.

Hæmorrhage of this kind arising in women *under* thirty may be due to the following causes in order of frequency : Endometritis, constitutional conditions, fibroids and polypi of the uterus, pelvic inflammations, subinvolution of the uterus, retroverted uterus incarcerated in Douglas' pouch, ovarian tumours (occasionally), inversion of the uterus, and extra-uterine fœtation. Flexions and versions of the uterus are said to cause no symptoms unless attended by pelvic inflammation or adhesions.

In women *over* thirty the above causes also may give rise to hæmorrhage, but in addition there are two other conditions which may be causally associated—viz., (1) the Menopause and (2) Malignant Disease. The sudden supervention of *metrorrhagia* with *acute pain* should always suggest a miscarriage or an extra-uterine fœtation (§ 329).

In women *past the menopause* some gross lesion of the uterus, especially Cancer or Uterine Fibroid, is nearly always present.

Many of these conditions are dealt with elsewhere, but menorrhagia

or metrorrhagia is the chief symptom referable to the reproductive organs in : (I.) Certain Constitutional conditions ; (II.) Uterine Fibroid or Polypus ; (III.) Subinvolution (in persons under thirty) ; (IV.) the Menopause ; and (V.) Malignant Disease (in persons over thirty). These conditions will therefore be differentiated here.

§ **323.** Hæmorrhage may depend upon certain CONSTITUTIONAL CONDITIONS. (1) Certain women of a plethoric habit of body, usually with florid countenances, may be troubled with too profuse periods all their lives, and a tendency to excessive flow on any trivial exciting cause. (2) Prolonged lactation or too many and too frequent pregnancies ; (3) residence in tropical climates ; and (4) mental over-work, especially if combined with a sedentary life, are said to produce it. (5) The vague condition we call hysteria, especially in that variety which is subject to flush storms, is frequently attended by menorrhagia. (6) The congestion in the circulation which attends some heart and liver diseases finds more or less relief in this way. The differential characters of the bleeding due to these causes are : (1) The menstruation may occur every third or second week, or even weekly, though in point of quantity it may or may not be increased. The flow, moreover, may be very readily excited, as by a hot bath, or after a day of unusual exercise. (2) The general symptoms after a time point to anæmia, combined with the symptoms of the constitutional cause in operation.

§ **324.** Hæmorrhage may, secondly, be due to a UTERINE FIBROID. The symptoms vary with the position of the tumour. These tumours may be submucous, interstitial, or subserous. When the fibroid is submucous or interstitial, the symptoms of uterine fibroid are (1) menorrhagia and metrorrhagia. (2) Leucorrhœa and sometimes dysmenorrhœa are present. (3) On examination with the sound the uterine cavity is found to be enlarged ; and (4) on bimanual examination enlargement of the uterus, which is usually hard and bossed from the presence of more than one fibroid, can be detected. There is a tendency for the submucous variety to become polypoid, remaining attached to the uterus by a pedicle. The *subserous* fibroid may present no symptoms at all for many years, and may even then be discovered by accident. Amenorrhœa may accompany such cases quite as often as menorrhagia, and the latter is never profuse. In short, pressure symptoms may be the earliest indication of a subserous fibroid. In uterine fibroids of all kinds the rate of growth, though it varies somewhat, is nearly always very slow ; but as the tumour increases we get symptoms of pressure upon the surrounding organs, such as frequent micturition, varicose veins, neuralgia in legs and back, indigestion, difficult respiration, or hydronephrosis.

UTERINE POLYPUS is another cause of irregular hæmorrhage. They are of three kinds. The most common forms are (1) fibroid polypi

(which result from submucous fibroids), and (2) mucous polypi. (3) Placental and fibrinous polypi occur, the first after labour or abortion, arising from retained portions of the placenta, the second from the stump of a growth previously removed.

Polypi present at first most of the symptoms of submucous fibroid. When very small they can be made out with certainty only by dilating the os and exploring the interior. Later on the polypus may be seen hanging from the os into the vagina on examination with the speculum. After a time it may slough, and cause an offensive discharge.

§ **325.** Subinvolution, or the non-return of the uterus to its normal size, is a very frequent cause of menorrhagia after labour or abortion. After a confinement the uterus begins to diminish in size, and at the end of about two months resumes its normal length of $2\frac{1}{2}$ inches. In cases of subinvolution we find (1) on the passage of a sound that the uterus is enlarged ; (2) it tends in most cases to be retroverted and lower than normal ; (3) the patient generally complains of backache, bearing-down pain, and leucorrhœa ; and (4) lassitude, weakness, and general malaise are usually present.

The *Causes* of subinvolution are important : (1) Getting up too soon after childbirth is probably the reason why this condition occurs so frequently among the poor ; (2) retained membranes or portions of placenta ; (3) pelvic inflammation ; (4) delayed labour or over-distension of the uterus ; and (5) the practice of not suckling the infant, account for this not infrequent condition, and therefore it is more often met with in those who have had numerous and rapid pregnancies.

§ **326.** The Menopause, or climacteric, is the epoch at which the sexual activity of the female undergoes involution, when the menses, which are the sign of that activity, cease. This may take place in three ways : (*a*) They may cease gradually, and more or less irregularly ; (*b*) quite suddenly ; (*c*) they may be attended by a series of hæmorrhages. The last method, which is quite as frequent as either of the other two, is the one with which we are now concerned.

The existence of this cause of menorrhagia or metrorrhagia can only be recognised by the attendant phenomena. (1) The age of the patient varies considerably between thirty-five and fifty-five, the average being about forty-five. (2) The occurrence of " flush storms," which consist of a hot stage, a cold stage, with or without shivering, and sometimes a stage of perspiration. (3) The other nervous phenomena which may occur at this time are extremely varied. There is generally an irritability and restlessness, and generally also a marked tendency to depression of spirits, causing the patient to burst into tears at the slightest provocation. This may amount to definite melancholia, especially when there is mental heredity. Sexual perversions, with a marked tendency to excess of all kinds, are apt to occur. (4) While fibroids and other gross lesions may undergo involution at this epoch, carcinoma, if

there be a predisposition, may make its appearance, and the case should be carefully watched from this point of view.

§ **327.** Malignant Disease of the uterus is clinically met with in four forms: (*a*) Cancer of the cervix, chiefly met with in multiparæ, between the ages of twenty-five and seventy; (*b*) cancer of the body, which is chiefly met with in sterile women, between the ages of fifty and sixty; (*c*) sarcoma of the uterus, which is rare, unless we include under that term certain fibroids which appear to take on the malignant features of spindle-celled or large round-celled sarcoma; and (*d*) deciduoma malignum, a very rare form following parturition.

The symptoms differ in the first three varieties. (*a*) Cancer of the Cervix is an epitheliomatous growth, which usually runs a somewhat rapid course. (1) On digital examination the os has a friable granular feel, which is so characteristic that this feature and the blood-stained discharge upon the finger are alone, in experienced hands, sufficient to diagnose the disease. (2) In some cases examination reveals a mushroom-like growth ("cauliflower excrescence") hanging down into the vagina, readily breaking down and readily bleeding. It has a tendency to spread to the vaginal wall, to the utero-sacral ligaments, broad ligaments, and body of the uterus, leading to a fixity of the uterus and hardness which is easily made out on palpation. (3) Metrorrhagia and menorrhagia are present. (4) In the intervals between the marked hæmorrhages there is a continuous leucorrhœa of pinkish-brown colour, often of a very offensive odour. (5) Local pain is usually a late symptom, but, like the wasting and the cachexia, is sure to supervene sooner or later.

(*b*) Cancer of the Body of the uterus is chiefly met with in sterile women over fifty years of age. The symptoms are: (1) Metrorrhagia, and in the intervals pinkish brain-like matter is discharged, which has a very offensive odour; (2) pain is an early symptom, and very severe; (3) on bimanual examination the uterus is found to be enlarged. Later on, as the disease extends to the broad ligaments, the uterus becomes fixed. This fixity to the educated finger is very characteristic of the disease. On the passage of a sound considerable hæmorrhage may take place. (4) The cachexia and other general symptoms resemble those of cancer elsewhere.

(*c*) Sarcoma of the Uterus is a relatively rare condition. Its symptoms do not differ materially from those of uterine fibroid, except in the rapidity with which the case progresses, and the liability to deposits elsewhere.

Prognosis of Hæmorrhage.—Uterine bleeding of itself is not fatal to life, but some forms are very intractable, and lead to considerable anæmia, debility, discomfort, and inability to fulfil the duties of life. (1) The undue bleeding at the menopause and of subinvolution tends to spontaneous recovery, and that which is due to constitutional conditions is usually amenable to treatment; so also, in many cases,

is that due to PELVIC INFLAMMATION. (2) ENDOMETRITIS is perhaps one of the most intractable of the causes, though this also is remediable by local treatment. (3) The prognosis in a case of FIBROID tumour depends very much upon its position. The submucous varieties (and mucous polypi, § 324) are readily treated, but if neglected these may slough, and produce death by exhaustion and septic intoxication. The subserous form is next in order of gravity, though this may give but little trouble for a great many years. The interstitial form is the most serious, and if there be much loss of blood and consequent prostration the patient can only live a life of invalidism. When of large size these tumours are very difficult to treat. Fibroids, when occurring near the menopause, should not be treated surgically, because they may spontaneously disappear. If the patient be not near the climacteric, and if the symptoms both general and local are serious, the question of extirpation of the uterus is to be considered, and it is now done with comparative safety. Dr. Gow[1] has published some very successful statistics of the operation, which compare very favourably with those of ovariotomy and other major operations. (4) CANCER is the most serious of all the causes of hæmorrhage, and if untreated tends in the course of one or two years to terminate in death from exhaustion, hæmorrhage, peritonitis, or uræmia by involvement of the ureters. The chance of recovery depends upon the diagnosis of the disease and its treatment surgically *at an early stage*. If cancer of the cervix is discovered before it has spread to the parts around, or if cancer of the body is taken in hand while the uterus is still freely movable, amputation of the one or extirpation of the other offers a fair prospect of recovery.

Treatment of Hæmorrhage.—(*a*) Symptomatic, in all forms. To relieve the hæmorrhage calcium chloride in large doses (20 grains or more) is the most recent and most popular of remedies, as it promotes the coagulability of the blood. Ergot, adrenalin, dilute sulphuric acid, tinctura hydrastis, tinctura hamamelidis, hot douches, tonics, chloride of iron, quinine, nux vomica, are all useful. If the hæmorrhage is alarming (*e.g.*, in a large fibroid) the uterus must be plugged with iodoform gauze after dilating the cervix up to a No. 7 Hegar dilator (§ 317). Leave the plug in for twenty-four hours only, then explore the interior. (*b*) Remedial treatment is directed to the cause, and must be adopted in addition to the foregoing. (*c*) In all cases general measures are required—the food must be nourishing, exercise must be avoided near the period, and the patient must rest in bed while the flow is profuse. While strong purgatives on the one hand must be avoided, it is extremely important, on the other hand, to avoid constipation. For the menorrhagia of the menopause bromides and calcium chloride are the best remedies, and these also relieve the attendant discomforts.

[1] *Lancet*, December, 1899, Med. Soc. Report.

§ **328. Amenorrhœa** is that condition in which the catamenia are either deficient or absent. The term *primary* amenorrhœa is applied to the condition in which menstruation has never occurred, as in rare cases where the patient is of a masculine type,where there is a congenital absence of the organs concerned in the function, and also in cases of infantile uterus and undeveloped ovaries. *Apparent* amenorrhœa is that form in which there is a feeling of fulness in the breasts and abdomen every month, but the menstrual flow is restrained behind an imperforate hymen, an occluded os or vagina. In *secondary* amenorrhœa the flow, after having been once established, ceases or becomes deficient for a time. *Physiological* amenorrhœa is the cessation of the menses which occurs in pregnancy, a fact which must always be borne in mind even amongst the most irreproachable patients.

In PREGNANCY, the physiological cause of amenorrhœa, (*a*) the *General Symptoms* are as follows : (1) Morning sickness is usually one of the earliest, coming on about the first or second, and ceasing at the fourth month ; (2) the mammæ present a dark areola around the nipple, they become enlarged and after the third month contain milk. (*b*) The *Local Signs* are : (1) On digital examination there is *a softness* of the os which is unmistakable to the educated finger ; (2) a gradual increase in the bulk of the uterus commences at once. These are the earlier symptoms. About the third or fourth month we have a series of unmistakable signs—viz., (3) about the eighteenth week fœtal movements can be felt by the physician, and (4) about the eighteenth week the fœtal heart-sounds (at the rate of 120 to 150 a minute) can be heard on auscultation, usually midway between the umbilicus and left anterior superior spine ; and (5) ballottement can be made out about the fifth or sixth month.

The *Causes* of SECONDARY AMENORRHŒA may be divided into constitutional and local causes. (*a*) *Constitutional* causes are by far the most frequent, especially anæmia, or chlorosis, and phthisis. It also occurs after severe illnesses, on account of some great grief (mental shock), and during prolonged lactation. (*b*) The most important of the *local* causes is perhaps an ovarian tumour, in which the state of the catamenia varies, but usually the flow is absent or irregular. Other causes are a chill during menstruation, inflammatory conditions in the pelvis, superinvolution of the uterus, and extra-uterine fœtation.

Treatment in constitutional causes consists in plenty of fresh air, exercise, good food, and general healthy living combined with iron tonics. Warm baths, especially warm hip-baths at the expected time, are useful. It is very important to keep the bowels regularly acting, and the old-fashioned remedy of the aloes and iron pill is most beneficial. Permanganate of potash in 2-grain pills has been recommended. It is always advisable to adopt the tonic treatment in young unmarried girls, and it is only after these have failed that

local causes should be suspected, or at any rate locally investigated (compare § 317).

Sudden Suppression of the catamenia is a form of amenorrhœa which requires special treatment. The flow has probably come on normally, and then suddenly ceased on the second or third day, and the patient suffers a good deal of general discomfort. In such cases the patient should put her feet in hot water or a mustard bath, or sit in a warm hip-bath, and then should get into a thoroughly warm bed with hot bottles and take hot drinks. Subsequently saline purgatives in constant small doses, and general attention to the health are indicated. When the time of the expected period again comes round, the procedure just mentioned should be adopted.

§ 329. **Extra-uterine Pregnancy** (or Fœtation) may become manifest by menorrhagia, metrorrhagia, or amenorrhœa. The term is applied to the condition where pregnancy takes place outside the uterus, generally in the Fallopian tube.[1] The tube usually ruptures at the second or third month after fertilisation, either into the broad ligament (extra-peritoneally) or into the peritoneal cavity.

Symptoms.—(1) In many cases paroxysmal pains are experienced in one iliac fossa ; (2) there is a history of amenorrhœa for some weeks or a month or two over time, followed in most cases by a history of irregular hæmorrhages from the uterus. A membrane or cast may be discharged from the interior of the uterus at the same time. (3) Other symptoms of early pregnancy, such as morning sickness, are present. (4) On bimanual examination a swelling is found in the fornix. In most cases, however, none of the above symptoms may be noticed by the patient, and advice may not be sought until the time of rupture of the tube, when the patient consults us for *severe pain* and *hæmorrhage*. Extra-peritoneal rupture is attended and followed by the symptoms of pelvic hæmatocele (§ 334) ; intra-peritoneal rupture by the symptoms of perforative peritonitis (§ 169). If the rupture takes place about the fourth week the shock is not so severe, and the hæmatocele often remains extra-peritoneal.

Causes.—It usually occurs in women over thirty, who have been sterile or have had no children for several years. Mr. Lawson Tait held the theory that after salpingitis the cilia of the tube were destroyed, and so led to fertilisation taking place in the tube instead of in the uterus.

Diagnosis.—The diagnosis is often extremely difficult, and as above mentioned, the patient may complain of no symptoms until rupture occurs. The symptoms which should be inquired for are sudden amenorrhœa, early pregnancy symptoms, irregular hæmorrhages, and pains in one iliac fossa. If, with these symptoms, the os uteri is found to be patulous and soft, and the uterus slightly enlarged, it is probable that the case is one of extra-uterine pregnancy.

Prognosis.—The condition is extremely serious ; 70 per cent. die if untreated. (*a*) An *extra-peritoneal* rupture is not so immediately serious, since bleeding is checked in the limited space formed by the layers of the broad ligament. If untouched, in favourable cases the fœtus may even live till full term, the patient going through a spurious labour, after which the liquor amnii and placenta are absorbed, and the fœtus becomes mummified, remaining *in situ* without causing any symptoms, and only discovered accidentally on *post-mortem* examination. (*b*) In less favourable (though, unfortunately, more common) cases, rupture into the abdominal cavity takes place (*intra-peritoneally*) giving rise to alarming and even fatal hæmorrhage. In rare cases the fœtus may live in the peritoneum till full term, and be delivered by abdominal section. In some cases of variety (*a*) a *secondary* rupture into the peritoneum occurs, and the case then runs the course described in (*b*).

Treatment.—The treatment of extra-uterine pregnancy may be divided into three stages : (1) If diagnosed before rupture occurs, laparotomy must be performed

[1] The question of ovarian pregnancy (*i.e.*, fecundation of the ovum *in situ*) is still *sub judice*.

and the tube removed ; (2) if diagnosed after intra-peritoneal rupture, the same procedure must be adopted ; (3) if an extra-peritoneal rupture has occurred, treat by rest, as in hæmatocele.

§ **330. Pelvic Pain.**—Pain in and about the pelvis is one of the commonest symptoms of disorder of the female reproductive organs. "Bearing down" is often spoken of ; and "backache" or pain over the sacrum is so constant a feature of uterine disorders that it has come to have that association in the minds of the laity. The position and character of pelvic pain vary with the different maladies, but its degree is largely influenced by the temperament of the patient. Reference has already been made to painful menstrual periods (dysmenorrhœa), but the causes of a continuous pain (without reference to the menstrual period), such as that now in question, may be conveniently grouped into (*a*) those pains which come on more or less suddenly (acute conditions), and (*b*) those which come on more or less insidiously (chronic conditions). It must, however, be remembered that no hard and fast rule can be laid down in this respect.

(*a*) *The* **pelvic pain** *came on* **acutely** *and recently ; it is accompanied by more or less* CONSTITUTIONAL DISTURBANCE—PERI- OR PARA-METRITIS, INFLAMMATION of the UTERINE APPENDAGES, PELVIC HÆMATOCELE, ACUTE CYSTITIS, or some other INFLAMMATORY CONDITION within the pelvis, may be suspected, and the reader should first turn to § 331.

If the PAIN *has come on* VERY SUDDENLY *with* FAINTNESS *and* NAUSEA—turn first to PELVIC HÆMATOCELE, § 334 ; *or if it be accompanied by* METRORRHAGIA, it is suggestive of MISCARRIAGE, or EXTRA-UTERINE FŒTATION, § 329.

§ **331. Perimetritis (Pelvic Peritonitis),** which is one of the most frequent causes of pain, is an inflammatory condition affecting the peritoneal surfaces around the uterus and its appendages. Exudation may be present, and in chronic cases the adhesions lead to a matting together of the pelvic viscera.

The *Symptoms* of ACUTE PERIMETRITIS are (1) acute pain across the lower part of the abdomen ; (2) the abdomen is distended and tender to palpation, and a suprapubic mass of matted intestine may be felt. The patient lies on the back with legs drawn up ; (3) on examination, the vagina is found to be extremely tender ; (4) on vaginal examination forty-eight hours later the uterus is found to be fixed, with a certain amount of exudation surrounding it, which may be so great as to push the uterus forwards ; (5) the general symptoms consist of high fever and quick pulse, with vomiting.

In CHRONIC PERIMETRITIS (1) the pain is felt across the lower part of the abdomen, and is often greater on one side ; backache is usually present. The pain is constant, of a bearing down character, worse at the menstrual period ; (2) dysmenorrhœa and sometimes symptoms of endometritis accompany it ; (3) on examination the mobility of the

uterus is found to be diminished, and thickenings, *chiefly in the posterior fornix*, can be felt behind the uterus, in which situation a kind of "roof" to the vagina exists. (4) The general symptoms consist of an inability to stand or to walk for any length of time; and in severe cases chronic invalidism with mental depression or hysteria results.

Causes.—(1) Inflammation of the vagina or endometrium extending up by way of the Fallopian tubes is a common cause of pelvic peritonitis; thus, after confinement or abortion, acute pelvic peritonitis is often caused by this extension of inflammation. (2) Menstrual regurgitation, or a chill during the menstrual period, may give rise to pelvic peritonitis. (3) Chronic pelvic peritonitis may be set up by ovarian tumours, fibroids, cancer, or tubercle. *Prognosis* and *Treatment* below.

§ **332. Parametritis (Pelvic Cellulitis)**, another cause of pelvic pain, is an inflammation originating in the connective tissue of the pelvis adjacent to the uterus. This also may be acute or chronic.

The symptoms of Acute Parametritis are (1) pain across the lower part of the abdomen, usually *shooting down one leg;* and the patient usually lies with *one leg drawn up to relieve the pain.* In a few cases no pain is complained of at first. (2) On examination swelling and tenderness are made out in *one of the lateral fornices*, or one postero-lateral quarter of the pelvis. No swelling is felt in Douglas' pouch (the posterior fornix) unless pelvic peritonitis is also present. (3) The general symptoms are those of fever, generally of a hectic type, with quick pulse; in those cases where no pain is felt attention is drawn to the condition by the rise of the patient's temperature.

In Chronic Parametritis there are backache, dysmenorrhœa, frequently uterine displacement (due to the contraction of the inflammatory tissue), and symptoms of endometritis. When it results in suppuration or "phlegmon" the pus may make its way in various directions upwards or downwards.

Causes.—Parametritis usually follows labour or abortion in which injury to the cervix, vagina, or perineum has occurred, with consequent *entrance of septic matter.* Injury by septic instruments may also produce it, and thus clumsy attempts at procuring abortion form a possible source. Apart from these, parametritis is practically unknown. The serious results which may be produced by septic absorption and the prompt effect of thorough asepsis are well illustrated in Fig. 92.

Course and Prognosis.—(*a*) In *acute perimetritis*, the acute symptoms should subside in a week; if widespread adhesions are present, part of the exudation will be absorbed, and part will remain, giving rise to the symptoms of chronic perimetritis. *Chronic perimetritis* is often incurable. The prognosis will depend (i.) upon the extent of the inflammation, and (ii.) its cause. If it is the sequel to an acute attack with widespread adhesions the patient will probably have chronic pelvic pain and dysmenorrhœa all her life. If due to extension from a diseased

organ, the patient will be subject to relapses with acute pain after any imprudence in the way of chills or over-exertion.

(*b*) In *acute parametritis*, if treated properly, the fever should subside in a week, and the exudation will probably be absorbed in three weeks. If the fever continues for four or five weeks pus has formed, and the patient will be invalided until the pus finds an exit (which may not be for months). The swelling felt in one lateral fornix becomes larger, pushing the uterus to one side, and later on a firm lump, which may extend to the iliac fossa, is felt along Poupart's ligament. The pus may point in the iliac fossa or follow the line of the vessels into Scarpa's triangle ; or it may burst into the vagina, bladder, or rectum.

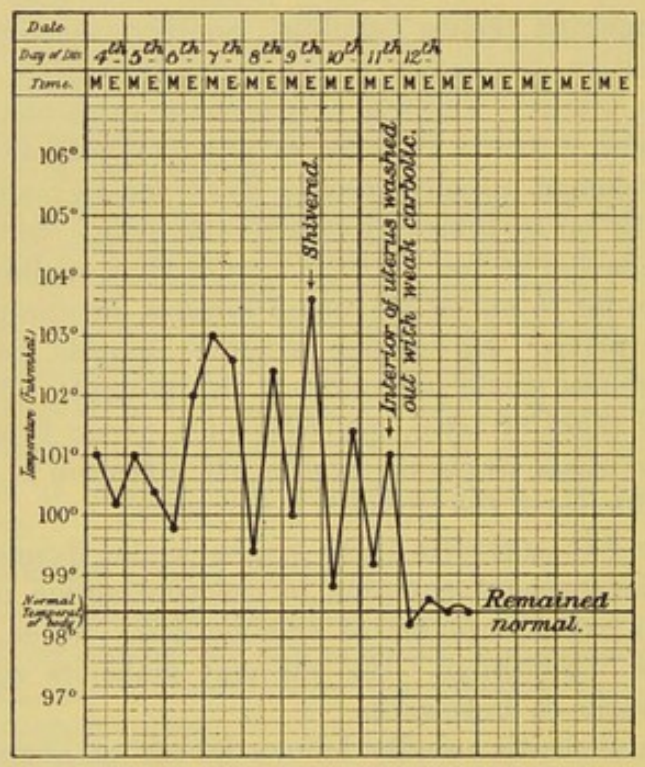


Fig. 92.—Chart of SEPTIC ABSORPTION showing effect of intra-uterine irrigation. Annie H., æt. 33, recently confined ; septicæmia seemed to be threatening, but after thoroughly washing out the interior of the uterus all the symptoms subsided.

In *chronic pelvic cellulitis* adhesions and fibrous tissue are formed rather than pus. These may be absorbed in time, but anteflexion or version of the uterus is a common result of the contraction of the utero-sacral ligaments which occurs in parametritis.

Treatment.—Acute peri- and para-metritis must be treated by (i.) absolute rest in bed ; (ii.) hot fomentations, turpentine stupes to the abdomen, and hot vaginal douches ; (ii.) saline purges ; (iv.) morphia, if necessary, to alleviate the pain. Quinine, 3 grains, t.d. in milk may be given. *Preventive* treatment consists especially (1) in cleanliness of the hands of the nurse or doctor who attends a case of labour or abortion, and (2) in the curing of a vaginitis or an

endometritis before it can extend up to the Fallopian tubes. The treatment of *chronic* peri- and para-metritis consists of (1) the administration of hot vaginal douches (up to 120° F.) daily, each douche lasting ten minutes; (2) ichthyol tampons; (3) treating the pain, dysmenorrhœa, menorrhagia, and other symptoms as described under those conditions. Cold or damp and undue exertion in walking or standing must be avoided; and a certain daily interval of rest in the recumbent position should be ordered. During and after convalescence it is highly important to avoid constipation; aperients or enemata should be given. When suppuration has occurred, the pus must be evacuated by free incision, preferably per vaginam.

§ **333. Inflammation of the Uterine Appendages** (viz., Ovaritis and Salpingitis) may also be a cause of pelvic pain.

Ovaritis is inflammation of the ovary, and should be distinguished from ovarian neuralgia. The *Symptoms* of ovaritis are so frequently accompanied by those of perimetritis that it is difficult to differentiate them. Indeed, *acute ovaritis* is found solely with acute peri- or para-metritis (*q.v.*). *Chronic ovaritis* may be recognised by (1) severe pain at the pelvic brim, extending down the thigh of the affected side; (2) pain increased by any pressure on the pelvic viscera (*e.g.*, by much standing, constipation, or flatus in the abdomen, and in severe cases by sitting); (3) menorrhagia and dysmenorrhœa, because endometritis so often accompanies ovaritis; and (4) dyspareunia. (5) The ovary is usually prolapsed, and therefore, per vaginam, a swelling, the size of a walnut, is found at the site of the ovary, to one side of or behind the uterus, acutely tender to touch, which causes a sickening pain. *General* symptoms, referable for the most part to the nervous system, very frequently supervene. The *Causes* of (1) acute ovaritis are sepsis after labour, abortion, or surgical operations; (2) chronic ovaritis may be due to the same causes as perimetritis, to alcoholism, to certain fevers (*e.g.*, mumps), or to the suppression of menstruation by a chill.

Salpingitis (inflammation of the Fallopian tubes) occurs in three forms, hydro-, pyo-, and hæmato-salpinx. (i.) When the fimbriated end of the tube is closed by adhesions, the exudation within, unable to escape, tends to accumulate in the tube instead of escaping by the uterine opening (hydrosalpinx); (ii.) when the tubes are filled with pus (tuberculous or gonorrhœal) the condition is named pyosalpinx; (iii.) in hæmatosalpinx the tubes are filled with blood.

The *Symptoms of salpingitis* are (1) pain across the lower part of the abdomen, usually greater on one side, often shooting down one leg; (2) on examination a sausage-shaped swelling is found, usually double, running from the lateral fornices to Douglas' pouch; (3) as perimetritis usually accompanies it, the uterus is less mobile than normal; (4) dysmenorrhœa and menorrhagia are usually marked. (5) As regards the general symptoms—in hydrosalpinx there may be none, but pyosalpinx is accompanied by fever. In a pyosalpinx of sudden onset (gonorrhœal), the fever may be very high. *Causes.*—(1) Acute salpingitis is due to septic or to gonorrhœal infection extending upwards; (2) the chronic pyosalpinx, which is found more commonly in young women, is usually due to tubercle; (3) a chronic or subacute vaginitis or endometritis extending upwards may result in salpingitis; (4) hæmatosalpinx is due usually to a ruptured extra-uterine pregnancy.

The *Prognosis* of *ovaritis* depends on the extent of the inflammation around. If there is much matting the case is really one of perimetritis. If the inflammation is confined to the ovary the prognosis is favourable, provided the cause be removable and the patient is not of a neurotic constitution. In *salpingitis* sterility may result from adhesions closing the fimbriated extremity (though this cannot be diagnosed with certainty). Pyosalpinx is dangerous to life, as it may at any time burst into the peritoneum. Tuberculous salpingitis is very chronic, and less

painful than the other forms. In all forms there is a tendency to relapse, and to peritonitis by extension rather than to spontaneous cure.

Treatment.—Acute and chronic *ovaritis* are treated like perimetritis (*q.v.*), together with hot applications to the hypogastrium when the pain is severe. Blisters and iodine applications over the iliac region have been recommended. If the suffering is severe the ovaries may require to be removed. Constitutional treatment must not be neglected—bromides, potassium iodide, and tincture of belladonna are beneficial. In *acute salpingitis*, when a pyosalpinx has resulted and the condition can be certainly diagnosed, laparotomy should be performed and the tube removed. In other cases rest in bed with hot douches may tide over the acute stage. In *chronic* salpingitis, rest, hot douches, and the ichthyol tampons may be tried for a period of two years at least. If this treatment fail, it will probably be necessary to remove the tubes.

§ **334. Pelvic Hæmatocele** is an effusion of blood either into the peritoneal cavity (intraperitoneal) or into the connective tissue of the broad ligament (extraperitoneal). Here there is a *sudden onset* of (1) severe pain in the lower part of the abdomen, accompanied by (2) faintness, perhaps unconsciousness, with (3) nausea, and in some cases vomiting. (4) If the hæmatocele occurs during the menstrual period the flow ceases usually for a few hours at the onset of the pain, but may return again. (5) On examination, the uterus, in the intraperitoneal variety of pelvic hæmatocele, is found pushed forwards behind the pubes, while in the extraperitoneal variety the swelling is smaller, and causes a lateral displacement of the uterus as in pelvic cellulitis. The intraperitoneal variety, if large, forms a lump which can be felt, on bimanual examination, both in Douglas' pouch and above the pubes, and the abdomen is tender and distended. After forty-eight hours, adhesions form and the uterus is fixed, and other signs of pelvic peritonitis may then ensue. The temperature begins to rise in twenty-four hours after the onset of pain—that is to say, when the pelvic peritonitis commences.

Diagnosis.—Intraperitoneal hæmatocele is diagnosed by the sudden onset of pain, the local signs, a history of suddenly suppressed menstruation or a history pointing to extra-uterine pregnancy. The extraperitoneal hæmatocele is extremely difficult to diagnose from pelvic cellulitis, unless a history pointing to extra-uterine fœtation be present.

Causes.—(i.) Pelvic hæmatocele occurs chiefly in multiparæ, between the ages of twenty-five and thirty-five ; (ii.) a reflux of blood during menstruation, variously ascribed to violent exercise, fright, or cold, may give rise to pelvic hæmatocele ; (iii.) a ruptured ovarian cyst ; (iv.) extra-uterine pregnancy (*q.v.*) is the usual cause of extraperitoneal hæmatocele, but this also may lead to intraperitoneal hæmatocele, when the tension bursts the broad ligament and the blood enters the peritoneal cavity.

Prognosis.—If hæmorrhage be large death has been known to occur in about an hour. In smaller hæmorrhages adhesions due to pelvic peritonitis or cellulitis follow, and the exudation may be (i.) entirely absorbed, or (ii.) may go on to suppuration with a danger of general peritonitis. The prognosis in such cases is that of pelvic peritonitis or cellulitis (*q.v.*).

Treatment.—The collapse is treated by stimulants, the pain with morphia and other usual methods. The radical treatment depends upon the cause : (1) In the early stages, if there is no history or sign pointing to extra-uterine pregnancy, leave the patient at rest in bed with ice to the hypogastrium ; (2) if there is a history of extra-uterine pregnancy, laparotomy must be immediately performed. In later stages leave the tumour alone unless there is definite evidence of suppuration, when the case becomes one of pelvic abscess, and is treated as such.

(*b*) *The* **pain** *is of a* **chronic** *character, is of considerable duration, and is* UNATTENDED *by* PYREXIA. Almost any of the different diseases mentioned in this chapter may be suspected. Perhaps

a vaginal examination may reveal ENDOMETRITIS, CHRONIC PERI- or PARA-METRITIS, or a UTERINE DISPLACEMENT; or careful bimanual examination may reveal a PROLAPSED OVARY or an INFLAMED TUBE. UTERINE DISPLACEMENTS and PELVIC TUMOURS alone remain to be considered.

§ **335. Uterine Flexions and Versions.**—The normal position of the uterus is one of slight anteflexion. The uterus undergoes physiological displacements according to the fulness of the bladder and rectum. In itself a displacement leads to no symptom; the symptoms so often associated with displacement are due in the majority of cases to the inflammatory processes in or near the uterus which have caused the displacement.

FORWARD DISPLACEMENTS (ANTEFLEXION).—On examination bimanually the os is found to be high up, and the fundus is felt unduly far forward. The sound passes with some difficulty. In single women a stenosis of the os or an elongated cervix may accompany a forward displacement of congenital origin. As above stated, *Symptoms* may be entirely absent, and attention is first drawn to the condition when other mischief, such as pelvic inflammation, endometritis, parametritis, or a history of dysmenorrhœa, sterility, or constantly recurring abortions, is present.

Causes.—(1) A congenitally ill-developed uterus is often displaced forwards. A forward displacement is diagnosed to be pathological in origin, as distinct from physiological, by the lessened mobility of the uterus, and the pain set up on attempting to move it. Forward displacements are found in association with (2) pelvic peritonitic adhesions, and (3) cellulitis affecting chiefly the utero-sacral ligaments.

Prognosis.—Anteflexion is a frequent concomitant of sterility. Its treatment is extremely troublesome, but if consistently and carefully carried out a radical cure is certainly to be expected unless the condition is due to a considerable degree of pelvic peritonitis or cellulitis, when the prognosis depends upon the removability of these conditions.

Treatment.—Treatment must be directed to any pelvic peritonitis or cellulitis present (*q.v.*). Ichthyol tampons and hot douches with purgative treatment will work wonders in the slighter forms. Massage is highly recommended where the anteflexion is due to the contraction of the utero-sacral ligaments. Division of the cervix is indicated in some cases.

BACKWARD UTERINE DISPLACEMENTS consist of *retroversion* and *retroflexion*. In a backward displacement there is also a certain degree of descent of the uterus. Retro-displacements in themselves cause no symptoms; and in some instances the uterus is congenitally retroverted. On examination a lump is felt in the posterior fornix

continuous with the cervix ; the os looks forwards in version, and is normally placed in flexion. The sound passes backwards, and the lump in the posterior fornix moves when the sound or the cervix is moved. *Symptoms* arise when pelvic adhesions are present, or when the displaced organ interferes with other organs in the vicinity. In such conditions, a retroverted uterus gives rise to (1) pain in the small of the back and the lower part of the abdomen of a bearing down character ; (2) dysmenorrhœa and menorrhagia ; (3) constipation and painful defæcation. (4) If pregnancy occur, the sickness of the early months is excessive, and after the fourth month there may be retention of the urine, with dribbling, and subsequently sloughing cystitis.

Diagnosis.—The diagnosis of a backward displacement is not difficult, but the diagnosis of the cause may be obscure. It is important first of all to determine whether the uterus is freely movable or not, as the prognosis and treatment differ.

Causes.—The causes of backward displacement are (i.) congenital ; (ii.) the dragging of adhesions consequent on pelvic peritonitis ; (ii.) changes in the uterine tissues, such as subinvolution, or tumours in the walls ; (iv.) relaxation of the ligaments, as after pregnancy ; (v.) sudden fall or strain ; and in a few cases (vi.) a habitually overdistended bladder. Several of these causes may act in combination ; thus, subinvolution together with a relaxation of the ligaments cause a retroversion with a certain amount of downward displacement of the uterus, as pointed out in Prolapse.

Prognosis.—(1) So long as the uterus is freely movable and not enlarged, there may be no symptoms until pregnancy occurs, when, as pointed out above, the symptoms may become serious. Most often, perhaps, constantly recurring abortions take place. (2) In the case where the uterus is bound down by adhesions there is a condition which, according to Playfair (*loc. cit.*), is " not fatal, but tends to life-long discomfort."

Treatment.—(1) Where the uterus is freely movable, replace it with a sound or by bimanual manipulation. A Hodge's pessary should be worn so long as the uterus gives any sign of returning to the backward displacement. Where there is pregnancy and the uterus cannot be replaced, even under chloroform, it may be necessary to bring on abortion. Where the condition is due to adhesions, it must be treated as in pelvic peritonitis and cellulitis.

§ **336.** The following are some of the more important **Pelvic Tumours and Vaginal Swellings** : (*a*) *Internal tumours*—(1) uterine fibroid ; (2) cervical or uterine polypus ; (3) cervical or uterine cancer ; (4) retroverted uterus ; (5) pelvic cellulitis ; (6) ovarian tumour ; (7) pyosalpinx ; (8) appendix abscess ; (9) pelvic hæmatocele ; (10) hydatid of the pelvis. (*b*) *External swellings* or swellings about the vulva may be due to (1) prolapse of the uterus ; (2) inversion of the uterus ; (3) prolapse of the vaginal walls (cystocele and rectocele) ; (4) cysts or tumours of the vaginal wall—*e.g.*, of Bartholin's gland ; (5) uterine polypus with a long

pedicle ; (6) local conditions of the *vulva*, such as abscess or labial thrombosis (§ 319) ; (7) *cysts of the vaginal wall* are usually found on the anterior wall, about the size of an egg and painless.

Most of these various conditions have already been fully referred to, but three conditions which may appear as external swellings remain to be described—PROLAPSE OF THE VAGINAL WALLS, PROLAPSE OF THE UTERUS, and INVERSION OF THE UTERUS.

§ 337. **Prolapse of the Vaginal Walls** is very common in multiparæ, especially of the anterior wall. It is then named cystocele, because of its close connection with the bladder ; indeed, the anterior vaginal wall may draw down the posterior wall of the bladder along with it. Prolapse of the posterior wall may occur, and when the rectum is prolapsed also, is named rectocele. But, as the rectum is not so intimately attached to the posterior vaginal wall, a prolapse of that wall is not usually a rectocele. The only symptom in addition to the swelling may be difficulty in passing water until the prolapsed part is pushed up. The diagnosis from a cyst of the vaginal wall is made by passing a sound per urethram and with one finger in the vagina, feeling the point of the instrument in the bladder.

Causes.—The predisposing cause of prolapse of the vaginal wall is a ruptured perinæum. The exciting causes are (i.) increased general intra-abdominal pressure, as in violent muscular efforts ; (ii.) a habitually full bladder ; or, more rarely (iii.) a habitually distended rectum.

Prognosis.—Prolapse of the vaginal walls in time usually leads to prolapse of the uterus. The *Treatment* of the two conditions is very similar (see below).

§ 338. **Prolapse of the Uterus** is its displacement downwards. Four degrees of displacement are described : (i.) The organ may occupy a position somewhat lower than normal ; (ii.) it may lie wholly in the vagina ; (iii.) it may have partly or entirely passed through the vaginal orifice (procidentia) ; and (iv.) in extreme procidentia it lies entirely outside the vulva, the body lying in the inverted vaginal wall.

In slighter cases the uterus is seen coming down on asking the patient to strain. In severer degrees the os uteri can be seen and the body of the uterus and the ovaries can be felt. The other symptoms of prolapse of the uterus are : (i.) The uterus is enlarged, the cervix is frequently hypertrophied, there may be accompanying endometritis or endocervicitis ; (ii.) there is difficulty in passing water till the prolapsed organ is pushed up ; (iii.) sometimes there is a weight or a bearing-down feeling in the pelvis, but more often no pain is complained of, and only the discomfort of the lump during walking and sitting is remarked ; (iv.) the uterus is usually retroflexed, and therefore the sound is found to pass backwards ; (v.) leucorrhœa is usually troublesome. Ulceration of the external parts is apt to supervene on procidentia.

Causes.—(1) The predisposing causes of prolapse of the uterus, as in prolapse of the vagina, are (i.) a ruptured perinæum ; (ii.) a relaxed condition of the parts after labour ; and (iii.) a laborious occupation which demands much muscular strain, such as that of a washerwoman. The exciting causes are (i.) increased intra-abdominal pressure, such as occurs with muscular work and tight lacing ; (ii.) the traction from below exerted by a prolapsed vaginal wall ; (iii.) the increased weight of the uterus in cases of subinvolution or tumour of the wall.

Treatment.—Preventive treatment is highly important. Every woman must rest sufficiently long after labour to insure involution of the uterus. All perinæal lacerations must be repaired as soon as possible. *Curative Treatment.*—In slight cases the uterus must be replaced by pushing up first the posterior vaginal wall, then the uterus, then the anterior vaginal wall. Tampons of ichthyol and glycerine are inserted and changed every two or three days. When any inflammation or undue swelling has been reduced by these means and by hot douching, the insertion of a ring pessary is sufficient. In cases where procidentia has occurred a cup and stem pessary may be necessary. After the menopause prolapse may be difficult to cure, because a pessary in the vagina of old people is so apt to cause ulceration. In some conditions surgical interference is called for.

§ 339. Inversion of the Uterus.—Sudden inversion of the uterus may occur in the third stage of labour, when the fundus is relaxed, but here we are concerned only with the chronic form of inversion. Here the fundus alone may be inverted through the os, or the whole uterus may be inverted. (1) The swelling is red, bleeds readily, and is tender. (2) The sound cannot be passed the normal distance, if at all. (3) Bimanually the fundus is found absent; and if a sound is placed in the bladder in the middle line and the finger in the rectum these can be made to meet without any uterus being felt. (4) There may be symptoms of bearing-down, menorrhagia, and leucorrhœa. The *Diagnosis* may have to be made from fibroid polypi; but here the fundus is not absent from its usual position.

Causes.—Chronic inversion of the uterus is a rare condition. It may be (1) the sequel to acute inversion if the patient survive the shock; or (2) it may be due to the dragging of a fibroid tumour or polypus. This latter may occur with the traction exerted in the operation of removal.

Prognosis.—There is no tendency to spontaneous cure. Death may occur after a long period of suffering and anæmia, from exhaustion or septicæmia. The *Treatment* is altogether operative, and we must refer the reader to a textbook on Gynæcology.

§ 340. Disordered Functions.—Most of the symptoms previously dealt with are in reality disordered functions; but under this heading it is proposed to discuss (*a*) Disordered Micturition (Retention, Unduly Frequent, Painful, or Difficult Micturition and Incontinence; (*b*) Painful Defæcation; (*c*) Painful Sitting; and (*d*) Dyspareunia.

(*a*) **Disordered Micturition** is dealt with more fully in kidney diseases (§§ 311 to 313); here only a few of those special to the female will be mentioned.

I. Retention of the Urine.—The *Causes* peculiar to women are fibroids, malignant disease of the cervix involving the vagina, tumours of the vagina, a retroverted uterus (especially when about the fourth month of pregnancy). In these conditions the retention is due to obstruction of the urinary passage consequent on pressure over the mouth of the bladder. The condition is also found in reflex retention after operations on the perinæum and in hysteria.

II. Frequent Micturition may be produced in women by (i.) pressure on the bladder from a tumour or an enlarged anteflexed uterus; (ii.) a vascular caruncle of the urethra; (iii.) cystocele; (iv.) pelvic inflammation, especially during the early stages; (v.) calculi and gravel; and (vi.) various nervous conditions.

III. Painful Micturition is found especially in connection with urethral caruncle and in the early stages of pelvic inflammation or ovaritis.

IV. Incontinence of the Urine is found (i.) in vesico-vaginal or vesico-uterine fistula; or (ii.) after dilatation of the urethra has been performed—*e.g.*, as a preliminary to lithotrity.

V. Difficult Micturition is found (i.) after labour, when the parts are swollen and bruised; (ii.) with prolapse of the uterus, in which case the symptom is relieved on pressing upwards the prolapsed parts; (iii.) all causes of incomplete obstruction.

(*b*) **Painful Defæcation** may be due to (i.) retroverted and retroflexed uterus, especially when bound down by adhesions; (ii.) an incarcerated retroverted pregnant uterus; (iii). pelvic inflammation when acute; (iv.) ovaritis; (vi) prolapsed ovary; (vi.) coccydynia; and (vii.) a fibroid or other uterine tumours pressing upon the rectum.

(*c*) **Painful Sitting and Coccydynia** is often associated with painful defæcation. (1) The commoner *external* causes of painful sitting are (i.) a vascular caruncle of the urethra; (ii.) vulvitis and all other acute conditions of the vulva; (iii.) hæmorrhoids or fissures of the anus. (2) The *internal* causes of painful sitting may depend upon (i) an increased pressure within the pelvis—*e.g.*, pelvic inflammation, or any tumour within the pelvis; (ii.) injury or inflammation affecting the sacro-sciatic and the sacro-coccygeal ligaments; (iii.) a movable condition of the sacro-iliac joints after parturition; or (iv.) a rheumatic condition of the same joints.

(v.) Dislocation, inflammation, or "neuralgia" of the coccyx is also a recognised cause of the condition.

Diagnosis.—The diagnosis of pelvic inflammation is treated of elsewhere. *Neuralgia* of the coccyx is known by the fact that the coccyx is sensitive to the touch. It may be connected with constipation or disorder of the rectum. Injury of the sacro-sciatic or sacro-coccygeal ligaments is known by (i.) the history of pain often dates from childbirth, or from the injury which produced it; (ii.) pain is produced by pressure on the ligaments, which tightens them; and (iii.) there is an absence of swelling or dislocation of the bone. *Dislocation of the coccyx* has no pain or tenderness, and is known by the fact that the bone, in most conditions, is displaced backwards. When the dislocation is found to be forward, it is much more painful, so that the patient usually sits on one ischial tuberosity—*i.e.*, sits sideways. In a *movable condition* of the joints there is a history of pregnancy with lameness towards the end of gestation, and the patient complains of pain over the pubic bone. In slight cases it may be very difficult to diagnose. *Rheumatism* is known by the absence of other local signs and by the shifting character of the pain, and perhaps the fact that the patient has other manifestations of rheumatism.

Prognosis and Treatment.—Vulvitis and pelvic inflammation are treated of elsewhere. Inflammation and neuralgia of the coccyx are usually cured by laxatives, hot baths, and sedative applications. Injury which has affected the ligaments may also be cured by laxatives and hot baths, but the improvement is slower. Some advise in extreme conditions the division of the ligaments. Dislocation of the coccyx, if backward, may be a cause of no great inconvenience, but if recent may be reduced at the time; if of old standing it should be left alone. A forward dislocation, on the other hand, is much more troublesome, and may require the removal of the coccyx. A movable condition of the joints tends spontaneously to recover. It may be necessary to make the patient rest for a time, and afterwards to walk with a tight bandage across the pelvis.

(*d*) **Dyspareunia** (painful coitus) may arise from a variety of causes. (1) The most frequent is a functional spasm of the sphincter vaginæ, associated perhaps with a general neurotic state. In these circumstances the attempt to pass a speculum will sometimes elicit the same spasm, but may also be a means of cure. (2) Various other local conditions should be carefully looked for, such as a vascular caruncle of the urethra, vulvitis, or vaginitis (see above). Fissures or small ulcers between the folds of the parts, or hidden by the remnants of the hymen, are apt to be perennial causes of discomfort, which will remain undiscovered from month to month, and perhaps year to year. (3) Ovaritis or a prolapsed ovary may produce considerable pain on deep penetration. (4) Parametritis (especially when associated with endocervicitis), perimetritis, and retention of foreign bodies, are also apt to become causes of dyspareunia. (5) Masturbation in the female. (6) Finally there may be, though this is relatively rare, a disproportion between the parties concerned.

Prognosis and Treatment.—The condition of dyspareunia is apt to lead to considerable discomfort, not only to the individual, but to home life in general, and may lead to far-reaching consequences; and when at length the aid of the physician is sought it behoves him to make his investigation with the greatest care, and express his opinion with considerable tact. The first step is to make a very careful and minute examination in a thoroughly good light and under the most favourable circumstances for a local investigation, in view of the minute causes which may underlie the difficulty. The passage of a good-sized speculum will often cure vaginismus and many of the local conditions referred to under (2). A weak cocaine ointment or small doses of bromide may be tried. Childbirth is a frequent cure of vaginismus and many of the causes mentioned. The treatment of the other causes will be found elsewhere.

CHAPTER XV

PYREXIA

MICROBIC DISEASES.

When a patient is suffering from some general or constitutional derangement, he complains of a vague "feeling of illness" (*i.e.*, malaise), or of "weakness" (debility, asthenia). He feels "generally" ill, and perhaps looks ill, but may be unable to mention any localising symptom, such as pain in the side or palpitation. Now, the first thing to do in such circumstances is to ascertain whether he is feverish or not, because all such conditions may be divided into two large clinical groups: A. **Debility with pyrexia,** which includes the Acute Specific Fevers and disorders in which there exists some localised inflammation; and B. **Debility without pyrexia,** which includes the different forms of Anæmia and various toxic and nutritional disorders. The latter will be dealt with in Chapter XVI. In this chapter we are concerned solely with the various conditions attended by elevation of the body temperature.

§ **341. Definitions.**—The term **Acute Specific Fever** (or Specific Febrile Disease) has been applied to those fevers which are due to a specific or special poison (now known to be microbic), introduced into the body from without, and which run a definite course. If the poison was contracted from a previous case, but without contact with the patient, it was said to be an *Infectious* disease (*e.g.*, scarlatina); if the disease was produced only by actual contact with a person suffering from the malady, it was called *Contagious* (*e.g.*, syphilis); but these terms have always been used somewhat loosely and indifferently. It would be out of place to enter here into the question of the nature of this poison; but suffice it to say that there is direct or inferential proof in all the acute specific fevers that it is of microbic origin. At first the microbes themselves were supposed to be the active agents of these diseases, but now in most cases the *causa vera* of the pyrexia and other symptoms is known to be a toxin or toxins which are produced by the microbe. This branch of knowledge has received enormous additions to it during the last quarter of a century (*cf.* §§ 386 *et seq.*).

The subject of Bacteriology will be referred to in a later chapter, and it will be sufficient to mention here the chief **clinical characteristics** which cause us to suspect a disease of being **microbic in origin.** They are three in number:

1. The occurrence of the disease in question in an *epidemic* form—*i.e.*, in the form of an outbreak, or as a series of cases which suggest that the patients contracted the disease either from one another or from a common source, the infection being conveyed to them through the air, the water, or other ingesta. Dietetic poisons (organic and inorganic) must be excluded.

2. Two features are common to all microbic diseases : (1) *Pyrexia* is present at some time during the course ; and (ii.) all the cases of disease run a *definite course*—definite onset, gradual increase to an acme or fastigium, defervescence, gradual or sudden, followed by complete restoration to health, or death.

3. The constant presence in the blood, tissues or excretions of the patient of a *microbe*.

The *pathological proof* that a particular microbe is causally related to the disease consists in applying certain experimental tests (see § 386).

Epidemic, Endemic, and Sporadic are terms by which it is usual to express the relative prevalence of infectious diseases. A disease is said to be *Epidemic* when a large number of cases arise by infection from a common source or from one another at one time, followed by an interval in which none arise. Thus epidemics of measles, scarlatina, and diphtheria arise in the Metropolis and elsewhere from time to time. A disease is said to be *Sporadic* when it occurs only in isolated cases. Thus we speak of a sporadic case of mumps when no other cases of it have been known to occur about the same time and in the same district. An *Endemic* disease is one which is constantly present in a certain district. Thus enteric fever is endemic in London, ague in Central Africa and other marshy areas, and cholera in India.

PART A. SYMPTOMATOLOGY.

§ **342. Pyrexia and Symptoms which may attend it.**—Pyrexia may in some instances be unattended by any symptoms, but in nearly all cases the patient whose temperature is elevated complains of feeling "chilly," or he may have shivering or rigors ; or perhaps he feels "burning hot." Headache, restlessness, and vague pains in the limbs and back are also common symptoms, in addition to the malaise or weakness. His skin is hot and dry to the touch, his pulse and respiration are rapid, his appetite is bad, tongue furred, and bowels confined, his urine scanty and high coloured. In severe cases of fever there is great prostration, considerable mental dulness, and there may be delirium, or the "typhoid" state. By these symptoms we suspect the presence of pyrexia, and the suspicion is confirmed, and the degree of fever ascertained, by the clinical thermometer (see below). The various STAGES through which microbic disorders pass, and the three important symptoms or conditions which are met with in patients suffering from pyrexia—namely, RIGORS, DELIRIUM, and the "TYPHOID STATE"—will now be separately described.

§ **343. Incubation and other Stages of Acute Specific Fevers.**—There is nothing more characteristic of microbic or specific diseases than the *definite course* which they run. It is a curious fact not yet fully explained, that a person does not develop the disease directly after he has been exposed to infection. The interval is called the stage of *incubation*. The patient may be quite well during this stage, or feel a little malaise. Its duration is variable in most diseases, and each disease differs from another (Table, p. 474). This period corresponds to the time during which a healthy person who has been exposed to infection needs to be isolated (placed in quarantine, as it is called), to see if he will develop the disease. A glance at the first column in the table will show that a period of THREE WEEKS will cover the incuba-

TABLE XXI.—SHOWING INCUBATION, DATE OF ERUPTION, AND DURATION OF INFECTION OF THE PRINCIPAL INFECTIVE DISORDERS.

DISEASE.	INCUBATION PERIOD.	DAY OF DISEASE ON WHICH RASH APPEARS.	INFECTIOUS PERIOD, or period during which the *patient* need be isolated.
Varicella.	10 to 19 days, average 14.	The rash is usually the 1st symptom noticed.	Till all scabs have separated, 2 to 4 weeks.
Scarlet Fever.	1 to 5 days, average 2½.	**2nd.**	From commencement till all peeling ceases. Average 5 to 6 *weeks*. Otorrhœa may retain infection for *six months* or more.
Small-Pox.	12 days.	**3rd.**	From commencement till not a trace left of scabs or desquamation. Most virulent in vesiculation, pustulation, and scabbing. 3 to 8 weeks.
Measles.	7 to 14 days, average 10.	**4th.**	*Great* in early period *before* rash out. Till scaling and cough cease. Average 3 *weeks*.
Rötheln.	7 to 21 days, average 10.	**3rd** or **4th.**	10 to 14 days from commencement.
Typhus.	Rarely less than 12.	**4th** or **5th.**	Probably 3 to 4 weeks.
Enteric.	3 to 21 days, average 10.	Average **2nd week.**	Several weeks after pyrexia has ceased.
Dengue.	2 to 6 days.	Initial rash 1st day. Terminal rash 4th.	
Diphtheria.	2 to 6 days.	None.	At *least* 21 days after disappearance of membrane and all throat mischief.

The period of incubation of the other microbic disorders so far as we know is given approximately below. This is important, as the duration of quarantine depends on the period of incubation.

Ague, 12 hours and upwards.
Anthrax, 2 or 3 days.
Gonorrhœa, 2 or 3 days.
Influenza, 3 or 4 days.
Plague, 3 to 7 days.
Glanders, 3 to 18 days.
Relapsing fever, 4 to 10 days.
Whooping-cough, 6 to 12 days.
Malta fever, about 9 days.
Erysipelas, 3 to 6 days.
Cholera, under 14 days.
Yellow fever, under 18 days.
Tetanus, under 21 days.
Mumps, 12 to 21 days.
Syphilis, 15 to 25 days.
Hydrophobia, 40 days or more.
Tubercle, probably some weeks.
Beri-beri, Pneumonia, Septicæmia, Cerebro-spinal fever, Infantile diarrhœa, Psilosis — unknown.

tion of all the eruptive fevers. The actual *invasion* or development of the symptoms of the disease is more or less abrupt, except in enteric fever and sometimes measles. An *eruption* appears upon the skin within the next four days (except in enteric fever) in those diseases which develop a rash, and which are called on that account the Exanthemata. The fever and other symptoms go on increasing until the

acme is reached. Finally the last stage—the stage of *defervescence*—supervenes, and gradually the patient convalesces.

§ **344. Rigors** often indicate the onset of pyrexia. A rigor is an attack of shivering attended by elevation of temperature, rapidly followed (usually) by sweating and a fall in the temperature. Such an attack may vary widely in severity from a simple feeling of " chilliness down the back, like cold water," to a shaking of the whole body, so that the patient shakes the bed beneath him. Severe rigors occur typically and *regularly* in the course of ague, and also at frequent but *irregular* intervals throughout the course of Septicæmia. In childhood, rigors are often replaced by convulsions.

1. First, ascertain that the shivering is not of nervous origin, because a trembling much resembling a rigor may occur as a result of pure fright or from slighter causes in nervous people.

2. Procure, if possible, a series of temperature records, because rigors occur in association with several conditions which can only be differentiated in this way.

Causes.—The causes of rigors are very numerous, but they are best approached in a general way as follows :

(*a*) Coming on in a person *previously healthy*, one should always suspect the advent of some acute illness. In children the eruptive fevers are often ushered in with either convulsions or rigors. In adults, pneumonia, peritonitis, pyæmia, tonsillitis, the eruptive fevers or influenza may be suspected.

(*b*) *Septic Infection.*—When rigors *supervene in the course of an illness* of any kind, abscess or pent-up pus in some position should always be the first thing thought of. *Before the days of the thermometer the doctor used to rely upon shivering and sweating as an infallible indication of the formation of pus.* In a case of pleurisy with effusion, for instance, which has hitherto been serous, the occurrence of shivering indicates that the contents of the chest have become purulent (empyema). Similarly, a rigor occurring with otitis media may point to cerebral abscess or sinus thrombosis. Rigors occurring in a case of cardio-valvular disease indicate the formation of emboli, or the supervention of malignant endocarditis (*i.e.*, arterial pyæmia, Wilks). Shiverings and sweatings are apt to occur during the course of tuberculosis and many other conditions mentioned under the Causes of Intermitting Pyrexia (§ 377). If no obvious cause for an attack of shivering appears, we may suspect some internal ulceration or suppuration, such as appendicitis, or ulceration in some part of the urinary, biliary, or alimentary canals. If the rigor is due to a collection of pus, there will be found a definite leucocytosis.

(*c*) Some *shock to the nervous system* may produce rigors. The passing of a catheter is often followed by a severe rigor, and sometimes the temperature goes suddenly up to 105° or 106° F., and as suddenly down

again. Irritating substances in the alimentary canal may produce rigors reflexly. Sudden obstruction in the biliary or renal passages is often attended by rigors, followed by a feeling of heat and sweating, and the temperature may go up to 105° F. (Murchison).

(*d*) *Neurasthenic* and *hysterical* patients are very apt to have shivering attacks, but these are unattended by elevation of temperature. Attacks of shivering may also constitute a symptom of *vaso-motor disorder*. It is, for instance, a symptom of the reaction which follows, and often forms part of the "flush-storms" chiefly met with at the climacteric —"flushes and shivers," as the patients call them. In these also there is no elevation of temperature.

The *Prognosis* and *Treatment* belong to the several causal conditions, but in any case the patient should be kept warm in bed with a hot-water bottle to his feet, and a full dose of opium, combined with bromide, to soothe the nervous system, and in septic or malarial cases 5 to 10 grains of quinine.

§ **345. Delirium,** or incoherence of speech, is another symptom which frequently accompanies pyrexia. The older authors used to describe three varieties of delirium : (1) Delirium ferox, in which the patient is very violent and maniacal ; (2) typhoid delirium, in which the patient lies on his back muttering, with subsultus tendinum ; (3) delirium tremens, in which there is great sleeplessness, hallucinations and tremors, not necessarily due to alcohol. The nature of the delirium is not always constant in any given disease. For clinical purposes, the *causes of delirium* may be divided into two groups—FEBRILE and NON-FEBRILE. It is important, therefore, to take the temperature at once in every case of delirium. Alcoholic subjects and children are predisposed to delirium when attacked with only slight fever.

a. Febrile Delirium, or delirium with elevation of temperature, may arise under four circumstances :

1. DISEASES OF THE BRAIN, such as tuberculous meningitis. This kind is generally accompanied by pain in the head, vomiting, intolerance of light, and paralysis of cranial nerves.

2. ACUTE LOCAL INFLAMMATIONS in other parts of the body, such as pneumonia. It is advisable, therefore, to examine all the organs of the body.

3. All the ACUTE SPECIFIC FEVERS are liable to be accompanied by delirium. The tendency, however, varies considerably, and it is important to bear this in mind, because, as a prognostic indication, delirium occurring in a disease like measles or acute rheumatism, in which it is rare, has a much more serious meaning than when it occurs in pneumonia, for instance, where it is usual (see Table XXII.). Occurring in acute rheumatism, it is generally an indication of pericarditis, endocarditis, or some other serious complication.

4. Certain cases of DELIRIUM TREMENS of a SEVERE KIND are accom-

panied by an elevation of temperature. Indeed, the prognosis in this affection may largely depend upon the temperature. We must be careful to exclude local inflammations in such cases, for they are apt to come on very insidiously. In the worst cases of ACUTE DELIRIOUS MANIA also the temperature may be considerably elevated (see *b* 6, below).

TABLE XXII.—SHOWING THE RELATIVE FREQUENCY OF DELIRIUM IN THE VARIOUS MICROBIC DISORDERS.

Frequent in—	*Occasional in—*	*Rare in—*
Confluent Small-pox	Remittent Fever	Influenza
Typhus	Yellow Fever	Mumps
Lobar Pneumonia	Small-pox (modified)	Dysentery
Enteric Fever (after 1st week)	Measles	Cholera
Meningitis	Relapsing Fever	Acute Rheumatism
Cerebro-Spinal Fever		Ague
Erysipelas		Diphtheria
Plague		Rötheln
Malignant Endocarditis		Varicella
Scarlet Fever		
Septicæmia		

b. Non-febrile Delirium may arise under six conditions :

1. DELIRIUM TREMENS (Delirium e Potu) is, as just mentioned, usually unattended by elevation of temperature, and is undoubtedly the commonest cause of non-febrile delirium. It is recognised by the history, the muscular tremors, sleeplessness, and the characteristic hallucinations.

2. CHRONIC RENAL DISEASE, and especially chronic interstitial nephritis, gives rise in its advanced stages to a muttering delirium or incoherence, which thus becomes a symptom of the gravest import, and generally heralds coma and death. The delirium is due to uræmia, and occurs in other renal diseases.

3. POST-FEBRILE DELIRIUM (Post-Febrile Mania).—During the convalescence of pneumonia, enteric fever, and other exhausting diseases, especially such as run a protracted course, mental symptoms may develop. These symptoms, which—in most of the cases I have met with—make their appearance without any warning, give great uneasiness to the friends. Nevertheless, by means of good food, tonics, and fresh air, such mental symptoms will entirely disappear.[1] Before venturing on a prognosis, however, inquiry should always be made for any family history of mental disease, for an hereditary taint greatly lessens the chance of recovery. The condition is recognised by the

[1] A recent case of this affection which I have seen was that of a lady, æt. thirty-nine, who, after a protracted illness with subacute rheumatism, developed mental symptoms which lasted for some three months, until the administration of opium gave her the necessary quiet, and she completely recovered. She had delusions, wanderings at night, and serious loss of memory. She always addressed me as " Dr. Devill."

history of the previous malady. Sometimes the mental derangement consists simply of loss of memory, especially for the names of persons and things, but more often the mind "wanders," and there are delusions.

4. Reflex Delirium.—Trousseau[1] mentions cases of children with intestinal worms who had delirium, and several cases are mentioned by the same author which were caused by the tickling of the soles of the feet. The transient delirium connected with the severe pain of childbirth is probably of the same nature. I am inclined to agree with Griesinger,[2] who says that "mental diseases caused by intestinal worms would be very interesting and more practically useful if they could bear a closer investigation." Nevertheless, the transient delirium or mania met with at the climacteric comes with some probability in this category, the reflex cause being situated in the generative organs.

5. Deliriant Drugs should always be suspected when delirium develops suddenly in a person in health, especially children in the country, in the absence of any of the foregoing. The most important are belladonna, hyoscyamus, stramonium, and others of the solanaceæ, camphor in rare cases, ænanthe crocata, cocculus indicus (with which beer used to be adulterated), poisonous fungi, and sometimes salicylic acid in large doses. Morphia in some people invariably produces delirium.

6. Acute Mania sometimes comes on very suddenly, and, as previously mentioned, only differs from "delirium ferox" or maniacal delirium in not being referable to some bodily disease or toxic condition of the blood. We are enabled to identify this condition by (1) the temperature not as a rule being elevated; (2) by its affecting a person previously in good bodily health; and (3) the exclusion of any organic lesion by a careful examination, both of the nervous and other physiological systems. As regards the temperature there is an exception in the rare and serious condition known as "acute delirious mania," in which marked pyrexia is present.

Prognosis.—Febrile delirium is not necessarily a grave symptom when it is associated with a *disease in which its occurrence is usual—e.g.*, pneumonia—and especially when the cause is only temporary; but its presence adds considerably to the gravity of a case if the occurrence of delirium is unusual (see Table, p. 477), for it indicates a very severe attack, or the occurrence of complications, or both. *Non-febrile* delirium is a grave symptom in chronic renal disease. The prognosis is serious as regards recovery in all patients who have an hereditary tendency to mental disorder. In acute mania the prognosis is very grave.

Treatment.—It is necessary to provide a nurse or attendant, and restraint may be called for. *Remedial Treatment.*—An ice-bag to the

[1] Clinical Lectures: New Syd. Soc. Translation.
[2] Griesinger on Mental Diseases: New Syd. Soc. Translation, p. 197.

head for an intracranial inflammation ; good nourishing food for mania and post-febrile delirium ; a brisk purge for uræmia. Alcohol is indicated if the pulse is weak, but if it is strong and bounding, alcohol, as a rule, aggravates the condition. The *symptomatic treatment* consists of sedatives, such as chloral and the bromides. Opium and morphia require caution. In delirium tremens, for example, it does a great deal of good in some cases by procuring sleep, but in others it only aggravates the maniacal condition. In post-febrile delirium and other conditions where the brain is suffering from malnutrition, opium in small doses is a most valuable remedy, and may be given without fear if the kidneys are healthy.

§ **346. The Typhoid State** may be described as a condition of unconsciousness (coma) or semi-consciousness attended by elevation of temperature and muttering delirium, due to a toxic condition of the blood. The name of this condition was derived from its frequent association with typhus, but it is met with in many other fevers. With reference to the question of pyrexia, it should be stated that the comatose condition, due to renal disease (uræmia), advanced liver disease, and various poisons (particularly opium), has sometimes been described as the typhoid state, but these are apyrexial conditions, and it is preferable to include only those with pyrexia. In short, the typhoid state corresponds clinically to a state of coma *plus* pyrexia and muttering delirium.

Symptoms.—The typhoid state is always secondary to some febrile condition, in the course of which it arises. The first symptom usually noticed is sleeplessness with delirium, generally of the muttering variety, but by-and-by stupor supervenes, which gradually deepens. The mental faculties are obscured, but the unconsciousness is not always so complete as one would imagine. The tongue is dry, brown, and rough, and sordes collect upon the teeth. The pulse is rapid, feeble, and irregular, and the heart-sounds distant. The respiration is usually rapid, but shallow. The pupils are dilated, but the patient does not see. Nevertheless, he looks about at imaginary objects—"coma vigil." Dysphagia may supervene, and is a very serious indication of profound stupor. Stertorous respiration only occurs in like circumstances, and is another grave indication. The profound disturbance of the nervous system is evidenced by restlessness, subsultus tendinum (muscular twitchings), floccitatio (picking at the bed-clothes), and, in extreme cases, convulsions. The temperature is elevated, its height and course depending chiefly upon the nature of the primary malady.

Diagnosis.—(1) The "typhoid state," as above mentioned, may be distinguished from *coma* by the absence of pyrexia, and the evidences of renal or liver disease, apoplexy, or other cause of the coma. (2) Certain acute *inflammations of the brain* are, however, attended by pyrexia, and offer considerable difficulty. This is particularly the case with

tuberculous meningitis. The presence of optic neuritis, paralysis of the cranial nerves on the one hand, and the signs of the primary malady which has produced the typhoid condition on the other, are the only means upon which we can rely.

Causes.—Patients with an alcoholic history are predisposed to the development of the typhoid state. Renal fibrosis (chronic interstitial nephritis) offers a similar predisposition.

1. The Acute Infectious Fevers are the commonest causes, and particularly typhoid and typhus. It occurs as an ordinary symptom in the course of these two diseases, and in some others (see Table XXIII.). In some other diseases it occurs only occasionally, and in others it is rare. If it arises in either of these latter groups, it indicates either (1) a very severe variety of the disease, or (2) some serious complication ; and in any case that the patient is likely to die.

Table XXIII.—Relative Frequency of the Typhoid State in Different Diseases. Alcoholic Subjects and Patients with Granular Kidney are predisposed to the Typhoid State.

Frequently met with, especially towards the end, in—	*Occasionally met with in—*	*Rare in—*
Typhoid (Enteric) Fever	Scarlatina	Measles
Typhus	Diphtheria	Cholera
Confluent Small-pox (unmodified)	Cerebro-Spinal Fever	Variola (modified)
Erysipelas (severe)	Anthrax (Internal)	Varicella
Septicæmia (including Malignant Endocarditis and Osteomyelitis)	Remittent Fever	Dysentery
Meningitis		Ague
Lobar Pneumonia		Relapsing Fever
Acute Miliary Tuberculosis		Acute Rheumatism
Acute Glanders		
Acute Anthrax		
Jungle Fever		
Comatose and Hæmorrhagic Malaria		
Yellow Fever		
Plague		

2. Certain Local Inflammatory Disorders may be attended by the typhoid state, such as acute lobar pneumonia, acute pulmonary tuberculosis, ulcerative endocarditis, and acute meningitis.

3. Certain acute Idiopathic Inflammatory Diseases may, in rare instances, be attended by the typhoid state, such as acute gout and very intense forms of delirium tremens. It is extremely rare in acute rheumatism, unless accompanied by peri- or endo-carditis.

Diagnosis of the Cause.—The clinical investigation should be conducted on the same lines as in cases of pyrexia. Is it due to *local* or *generalised* inflammation ? First, every organ in the body should be thoroughly examined so as to exclude local disorders. Secondly, we proceed to the diagnosis of the general fevers from one another, and, if

possible, obtain a series of temperature records. In cases where the cause of the typhoid condition is obscure, septicæmia should always be suspected, and its origin carefully sought.[1]

Prognosis.—The typhoid state, like delirium, has a less serious import in diseases such as enteric fever, in which it is habitually met with. But it is always a grave condition, and indicates profound cerebral depression. Occurring in the course of scarlatina, erysipelas, or measles, it often indicates pulmonary or cardiac complication, and is proportionately serious. As regards symptoms, the profundity of the stupor is a measure of the intensity of the microbic toxæmia, and dysphagia, stertor, or convulsions are generally lethal signs.

The Treatment of a condition such as this arising in the course of so many diseases must necessarily vary, and our first duty is *to ascertain what disease is in operation.* It is, however, due in all cases to the effects of the toxin upon the central nervous system. The blood poison consists partly of the microbic toxins and partly of the excessive nitrogenous metabolism incidental to pyrexia. The indications are (1) to eliminate the poison by diuretics, diaphoretics, and aperients ; and (2) to stimulate and support the patient's strength by nutriment and stimulants. Alcohol was formerly given in large quantities. Dr. Murchison treated patients admitted on alternate days into the London Fever Hospital on opposite methods, and found that they recovered just as well without alcohol ; though, on the other hand, it did no harm. In practice, the state of the pulse and of the heart should be our guide. As regards symptomatic treatment, if the delirium be very violent, sedatives, such as chloral or bromide, are indicated if the heart will stand them. Opium should be avoided, as it prevents the elimination of the poison. For the treatment of Hyperpyrexia see § 392.

PART B. PHYSICAL EXAMINATION.

The clinical investigation of pyrexial disorders consists of (1) Clinical Thermometry ; (2) An Examination of the Organs ; and (3) Bacteriological Investigation.

§ **347. Clinical Thermometry and Types of Pyrexia.**—The temperature is ascertained by means of the clinical thermometer—a little instrument which is now found in most well-ordered households.[2]

[1] While I was Medical Superintendent at the Paddington Infirmary a young woman was brought in with all the symptoms of the typhoid state. The subsequent course of the temperature and the occurrence of sweating and rigors declared the disease to be septicæmia, which was traced to a pelvic origin. She died, and the case was brought home to a professional abortionist, who was sentenced to penal servitude.

[2] Owing to the shrinkage of the glass, all glass thermometers are apt after a time to read too high unless they have been stored for months or years before the scale is marked and zero fixed. A clinical thermometer, for instance, may, at the end of a year after manufacture, read a whole degree too high. Hicks, of Hatton Garden, has patented a process of annealing thermometers which obviates this error, and does away with the necessity of prolonged storage.

The temperature of the body is usually taken in the axilla or the mouth. The temperature may also be taken in the rectum, where it may be ½° to 1° higher than in the mouth. The temperature in the mouth is usually higher than in the axilla, which is best regarded as the normal. In children the thermometer may be held in the groin-fold or "crutch." The normal temperature of the body varies between about 97·8° and 99° F.; average 98·4° F. It is highest about 8 p.m., and lowest about 4 a.m. It tends to be lower in old age and higher in infancy, especially after an attack of crying. The temperature is subnormal after a loss of blood, during convalescence, in cardiac failure, and in all states of collapse.

A temperature of 100° is regarded as slight fever.
" " 102° " moderate fever.
" " 104° " high fever.
" " 105° and upwards is regarded as hyperpyrexia.

The Temperature Chart.—*Very little information can be derived from a single observation of a patient's temperature, and in all cases of pyrexia one must know the course which it runs from day to day and hour to hour.* In most cases of fever it is hardly possible to come to any conclusion without seeing a "chart" of the case—*i.e.*, a series of records. In all cases of pyrexia the temperature should be taken **and recorded** morning and evening; and in all acute cases it should be taken four-hourly. In cases of suspected tuberculosis and some other affections it is important to obtain hourly records throughout the day, otherwise slight elevations may be missed. The pulse and respiration should also be observed, especially in abdominal inflammations, where the temperature alone does not give us a true idea of the amount of mischief which is going on. The onset of the pyrexia may be gradual, as in enteric fever or diphtheria, but more often it is sudden and accompanied by a rigor, as in scarlet fever or pneumonia. Remember that the *onset is very sudden* in scarlatina, small-pox, and erysipelas; it is *gradual* (taking perhaps two or three days) in measles and pertussis. During the next few days the temperature generally increases until the *acme* is reached. The termination may be gradual, when it is said to terminate by *lysis*, as in enteric; or pyrexia may terminate suddenly by *crisis*, as in pneumonia.

Types of Pyrexia.—In the absence of any eruption, the course of the temperature is our best, and may be our only, guide. It is usual to describe three types of pyrexia, according to the course which the temperature pursues from day to day (Fig. 93): (i.) *Continued or Continuous Fever*, where the temperature remains at about the same elevation throughout the day, and where the *diurnal variation never exceeds the normal diurnal variation* — viz., one, or at most one and a half degrees; (ii.) *Remitting Pyrexia*, when the diurnal variation is greater than the normal diurnal variation, but where the temperature

never comes down quite to normal; (iii.) *Intermitting Pyrexia*, where the temperature at some time of the day is normal or subnormal, and at another time of the day, usually in the evening, it is raised one, two, or more degrees. But for clinical purposes the two latter may be grouped together, and thus we have TWO GROUPS of fevers—one in which the pyrexia is practically CONTINUOUS, and another in which there is a remission, or INTERMISSION, once or oftener during the twenty-four hours, usually in the morning.

The following are useful facts to remember concerning temperatures: (i.) The sudden advent of high fever in a previously healthy person without other symptoms indicates, in England, Scarlet Fever, Small-

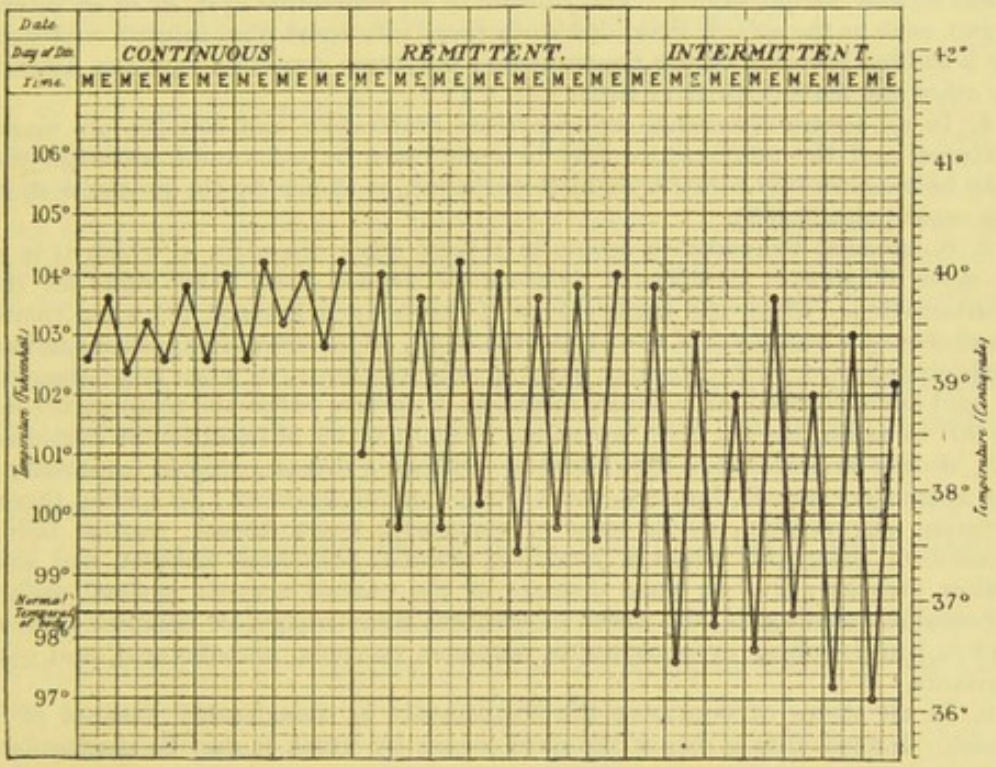


Fig. 93.—TYPES OF PYREXIA.—Continuous pyrexia showing only the normal variations in the morning and evening. Remittent pyrexia showing a drop of several degrees each day. Intermittent pyrexia where the temperature comes down to normal at some time every day.

pox, or Erysipelas, and sometimes Pneumonia. A very gradual advent is suspicious of Enteric Fever. (ii.) A fresh rise after the temperature has begun to fall indicates a complication or a relapse. (iii.) A sudden fall in the course of a fever (especially Enteric Fever) may indicate internal hæmorrhage, perforation of the peritoneum or pleura, or profuse diarrhœa. (iv.) A considerable rise in diseases usually non-febrile, such as tetanus, delirium tremens, cholera, cancer, epilepsy, apoplexy, etc., generally indicates a fatal termination.

§ 348. Subnormal Temperature.—The temperature of the surface of the body as indicated in the axilla, is rarely more than one or two degrees below normal. When it is below 96° the condition usually amounts to collapse. Subnormal

temperature is not so important, for purposes of diagnosis, as elevation of temperature; but in the first four instances given below it may aid us in their differentiation. Subnormal temperature adds to the gravity of the prognosis in most wasting disorders. In regard to treatment, temperature readings below the normal are indications for the administration of stimulants and nourishment.

Causes.—1. Subnormal temperature as an indication of *lowered vitality* occurs in normal circumstances in the aged, in whom the temperature is habitually several fractions of a degree below normal.

2. A subnormal temperature is of considerable diagnostic significance in the *prodromal stage of tubercle*, and especially tuberculous meningitis. If a carefully recorded series of temperatures in a person suspected of tubercle show a subnormal morning and a normal evening temperature (or *vice versa*), it adds to our suspicions.

3. The temperature takes a *sudden drop* in internal hæmorrhage or perforation of the bowels. In enteric fever this sudden fall may be the only indication of these serious complications. The rupture of an abdominal cyst, or of an internal organ, such as the spleen, liver, or kidney (very rare apart from injury), is attended by a sudden lowering of the temperature; but these conditions are also attended by other and more distinctive signs.

4. In all severe *abdominal inflammations* prostration and collapse are marked features, and the temperature may in some cases be subnormal, although there may be considerable constitutional disturbance, as shown by the prostration, and the rapid pulse (§ 165).

5. Subnormal temperature occurs in several other disorders in which it is not of much diagnostic significance, because we depend upon other signs for their identification. Thus, the temperature of the body is lowered (i.) when there is an excessive withdrawal of heat from the body, as in cases of exposure combined with privation, or with extensive weeping skin eruptions; or when large quantities of fluid are evacuated, as in severe diarrhœa or cholera (when the temperature may be 90° in axilla, though 105° in rectum); (ii.) in states of inanition or cachexia—*e.g.*, during convalescence from fevers, Addison's disease, cancer (especially of the alimentary canal), diabetes, and chronic mental disorders; (iii.) when there is deficient oxygenation, as in cases of congenital heart disease, cardiac failure, alcoholism, idiopathic anæmia, jaundice, uræmia, pernicious anæmia, and acute yellow atrophy; (iv.) in some diseases of the central nervous system, such as tuberculous meningitis, the onset of cerebral hæmorrhage, or cerebral tumour; and (v.) in poisoning by phosphorus, atropine, morphia, carbolic acid, and other irritants.

6. In all states of COLLAPSE the temperature is considerably lowered (2° or more). Indeed, this is one of the chief means by which it may be distinguished from syncope.

§ **349. Examination of Organs.**—All the viscera must be carefully examined in accordance with the scheme of case-taking, pp. 7 and 8, so that local causes for the pyrexia may be excluded. For *clinical* purposes there are two great groups of causes of pyrexia: (*a*) **local inflammations** such as pleurisy, appendicitis, abscess of the liver, etc., on the one hand; and (*b*) **general inflammatory (constitutional) conditions,** like scarlatina, rheumatic fever, and pyæmia on the other.

If any local inflammation is found, turn to the chapter dealing with the diseases of that part. But it must still be remembered that some constitutional disease (*e.g.*, some fever) *may* be present, of which the local disease is a complication. Thus pneumonia, which would be discovered in the course of our examination, is a frequent complication of enteric fever; and endocarditis of rheumatic fever or scarlatina. There are two features which may lead us to suspect a combination of disorders such as this: (1) The signs and symptoms of the local disorder may be of an aberrant type (*e.g.*, see Aberrant Types of Pneumonia, § 87*b*); and (2) the

constitutional disturbance presented by the patient would be greater in degree or different in kind than would accompany the local disease if it were the only disease present.

§ **350. The Examination of Blood** often affords most valuable information, and it may be useful to make a blood-count or film (§§ 397 and 398), to take a Wright's capsule specimen (§ 387) for Widal reaction, or opsonic investigations, or to take a larger specimen of blood for bacteriological investigation.

PART C. THE DIAGNOSIS, PROGNOSIS, AND TREATMENT OF MICROBIC DISORDERS.

§ **351. Routine Procedure and Classification.**—In cases of pyrexia we must investigate, as in other cases, three questions :

First, THE LEADING SYMPTOM complained of by the patient will be one or more of those mentioned in § 342.

Secondly, THE HISTORY OF THE ILLNESS. The *date* when the symptoms commenced—*i.e.*, the PRECISE DURATION OF THE ILLNESS—is a most important matter. A few of the fevers—*e.g.*, enteric fever and diphtheria—commence insidiously ; but the majority are ushered in suddenly, very often with an attack of shivering (a rigor). Throughout the entire course of every case of fever the physician should have constantly in mind the " day of the disease,"[1] so that he may know what events to expect at that particular period of the case. In enteric fever, for instance, on the fourteenth day the diurnal range of the temperature should commence to be more marked, and during the next few days special care should be exercised to avoid hæmorrhage or perforation.

Thirdly, THE EXAMINATION OF THE PATIENT comprises three important matters : (1) Physical examination ; (2) is there, or has there been an eruption ? and (3) the temperature and its course.

(1) EVERY ORGAN must be systematically examined (scheme of Case-taking, pp. 7 and 8), and as carefully and thoroughly as the patient's condition will allow, in order that we may DETECT OR EXCLUDE ANY LOCAL DISEASE. This is important, because all cases of pyrexia are associated with or due to some **local inflammatory disease,** or some **generalised febrile disorder** (*e.g.*, enteric fever), or both.

(2) WHETHER THERE IS OR HAS BEEN ANY ERUPTION is the next question. The first of the groups (*vide infra*) into which all fevers may be divided comprises those in which an eruption distinctive of the disease appears within the first four days (with one exception) after the illness. The day on which it appears in each disease should always be at our fingers' ends (Table, p. 474).

[1] Students do not always understand quite correctly the meaning of this phrase. For instance, the fourth day of a disease is the third day *after* its commencement. Thus the eruption of measles appears on the fourth day, and, supposing the patient were taken ill on a Monday, the eruption would appear on Thursday.

(3) The temperature **and its course** is the next thing to investigate; and it is of the greatest importance to obtain a chart or succession of readings, after the manner described in § 347.

The **classification** of pyrexial disorders may conveniently be based upon the results of our examination — namely, the eruption, if present, and the course of the temperature.

Group I.—Exanthemata or Eruptive Fevers—*i.e.*, fevers which are characterised by an eruption distinctive of each disease appearing on one of the first four days of the illness (§ 352).

Group II.—Continued Fevers—*i.e.*, fevers in which the temperature runs a more or less continuous course, and which present no eruption during the first four days (§ 363).

Group III.—Intermittent Fevers—*i.e.*, fevers in which the temperature runs an intermittent (or remittent) course, and which present no eruption (§ 377).

If the physical examination reveals signs of disease of some particular organ, reference should be made to § 349, and to the chapter on diseases of that organ.

GROUP I. THE EXANTHEMATA OR ERUPTIVE FEVERS.

In all the diseases in this group the onset of the pyrexia is more or less abrupt, and a well-marked general eruption appears during the *first four days* of the illness. The course of the pyrexia varies considerably in the disorders in this group. When the eruption comes out it generally drops, except in measles and scarlet fever.

Common.		*Rare.*	
I. Chicken-pox (first day) ..	§ 352	VIII. Dengue (first day) ..	§ 359
II. Scarlet fever (second day)	§ 353	IX. Typhus (fourth or fifth day)	§ 360
III. Erysipelas (second day) ..	§ 354	X. Anthrax (first day) ..	§ 361
IV. Small-pox (third day) ..	§ 355	XI. Acute glanders ..	§ 362
V. Measles (fourth day) ..	§ 357		
VI. Rötheln (third or fourth day)	§ 358		
VII. Enteric fever (usually tenth day). Influenza, plague, and other members of Group II., occasionally present early rashes.			

In each of the exanthemata the eruption has special and distinctive characters of its own, which, together with the day of the disease on which the eruption appears, enable one to differentiate the members of this group from one another. Scarlet Fever may be regarded as the type, but it will be convenient to take them in the order in which the eruption appears. Typhus is hardly ever seen in the present day. Dengue is not met with in England. Anthrax and Glanders are, like hydrophobia, derived from the lower animals.

§ **352. I. Varicella or Chicken-Pox** (synonym: Variola Crystallina, Spiriæ, Nathæ, Ligitime, or Water-pock) may be defined as an acute contagious disease, manifested by an eruption of successive crops of

large vesicles, usually accompanied by slight exacerbations of fever. It is in most cases a trivial disorder of childhood. A thousand years ago it was described as a spurious form of small-pox (Heberden), and confounded with it till later years.

Symptoms.—The *rash is generally the first sign noticed*, though it may have been preceded by a feeling of "chilliness" or feverishness one to three days before its appearance. It consists of pink, slightly raised papules, which in the course of twelve or twenty-four hours become vesicular. In a few days the vesicles enlarge, become depressed in the centre, and finally dry into scabs which fall off in five days, rarely leaving any extensive scarring. The essential feature of this eruption is that it comes out *in successive crops*, and consequently we see different stages of the rash on the same area of skin. This process goes on for about a week, when the disease may be considered to terminate. The rash starts on the chest and neck, and usually invades the whole body except the face and hands (the most frequent situations in small-pox). It may invade the mucous membrane. The whole disease seldom lasts longer than ten days, and it may be so trivial as to pass unnoticed by the patient. The temperature rarely exceeds 103° F. A case ceases to be infectious after the scabs have separated. The period of incubation is uncertain, but it is believed to be between ten and nineteen days.

Diagnosis.—Modified Variola is the chief disease from which it has to be differentiated, although this should not be difficult, because in small-pox (i.) the rash comes out definitely on the third day ; (ii.) it does not appear in successive crops ; (iii.) its favourite situations are the face and wrists ; and (iv.) the constitutional symptoms are very definite and characteristic. *Herpes* is distinguished by the limited area, and grouping of the vesicles. *Pemphigus* is distinguished by the size and chronic character of the blebs. *Dermatitis Herpetiformis* is distinguished by its chronic character, and by the vesicles occurring in groups.

Etiology.—Varicella is essentially a disease of childhood. It occurs in epidemics, though it is nearly always endemic in London. One attack usually confers immunity, but there are many authenticated cases of second or even third attacks. Other infectious fevers predispose to it.

Prognosis.—Deaths are very rare. An attack is usually over in a week or ten days, but it is apt, particularly in adults, to be followed by weakness, which indeed may be more troublesome than the disease itself. *Untoward symptoms*, such as gangrene and hæmorrhage into the vesicles, are rarely met with. *Complications* are few in number, the chief one being catarrh of the respiratory passages.

Treatment.—The itching is generally the chief trouble, and this may be relieved by chloral, creolin baths, or calcium chloride. Quinine and arsenic are the best remedies for the resulting weakness.

§ 353. II. **Scarlet Fever** (synonym: Scarlatina) is one of the most serious, and one of the commonest, of the eruptive fevers. It may be defined as a contagious febrile disease attended by inflammation of the tonsils. and a punctiform eruption on the skin, followed by desquamation. There are five characteristic *Symptoms*. (1) After a period of incubation which varies from one to five days, there is a *sudden advent* of high fever. The occurrence of this sudden pyrexia is of itself extremely characteristic of scarlet fever, small-pox, and erysipelas, and, occurring in a child previously healthy, is always suspicious of scarlatina. Vomiting also occurs in the latter in 80 per cent. of the cases (Caiger[1]). The temperature gradually subsides to normal about the fifth day in mild cases. It does not, as in small-pox, subside when the rash comes out (Fig. 94). (2) A *sore throat* appears on the first day, with the fever, and gives a characteristic scarlet colour to the tonsils. The swelling is greatest about the fourth day. Sore throat occurs with several of the exanthemata. In scarlet fever it is the tonsils and pharynx that are affected (never the larynx); in measles the larynx is chiefly affected; in small-pox both the larynx and pharynx are involved. The inflammation may become very severe. (3) The *eruption* is the next symptom, and it is generally remarkably regular in its appearance—twenty-four to thirty-six hours after the advent of pyrexia. It has two elements—a generalised red blush, disappearing on pressure, and a number of minute points slightly raised and redder than the surrounding skin. It appears on the front of the chest, axillæ, and arms, and is last developed on the limbs *and palms;* but in the latter position there is *no* punctate or papular eruption as there is in measles. The face is flushed, but has no punctiform rash. It continues well marked until the fourth or fifth day, then declines, and is generally completely gone by the seventh or eighth day, except on the outer side of arms and legs. (4) The *strawberry tongue* is seen typically about the fourth day. It is due to the clearing of the fur, which leaves a bright red surface, with marked fungiform papillæ. (5) *Desquamation* is apt to occur with any severe skin inflammation, but it is more characteristic in this than in any other fever. It begins about the fourth day, and continues for three weeks—first on the face, and, following the order of the rash, *last on the palms and soles*. In the latter positions the flakes are large; elsewhere they are small and branny.

Varieties.—There are, according to Dr. F. F. Caiger, three chief varieties: (1) The *Benign*, simple or ordinary type as above described. Various symptoms—*e.g.*, rash or sore-throat—may be absent, and these cases are spoken of as *latent*. (2) In *Septic Scarlet Fever*, Scarlatina Ulcerosa, or Anginosa, " the ordinary symptoms are aggravated by the

[1] Dr. F. F. Caiger, in the article on this subject in Dr. W. H. Allchin's " Manual of Medicine." Macmillan, London, 1900.

presence of faucial ulceration, which, in addition to being a serious lesion in itself, provides a focus from which septic material is absorbed into the system" (Caiger, *loc. cit.*). (3) In the *Toxic* form the patient is seized with high fever, delirium, and perhaps convulsions; the rash is very intense, but the throat symptoms ill-marked, and the patient dies during the first week. *Malignant* or Typhoid Scarlet Fever, in which there is low muttering delirium, usually a marked rash, and death without complications in a few days, is a very rare variety in the present day. In the *Hæmorrhagic* form petechiæ appear under the skin and mucous surfaces.[1] These last are intense varieties of the toxic form.

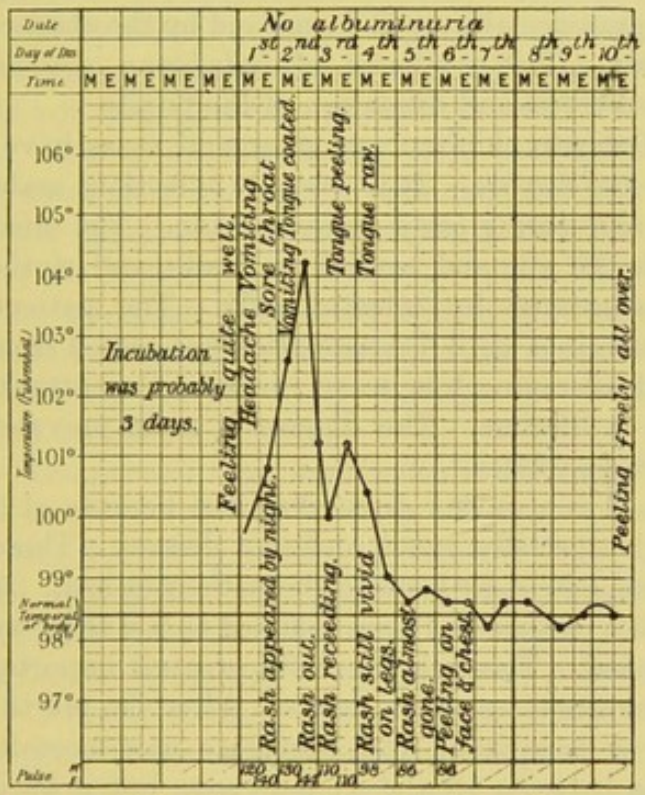


Fig. 94.—SCARLET FEVER. Arthur M——, æt. 5. A typical mild case, specially as regards the initial symptoms, the rash, the tongue, and the desquamation. The various incidents are shown on the chart, for which the author is indebted to Dr. F. F. Caiger.

Diagnosis.—The diagnosis of scarlatina is not difficult in typical cases. The abrupt advent of high fever, accompanied by vomiting and sore throat, in a child who has not had the disease, is always extremely suspicious, and if the disease is prevalent the diagnosis is almost certain. During the first few days the greatest difficulty is sometimes experienced in the diagnosis from *quinsy*, in which there is frequently albuminuria at the onset, but less stupor and lethargy, generally less fever, and the history of previous attacks of quinsy. Slight albuminuria may be present in both conditions during the early stages. With-

[1] *Surgical Scarlatina* is an unfortunate name suggested for an erythematous rash, accompanied by constitutional symptoms, which sometimes occurs in surgical cases. Its nature is uncertain, and many doubt if it be scarlatina.

out the eruption it may be impossible to come to a definite conclusion, but in doubtful cases it is best to act as if the graver disease were present (see Table X., § 111). *Diphtheria* has no rash, but the characteristic membrane appears on the throat (see Table X.). *Dengue* (*q.v.*) is accompanied by severe articular pains and a morbilliform eruption on the fourth day. The diagnosis is easier when the eruption is present. The scarlatinal rash is distinguished from the rare early eruption of *small-pox* by the fact that the latter starts in the groins or axillæ, and is not so bright a red. Enema rashes and *Epidemic Exfoliative Dermatitis* are sometimes mistaken for scarlatina. A *septic rash may be scarlatiniform*, but is distinguished by fever of a pyæmic type, and the presence of a septic focus, and by the absence of characteristic punctation. The erythema of *belladonna poisoning* is accompanied by great thirst and dilatation of the pupils.

Etiology.—Delicate children and puerperal cases have a strong predisposition to the disease. It is a highly infectious malady, especially at the outset and during early desquamation. The infection is propagated through the air, and carried by books and clothes, and is not infrequently conveyed by infected milk. The patient is generally regarded as infectious until desquamation has ceased from the palms or soles, a period averaging four to six weeks. One attack usually gives immunity for a lifetime. The disease is most prevalent during the autumn and early winter.

Prognosis.—It is always a grave disorder, because of the liability to complications, especially renal and ear disease. These dangers are avoided to some extent by keeping the patient in bed. Murchison used to teach that if a patient had been confined to bed three weeks, nephritis rarely supervened. This point is of the greatest importance, as it is as likely to follow slight as severe cases. After the fourth week there is little danger of nephritis. The aggregate case-mortality under five years of age is between 10 and 11 per cent., but it varies in different epidemics. Over five it is about 2 per cent., and is lowest between puberty and thirty years of age. The hæmorrhagic and malignant forms of the disease are those attended by most danger. The danger varies with the malignancy of the symptoms, especially the throat symptoms, and the characters of the rash. Persistent vomiting indicates a severe attack. Delirium at night is more or less usual in bad cases, but violent delirium is a bad sign. A temperature of over 105° F. is a serious symptom. The disease often carries off the healthy and well nourished, and sometimes spares the delicate patient; but in the *puerperal state* and in those with *tuberculous* tendencies the prognosis is very grave.

The *Complications* and *Sequelæ* are very important, for they may cause death even after slight attacks. A considerable change has taken place in the nature of the complications and sequelæ met with in modern

years, chiefly owing to improved methods of treatment. Acute nephritis and tonsillar abscess used to be regarded as the chief dangers, but at the present day Dr. Caiger[1] gives otorrhœa and otitis media as the chief *complications*, attacking nearly 15 per cent. of all cases, and leading in many to defects in hearing, intracranial abscess, septic thrombosis, and other septic conditions. Next in order come simple albuminuria, attacking 8·26 per cent., definite acute nephritis only 3·12 per cent., and cervical adenitis 5·66 per cent. Acute nephritis appears usually at the end of the third week, very rarely after the fourth, its advent being indicated usually by vomiting and dropsy. Various forms of arthritis (3·75 per cent.) and secondary tonsillitis (2·36 per cent.) are met with, chiefly among adults. The other proportions given by Dr. Caiger are various forms of abscess, 2·58 per cent., ulcerative stomatitis, 2·11 per cent. ; broncho-pneumonia and bronchitis together, 2·54 per cent. Brawny swelling in the neck, cancrum oris, and noma pudendi are occasionally met with, and may need prompt surgical measures. Acute endocarditis and septicæmia never occur among the cases treated at the Metropolitan Asylums Board's Hospitals, but they are still liable to complicate cases treated in their own homes. Among the *sequelæ* acute and subacute rheumatism (or a septic arthritis very closely resembling it) and chorea are perhaps the chief. Post-febrile mania is also an occasional sequela.

Treatment.—The general treatment is dealt with in §§ 391 *et seq.*, but it will be well to make a few remarks on the symptomatic treatment. The throat is best treated by a chlorine gargle (F. 18). In children this may be applied by means of a syringe or a spray ; nitrate of silver and other caustic applications so long in vogue only aggravate the condition. For the glandular swellings apply glycerine and belladonna, or warm fomentations. Brawny swelling of the neck is a serious complication, and must be dealt with by early incision and frequent carbolic fomentations. For convulsions give purgatives (croton oil) and a hot air or steam bath, or a hot-water bath. The patient should be kept in bed for three weeks, whether the attack be slight or severe, chiefly to prevent renal complications. As regards *immunisation*, the micro-organism of Scarlet Fever has not been isolated, so that we are not yet in possession of an antitoxin.[2] Nevertheless, in view of the numerous septic complications of the disease, antistreptococcus serum has been tried.[3]

The *hygienic* treatment is considered in §§ 389 *et seq.*, but a study of

[1] Dr. F. F. Caiger. The complications of scarlet fever, based upon an examination of 30,417 consecutive cases, treated in the Hospitals of the Metropolitan Asylums Board.—Allchin's " Manual of Medicine," vol. i., p. 286.

[2] Dr. W. J. Class has isolated a diplococcus having specific characters from the throat and blood of patients (the *Lancet*, September 29, 1900). Gordon and Klein have isolated a streptococcus.

[3] See a case complicated by acute otitis media treated by antistreptococcus serum, with recovery.—Low, *Lancet*, 1898, vol. i., p. 779.

the long list of infective complications given above will show how important it is to treat this malady in a large and airy hospital instead of at home. The throat is in a highly vulnerable condition, and it is doubtless through this portal that the various infective organisms find entrance.

§ **354.** III. **Erysipelas** (Synonyms : The "Rose," or "St. Anthony's Fire") may be defined as an acute febrile contagious disease, characterised by a progressive marginated redness and tumefaction of the skin, usually attacking the face, or the neighbourhood of wounds. (1) *The Stage of Invasion.*—After an incubation period of three to six days the advent is abrupt, as in small-pox and scarlatina. The temperature on the evening of the same day may be 103° to 104° F., or more. Vomiting is very common, and so also are muscular pains, especially pain in the back,[1] like that of small-pox. (2) The *Eruption* begins about twenty-four to thirty-six hours after the advent of fever, as a red spot on the face or at the site of an abrasion (which may be microscopic). It enlarges, spreads, becomes bright red, tender, and pits on pressure. The advancing edge is sharply defined and raised, the receding edge indefinite. The eruption may vary in duration from three or four days to a fortnight. Delirium at night is not unusual. Convalescence becomes established, and desquamation occurs in the course of one to three weeks. During this last stage albumen may appear in the urine, if it has not appeared before.

Diagnosis.—Erysipelas is to be diagnosed from *erythema* complicated by cellulitis, in which the margin is less raised, and there is less fever. In *zoster* of the first division of the fifth nerve vesicles occur in groups, are limited to one side of the face, and are unattended by fever.

Varieties.—(i.) Phlegmonous erysipelas or gangrenous erysipelas are severe varieties with suppuration or extensive sloughing. (ii.) Erysipelas neonatorum is a very fatal variety ; death may be due to peritonitis by inflammation spreading along the umbilical cord ; (iii.) Erysipelas of the fauces is a severe variety, the eruption spreading to, or starting in, this situation. The disease may spread to the larynx and cause fatal dyspnœa.

Etiology.—It is a highly contagious malady, and spreads through the air to a considerable distance. Persons are predisposed to it by wounds and unhygienic conditions. It seems possible that even in so-called idiopathic cases the virus is introduced into the system through a minute and hardly visible scratch. The presence of a wound is the strongest predisposing cause, and it spreads amongst surgical patients with great rapidity. As regards age, infants and persons over forty are most liable. The disease is due to a variety of strepto-

[1] This is not usually mentioned as characteristic of erysipelas, and the first case I was called to I mistook for small-pox on this account. I have never met with a case in which it was absent, excepting in second or third attacks of the disease.

coccus. One attack gives no immunity; on the contrary, it predisposes, and some elderly people are liable to an attack of facial erysipelas every year.

Prognosis.—The *usual course* is favourable, but the disease is dangerous in infancy or old persons, alcoholic or plethoric patients, and those affected with chronic diseases. Death may occur by coma or syncope, preceded by incessant vomiting; or by the supervention of complications. Hyperpyrexia, persistent vomiting, lividity of the rash, and typhoid delirium are untoward symptoms.

Complications.—(i.) Subcutaneous abscesses either on the scalp or in the neck; (ii.) diffuse cellulitis, ending often in extensive sloughing; (iii.) acute œdema of the glottis from the extension of the eruption (a very serious complication); (iv.) hypostatic congestion of the lungs (very common), bronchitis, lobular pneumonia, pleurisy; (v.) peritonitis, especially when it occurs after parturition, and gastro-enteritis; and (vi.) nephritis, acute or chronic, though it is not so common as after scarlet fever—are some of the commoner complications. (vii.) Meningitis used to be mentioned as a frequent complication on account of the frequency of cerebral symptoms in erysipelas; but meningitis does occasionally occur. (viii.) Chronic ulceration or skin eruptions often disappear after an attack of erysipelas near them. This has happened even in the case of ulcers which have been of a malignant character.[1] (ix.) Pyæmia and ulcer of the cornea are among the sequelæ.

Treatment (Hygienic Treatment, see §§ 389 *et seq.*).—A mild aperient should be given when the eruption comes out, and this should be followed by iron in large and frequent doses—20 minims of the liquor ferri perchloridi every four hours. Ammonia and bark are sometimes given; and in Germany large doses of quinine. Alcohol may be required in large quantities. Warburg's tincture is useful. Tannin or liquor ferri perchloridi are applied locally for the pharyngitis. *Local Treatment.*—Antiseptics, or a dusting powder of starch and zinc oxide, or a lotion of acetate of lead and extract of opium (4 grains of each to the ounce), should be applied to the inflamed area. The eruption may sometimes be stopped by a subcutaneous injection of carbolic lotion, 1 in 20, along the margin. Some say it may be stopped by painting the advancing edge with nitrate of silver or ichthyol. Daily inspection must be made for abscess whenever the skin is tense; scarification relieves the tension, and may prevent the occurrence of suppuration. *Immunisation* is now obtainable by the use of antistreptococcus serum, and cases have been cured in this way (§ 386).[2]

§ **355.** IV. **Small-pox** (Variola) is a highly contagious eruptive fever, the eruption passing through the stages of papule, vesicle, pustule, and

[1] It has been suggested to inoculate erysipelas as a means of cure in this condition (*vide* Carcinoma).

[2] Washbourn, *Lancet*, 1899. vol. ii., p. 1019.

scab. In small-pox UNMODIFIED BY VACCINATION the symptoms are as follows : (1) After a very definite period of incubation of twelve days, characteristic constitutional symptoms occur—viz., sudden advent of high fever (101° to 104° F.), with severe headache and *pain in the back.* The most noticeable features of this primary fever are the severity of the pain in the back (which, in my experience,[1] is present even in the mildest cases), and the frequent occurrence of vomiting. During the stage of primary fever there is, as a rule, no eruption, but in a few cases a prodromal rash makes its appearance. This may be (i.) erythematous, generally found in the groins or other folds, occasionally it covers the whole body ; (ii.) morbilliform, usually occupying the apron area, but also occasionally diffuse ; or (iii.) a hæmorrhagic erup-

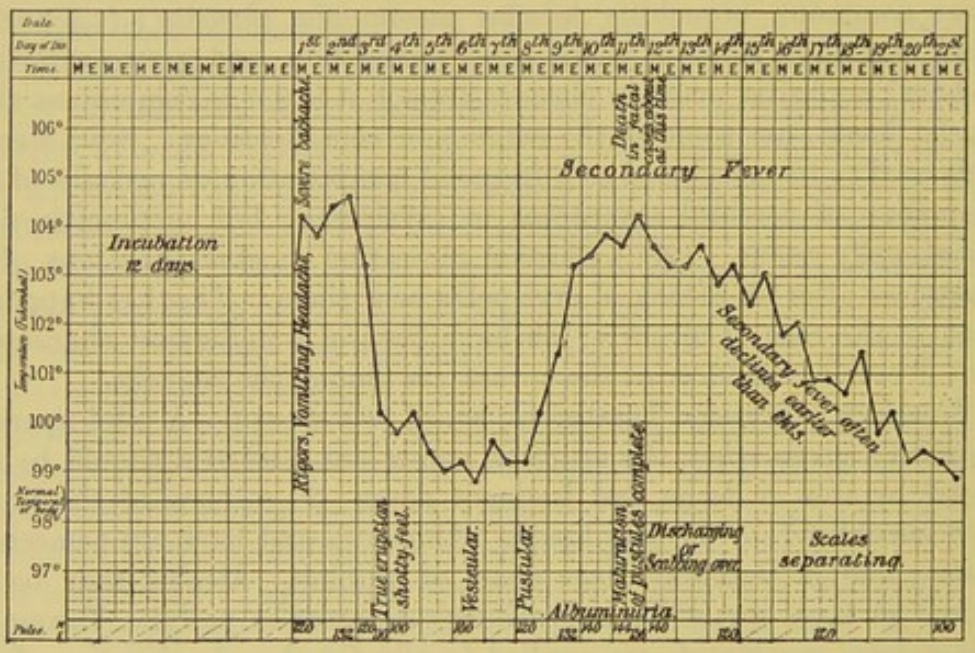


Fig. 95.—UNMODIFIED SMALL-POX.—Severe confluent case, unvaccinated, terminating in recovery. The various incidents are shown in the chart, for which the author is indebted to Dr. F. F. Caiger.

tion sometimes appears on the anterior surface of the abdomen and thighs. Prodromal rashes appear about the second day. The fever remains up until the third day, when the eruption appears. It then drops considerably—the patient, indeed, may feel comparatively well. About the seventh or eighth day, when the spots become pustular, a secondary or suppurative fever develops, which may be attended by rigors (Fig. 95). This secondary fever lasts six or eight days. (2) The eruption appears between the third and fourth day after the illness has commenced (fourteen days after infection), first as a crop of papules of *shotty hardness*, which can be felt even more readily than they can be seen, like small shots beneath the skin (Coloured Plate I.). They

[1] Report on the Warrington Small-pox Epidemic, by Dr. T. D. Savill ; Blue Book of the Royal Commission on Vaccination. Eyre and Spottiswoode, London, 1895.

first appear on the face and on the fronts of the wrists, and then the eruption travels downwards over the whole body, the abdomen, groin, and legs being least affected. The rash may occur in the mouth, pharynx, and larynx. Two days later the papules become vesicular. The eruption comes out in one crop, and is therefore never multiform in any given area of skin, as it is in varicella. Each vesicle enlarges, and by the sixth or seventh day has become pustular, presenting in typical cases, unmodified by vaccination, a depressed centre which is held down by a bridle, a feature known as umbilication. The next day (eighth day) the bridle ruptures, and each pustule becomes hemi-

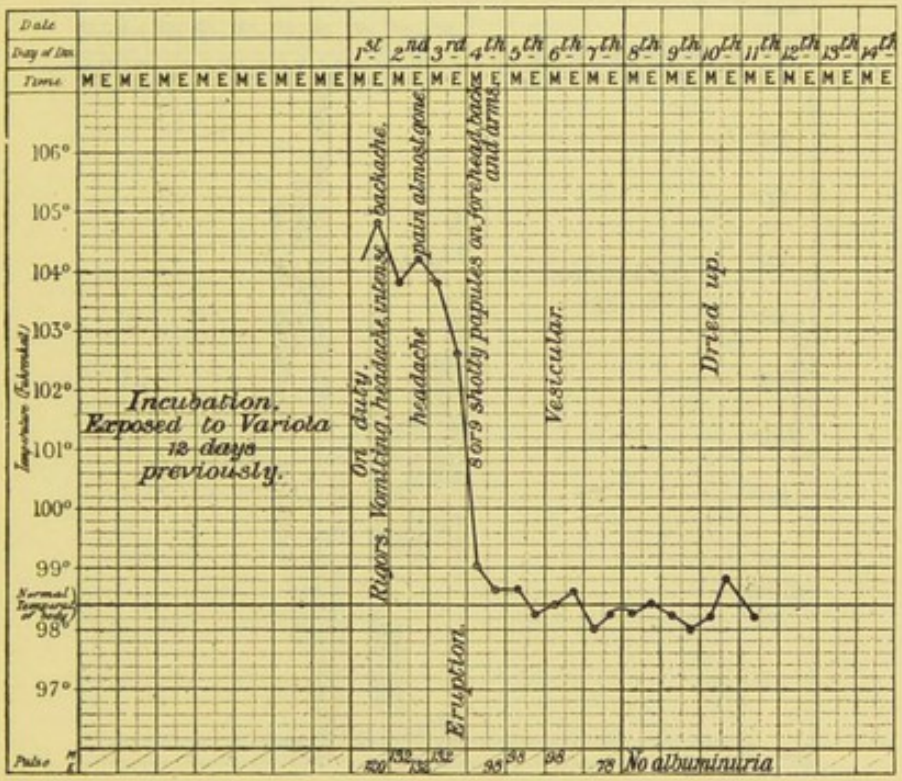


Fig. 96.—A mild case of MODIFIED VARIOLA occurring in a young woman, æt. 22, who had been vaccinated two years previously and who presented three visible cicatrices of the primary vaccination. Initial symptoms severe. **No secondary fever.** The author is indebted for this chart to Dr. F. F. Caiger.

spherical, about as large as a split pea, with an inflamed and indurated base. These pustules gradually dry into scabs, which fall off about the fifteenth to the twentieth day, leaving patches of congested skin, and in severe cases a pitted cicatrix. The extent of the eruption and the amount of inflammatory induration varies considerably. Sometimes only the face and wrists present a few spots ; sometimes the whole body is covered. The eruption on the legs always presents a proportionate retardation of development, since it appears last in this situation. Consequently, before certifying a patient as free from infection, the soles of the feet should be carefully examined, and found free from desquamation.

Modified Small-pox, or Varioloid (Fig. 96), is the term applied to the disease when modified by previous vaccination. The primary fever and early symptoms are indistinguishable from the unmodified form above described, and the eruption appears on the third day. Modified differs from unmodified small-pox in five ways: (i.) There is no secondary (suppurative) fever; (ii.) certain portions of the eruption abort and do not pass through all stages; (iii.) as a consequence, several stages of the eruption may occasionally be seen on the same portion of skin; (iv.) the general eruption may be very scanty, and may consist of not more than a dozen papules; and (v.) the constitutional symptoms are less severe.

Varieties.—It is sufficient to describe three varieties, according to the severity of the disease, the severity of the symptoms corresponding very closely with the character and extent of the eruption: (1) *Mild*,

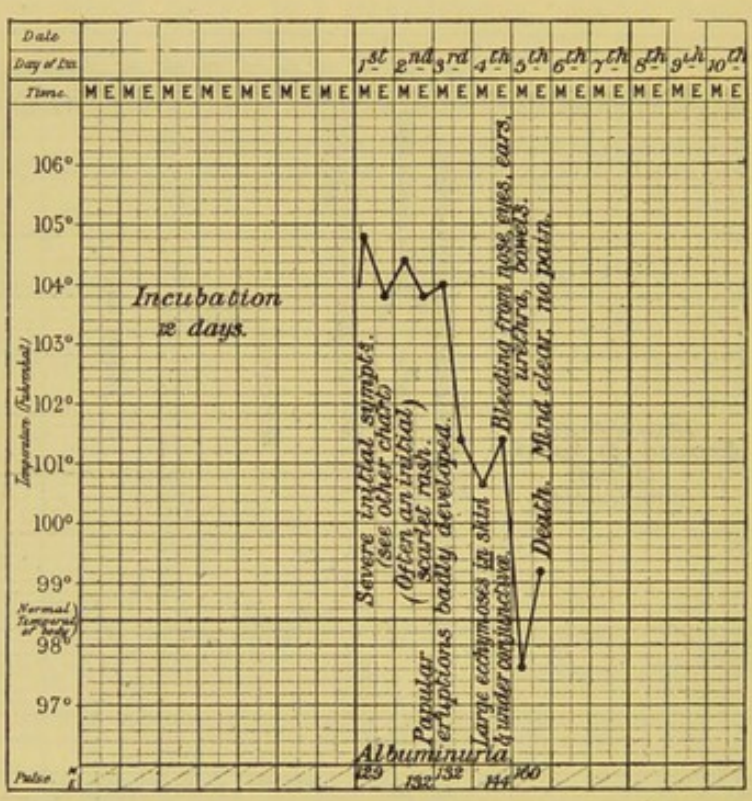
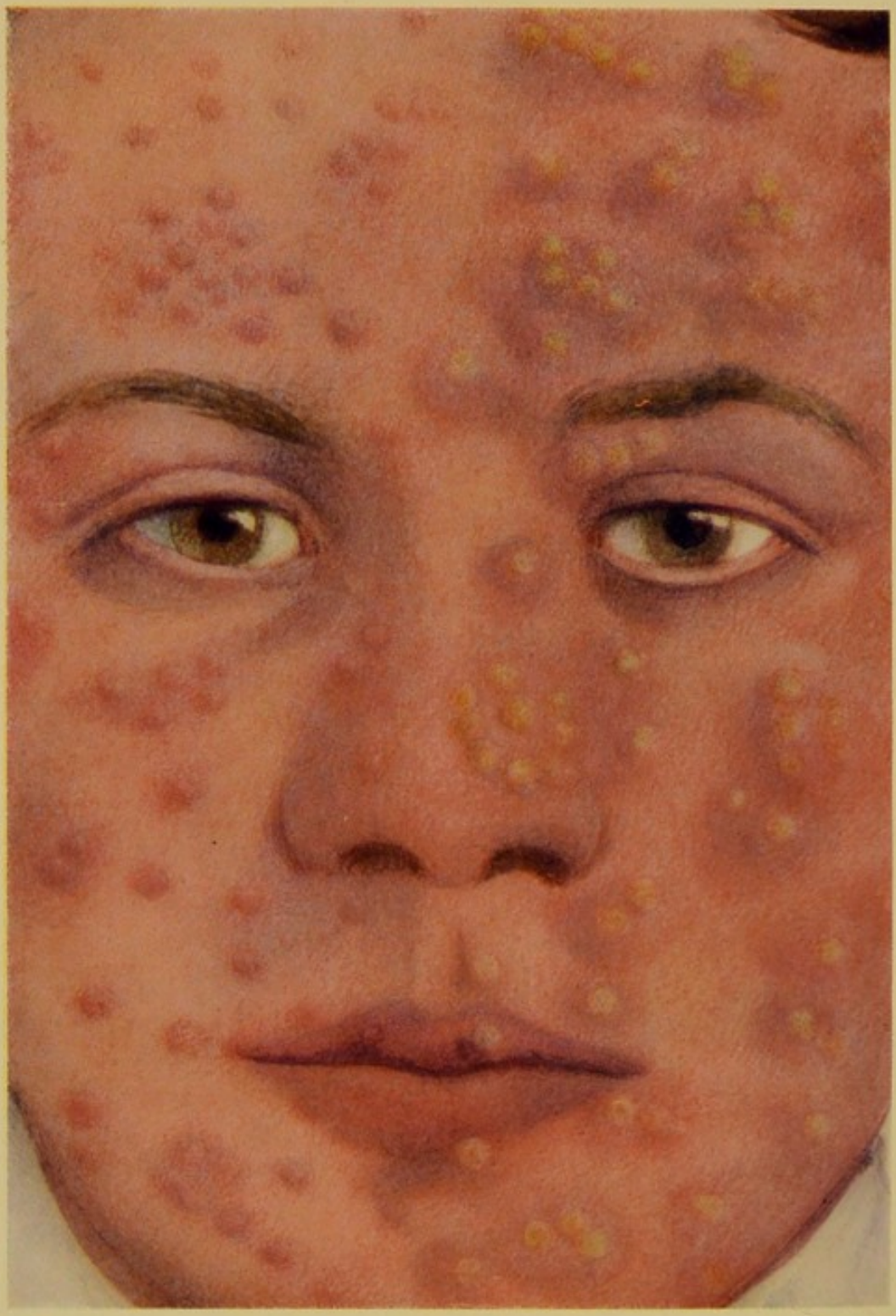


Fig. 97.—A case of Malignant Hæmorrhagic Small-pox (as distinct from those cases of confluent small-pox with hæmorrhages in the pustules).—Patient unvaccinated. Death occurred on the 5th day. The various incidents are shown on the chart, for which the author is indebted to Dr. F. F. Caiger.

(2) *Discrete*, and (3) *Confluent*. The *Hæmorrhagic* and *Corymbose* varieties are very severe, and, fortunately, rare; these adjectives referring to the character of the spots. In *Malignant* small-pox there are hæmorrhages into and beneath the skin, and death ensues early (Fig. 97).

Diagnosis.—In modern times, when nearly all cases of small-pox are modified by vaccination, the diagnosis is not always easy, and the greatest difficulty may be experienced in making a diagnosis even from acne. There are three important diagnostic features: (i.) Sudden advent of high fever; (ii.) headache, *backache*, and vomiting at onset of the disease, of which there should always be a history, even in the

PLATE I.



VARIOLA.

Right side of face (left of observer) represents the second day of the eruption. The other, pustular, side represents the sixth day of the eruption ; a few of the pustules show commencing umbilication.

Drawn from nature by Miss Mabel Green.

mildest cases ; and (iii.) the shotty character of the papules.[1] *Measles* is the disease which is most often mistaken for variola in the early stages of the case, and therefore two plates of these diseases are presented side by side (Coloured Plates I. and II.). Measles is distinguished by the redness of and the running from the eyes, and by the fact that in twenty-four hours the papules become smaller, whereas in small-pox they become larger. *Varicella* is distinguished by the inappreciable character of the premonitory constitutional symptoms ; by the eruption coming out in a succession of crops, so that one portion of skin may show several stages of the eruption ; and by the vesicles not being preceded or accompanied by any *shotty induration*. In febrile *roseola* or lichen, the fever lasts only twenty-four hours, the efflorescence appears all over the body at once, and it does not go on to any further stage. *Pustular syphilide* is chronic, and is unattended by any marked pyrexia.

Etiology.—The malady is highly infectious, but the microbe has not yet been discovered. Children, and especially infants, are particularly prone to the disease, and before the discovery of vaccination (A.D. 1776) it was a cause of considerably more than half the infantile mortality in Great Britain and other countries.[2] The poison is conveyed through the air to a considerable distance. Some believe it may be conveyed to a distance of miles, but this is very doubtful.[3] One attack confers immunity ; authenticated second attacks are extremely rare.

Prognosis.—Vaccination.—The case-mortality of small-pox in the present day is about 37 per cent. amongst the *unvaccinated ;* about 5 or 6 per cent. amongst all classes of the *vaccinated* taken together ; and about ½ per cent. amongst the *properly vaccinated.* The severity of the disease seems to depend almost entirely upon whether the patient has been recently and efficiently vaccinated.[4] In the healthy and recently

[1] See also a paper by the author " On the Diagnosis of the Early Stage of Small-pox," *Brit. Med. Journ.*, April 29, 1897.

[2] It is a fact of some interest that Warrington was the scene of an epidemic of small-pox in 1773, and the death-rate from the disease in that year was 26·5 per 1,000 (211 deaths, and, reckoning five inhabitants to a house, 8,000 inhabitants), all the deaths occurring in persons under nine years of age.—Dr. Thomas Percival, F.R.S., Phil. Trans., 1774, vol. lxiv.

In 1892-1893 Warrington was again visited by an epidemic, and the death-rate was then 1·1 per 1,000 of the inhabitants, who at that time had only about 1 per cent. unvaccinated persons among them.

[3] This question has been very hotly debated, but in the author's belief there are no definite evidences of small-pox being conveyed through the air to a greater distance than a few yards. It is extremely contagious, and all the cases supposed to be due to aërial spread can, if sufficient information can be procured, be explained by the conveyance of contagion either from person to person, or through some mediate agency.—Report on the Warrington Small-pox Epidemic, 1892-1893, pp. 64-77. Appendix to the Report of the Roy. Com. on Vaccination.

[4] The figures from the Warrington epidemic, 1892-1893, are very striking. In the *infected* houses there were 2,535 persons, and 2,223 of these persons had been vaccinated in infancy. Among these latter 521 (23·4 per cent.) were attacked, and 27 died, so that the case-mortality among them was **5·2** per cent. There were in the *infected* houses 107 unvaccinated persons, of whom 60 (56·1 per cent.)

vaccinated it is a comparatively trivial disorder, but in the unvaccinated, especially in infancy, it is one of the gravest diseases. The second factor in the prognosis is the question of *age;* and the official records of the unmodified outbreak in Warrington in 1773 show that of 211 fatal cases 166 were under three years of age. Alcoholism and plethora add to the gravity of the disease. The greatest danger is on the eighth day in the discrete, and on the eleventh day in the confluent form. As regards the *varieties*, the confluent, in which the rash comes out on the second day, and is very abundant, is much more dangerous than the discrete form. In the former the fever does not subside on the third day, and there is a great tendency to hyperpyrexia and complications. True hæmorrhagic small-pox is invariably fatal, but if hæmorrhage occurs into the vesicular or pustular rash, there is a slight chance of recovery. As regards untoward *symptoms*, the more severe the primary fever in the unvaccinated, the more severe will be the disease, but this is not so in the vaccinated; profuse salivation is a bad symptom; the case is grave if there be no swelling of the skin, and still graver if the swelling goes suddenly away; convulsions and other complications are unfavourable.

Complications.—(i.) Acute laryngitis or œdema glottidis is a common cause of death. Hypostatic congestion, pleurisy, empyema, and pneumonia are apt to occur. (ii.) The heart may be affected with peri- or endo-carditis; but myocarditis and granular degeneration are more common; (iii.) ophthalmia and consequent destruction of the eye is common in the East; painless corneal ulcers may form and perforate; and (iv.), for the rest, the complications are the same as those of scarlet fever, but nephritis is not so common.

Treatment.—It should be remembered that vaccination is capable of modifying the disease even after exposure to infection, because the incubation period of variola is twelve days and that of vaccinia only eight days. Vaccination may, therefore, be performed with efficacy during the first three or four days after exposure; and every member of an infected household should be vaccinated immediately the disease breaks out therein. As regards *therapeutic agents*, little is necessary in the Discrete form beyond a mild aperient and salines. In the Confluent form stimulants are necessary, and we must watch for complications, and meet them as they arise. If much salivation be present, it may lead to suffocation. The patient should be put into a warm bath and kept there for a considerable time. For sore-throat use gargles; for œdema glottidis, inhalations, or tracheotomy may be necessary. Many

were attacked, and 21 died, giving a case-mortality of **35·0** per cent. The figures also showed that, in proportion as the vaccination had been more efficient, the severity of the disease was less. Finally, among all the 667 cases which occurred in this epidemic, not one had been vaccinated or revaccinated within seven years of the attack.—Appendix to the Report of the Roy. Com. on Vaccination, 1894.

devices have been contrived to *prevent scarring* by the eruptions, such as powdering with zinc and starch powder, or with pulv. cretæ aromaticus, with a small quantity of disinfectant, or laying on lint soaked in glycerine and water, with a drop or two of carbolic acid. But all of these are of very doubtful benefit. There is, however, a method which promises to be really efficacious—namely, placing the patient in a room from which all but the red rays of the spectrum are excluded by pasting red paper over the windows. *Hygienic Treatment* is given in §§ 389 *et seq.*

The *Preventive Treatment* of small-pox is accomplished in the present day by three means—disinfection, isolation, and vaccination. Concerning the first two see § 389 ; for evidence of the efficacy of vaccination in the prevention and modification of small-pox see p. 497 and below. *Inoculation* used to be practised because it was found that the inoculated disease was milder, and gave just as much immunity from a second attack. Out of 20,000 inoculated by the brothers Sutton not one died. It was, however, declared illegal in 1840.

§ **356. Vaccinia.**—Vaccination is the production in a person of the disease called vaccinia, by inoculating him with the lymph taken from the udder of a cow or calf suffering from that disease. It was noticed in 1769 by a German that people engaged in the milking of cows were exempt from small-pox. Jenner, in 1775 and 1776 placed the subject on a scientific basis, and ascertained that the inoculation of a human being with the lymph taken from the unbroken vesicles on the udder of a calf suffering from vaccinia protected that person from small-pox. He was also the first to inoculate this disease (vaccinia) from person to person by taking the lymph from the vesicle on the arm which had matured on the eighth day after inoculation. Vaccination was made compulsory in 1853. In 1897 this law was repealed in response to an outcry among the public that syphilis and (?) other diseases could be conveyed from person to person in this way. Syphilis certainly has, in rare instances, been conveyed by arm to arm vaccination ; but by using calf-lymph this is entirely obviated ; and all public vaccinators now use lymph direct from the calf. Anyone who now goes before a magistrate and solemnly declares that he has " conscientious objections " to vaccination can procure exemption for himself and his children from compulsory vaccination.

Rules for Vaccination.—Calf-lymph is now universally used in Great Britain.[1] The best method is that of scraping the cuticle with a blunt-pointed lancet. The lancet should be kept scrupulously clean, and passed through a flame before using. The doctor's hands should be

[1] If human lymph is employed, it should be taken from a child, not an adult, and the child should be in good health, and free from any evidences or history of syphilis. The lymph should be taken from a vesicle before it becomes opaque, and before the areola has formed. It is better to vaccinate from arm to arm than from stored tubes.

clean, and the arm of the patient should be washed with soap and water before vaccination.

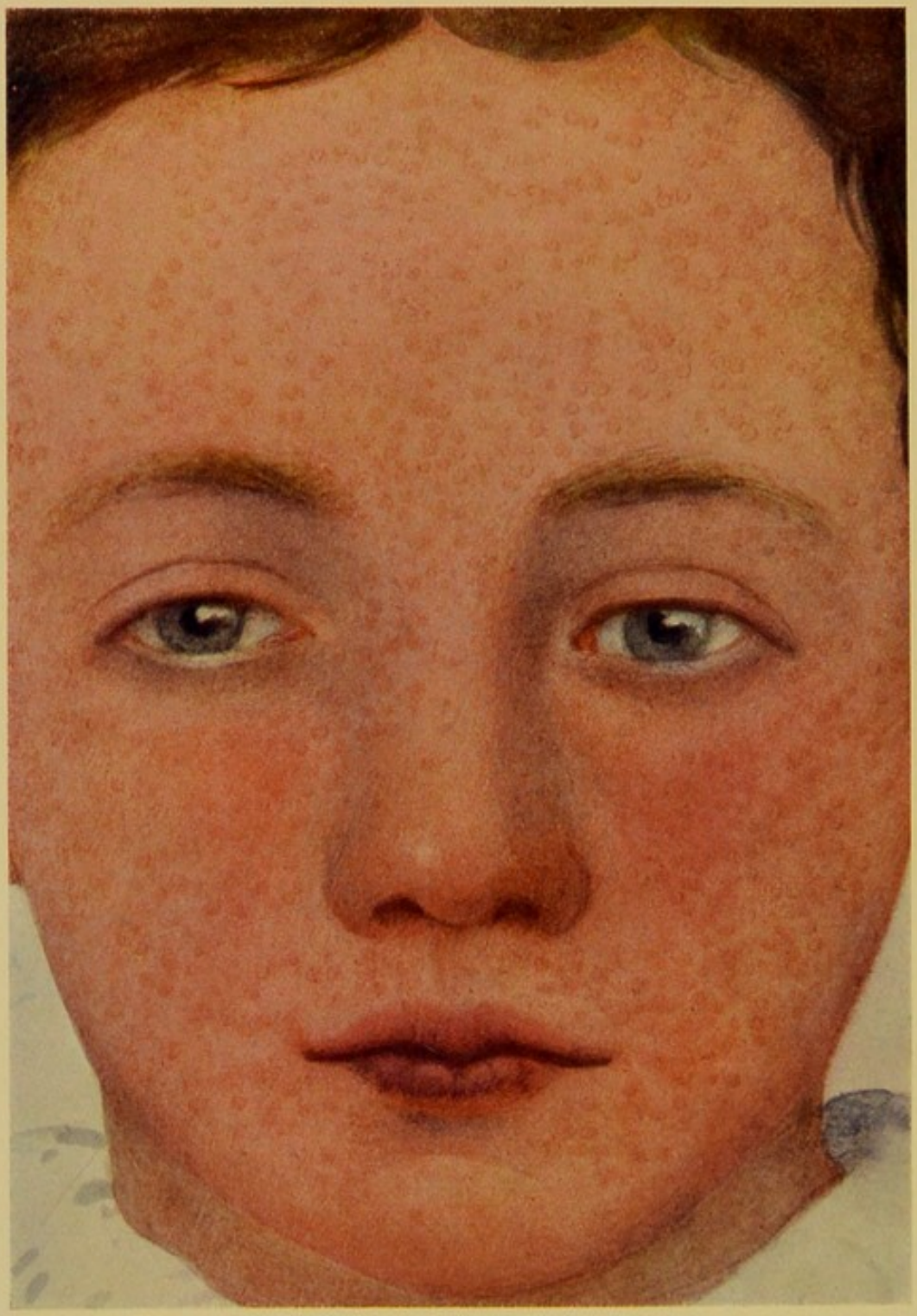
The Phenomena of Vaccination.—There are no symptoms for the first two days. On the second or third day a slight pimple, on the fifth day a bluish-white cupped vesicle appears, and on the eighth day (the same day of the week as that on which the operation was performed) the vesicle *becomes matured.* It should never become purulent, but the areola increases during the next two days. The contents then become cloudy, and after the tenth day they dry up ; the scab falls on the fourteenth or fifteenth day, leaving a pitted cicatrix.

The inquiries which the author made on behalf of the Royal Commission on Vaccination into the Warrington Epidemic (*loc. cit.*) went to prove (1) that efficient primary vaccination offers absolute protection against *infection* for the ensuing five or six years, and relative protection (gradually diminishing) for a considerable time ; (2) that primary vaccination lessens the *severity of the attack* of small-pox if contracted during the ensuing twenty or thirty years ; (3) that revaccination affords absolute *immunity from attack* during the ensuing five or six years, and relative protection for the rest of life ; and (4) that if everybody were vaccinated in infancy and again at twelve and twenty-one, small-pox would be exterminated.

§ **357.** V. **Measles** may be defined as an infectious febrile disease attended by catarrh of the respiratory passages, and by an eruption of minute elevated papules aggregated into irregular and often crescentic groups.

Symptoms.—(1) After an incubation period of seven to fourteen days, the pyrexia (Fig. 98) comes on abruptly, though not so suddenly as in scarlet fever, rising to 102° or 103° F. on the evening of the first day. The next day it usually declines a little. When the rash appears on the fourth day it rises again, remains up until the sixth day, and then falls by crisis. (2) The fever is attended by symptoms of coryza—for which, indeed, the case may be mistaken if the temperature be not very high. There are profuse lachrymation, running of the nose, and bronchial catarrh, the larynx and bronchi being specially involved. The fauces are sore, and mottled with redness, but not much swollen. (3) Koplik has described spots, which occur two or three days before the skin rash, found on the buccal mucous membrane opposite the molar teeth, and just within the angle of the mouth. They are not easy to see, and require a good light, when they appear as white stippling on a slightly raised purplish base. They occur in about 90 per cent. of all cases, and, consequently, lend great help to the diagnosis in the early stages. (4) The *eruption* appears on the third or fourth day (Coloured Plate II.). It consists of red, raised, well-defined flat papules, discrete at first, but afterwards running in patches. The colour is a reddish-brown, disappearing on pressure. The spots first appear on the *face*

PLATE II.



MEASLES.

The eruption, which is very plentiful, is eighteen hours old (second day of rash). Note the evidences of coryza in the eyes and nose.

Drawn from nature by Miss Mabel Green.

and side of the neck, where they are most abundant, and then pass downwards. Each papule reaches its maximum in about twelve hours, and then feels soft and velvety, thus differing from the early stage of small-pox papules. In twelve hours they begin to recede, and at the end of forty-eight hours to fade. By the eighth or ninth day the eruption of measles has completely disappeared, except that a brownish mottling of the skin may remain for some time after. Occasionally the macules become petechial. Sometimes the eruption suddenly disappears—the result of some internal complication, not, as is often supposed, the cause. The catarrh goes on increasing during the development of the rash, and they subside together about the seventh or eighth

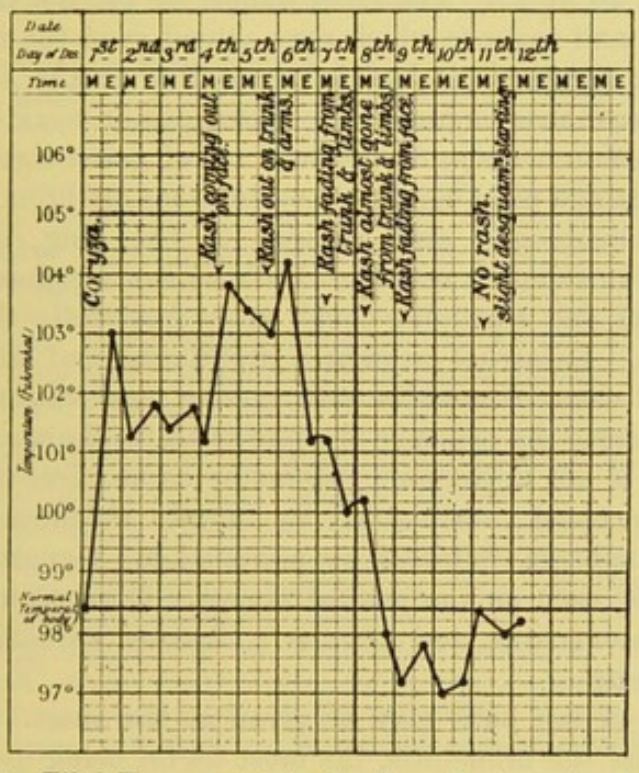


Fig. 98.—MEASLES.—Ethel H——, æt. 5 (under the author's care). Typical chart. The various incidents are shown upon the chart.

day, when convalescence commences. Slight desquamation of minute flakes, chiefly on the face, occurs sometimes.

The *Varieties* are less well defined than in scarlatina. The malignant or hæmorrhagic variety is very severe, and is attended by petechiæ and the typhoid state. The rash or catarrh may be absent in rare cases.

The *Diagnosis* from a severe "*catarrh*" is very difficult until the eruption appears. *Variola* often presents a difficulty, though the absence of catarrh, and the presence of pain in the back and vomiting, aid us considerably in diagnosing variola. The differences between the rashes are referred to above. *Urticaria* is somewhat like measles, but is recognised by the absence of catarrh and pyrexia. For the diagnosis of German Measles see § 358.

Etiology.—Measles is essentially a disease of childhood, and few escape. It is endemic in England, and outbreaks occur from time to time. The seasonal prevalence is in the spring and winter. The essential cause is probably a specific microbe, which has not yet been identified. It is conveyed chiefly by the breath and nasal mucus. Unlike scarlatina, it is as contagious before as after the eruption has appeared. One attack confers relative immunity ; second attacks are less common than in scarlatina, the majority of so-called second attacks being probably Rötheln.

Prognosis.—Measles is not a serious disease in itself, except in infancy. The case-mortality does not often exceed 1 or 2 per cent., though it may be as high as 10 or 12. The chief danger of the disease rests in the complications and sequelæ which may attend even the mildest case of measles. The prognosis is bad in proportion to the severity of the pyrexia and pulmonary symptoms. Strumous or weak children suffer most. Convulsions late in the disease are of grave significance. The most important and most common *complications* are bronchitis, broncho-pneumonia, pneumonia, and collapse of the lung, and diarrhœa, especially in the summer months. Phthisis is a recognised sequela ; it follows measles and whooping-cough more frequently than any other febrile disease. Catarrhal laryngitis, diphtheria, and laryngismus also occur. In all cases of measles with sudden aggravation of fever and no apparent cause, the presence of acute otitis media may be suspected. Cancrum oris is not uncommon, beginning as an ulcer on the internal surface of the cheek, surrounded by intense inflammation. Soon a black slough appears, followed by perforation. Gangrene may occur in other parts, such as the genital organs. Other complications are ophthalmia, stomatitis, and rhinitis. Caseous bronchial glands are common sequelæ.

Treatment.—A hot bath may be given at the onset. Remedies are directed against the bronchitis, the most useful being ipecacuanha and liq. ammoniæ acetatis, F. 53 (General Treatment, see §§ 389 *et seq.*).

§ 358. VI. **Rötheln,** or German Measles (synonyms: Rubeola, Epidemic Roseola, Hybrid Measles), may be defined as an acute contagious disease, characterised by sore-throat, catarrh of the respiratory passages, and an eruption of the skin, consisting of irregular hyperæmic patches, which afterwards become confluent. Clinically, it may be said to represent a combination of measles and scarlatina. Undoubtedly, many so-called cases of Rötheln are identical with measles.

The *Symptoms* vary somewhat in different epidemics. (1) After a period of incubation, variously stated to be from seven days to two or three weeks, the temperature rises to 100°, 101°, or 102° F. This is accompanied by sore-throat and coryza. Sometimes the glands in the neck and elsewhere are swollen, the most characteristic being the concatenate and occipital groups. When the eruption comes out, the other symptoms are considerably aggravated, but the whole attack rarely lasts more than a week. The rash may be the first indication of the disease, as the primary fever is sometimes so slight. (2) The eruption appears on the first, second, or third day, and consists of minute round or oval rose-red patches of hyperæmia, varying in size from a pin's head to a sixpence, very slightly raised, never papular. The rash at the outset is like that of advanced measles.

In a day or two it becomes confluent, or nearly so, and the whole skin presents a scarlet hue, so that the case may be mistaken for scarlatina. The eruption first appears on the face, and at the end of twenty-four hours the whole body is involved. It lasts from two or eight days, and the severity of the attack is in direct ratio to the duration and severity of the eruption. It is sometimes followed by slight desquamation. The disease has to be *diagnosed* from *scarlatina*, in which there is no catarrh, and no " measly " eruption at the beginning of the attack. In *measles* there are no enlarged glands, no special involvement of the tonsils, but little sore-throat, and no extensive confluence of the rash. In non-specific *roseola* (rose-rash), there are no catarrh and no sore-throat.

Etiology.—It is mainly a disease of childhood, but sometimes attacks adults. It is not so contagious as either scarlatina or measles (Murchison). One attack confers immunity.

Prognosis.—It is a more trivial disease than measles, though there is a liability to some of the same complications as occur with measles and scarlatina (*q.v.*). The occurrence of severe bronchitis and tonsillitis are the worst symptoms we have to deal with.

The *Treatment* is like that of measles.

§ **359.** VII. **Dengue** is a highly contagious fever, most commonly met with in India and America, characterised by a fugitive scarlatiniform eruption on the skin and severe articular pains. The incubation period of dengue is very short, probably a few hours or a day or two. Great epidemics occur in Africa and on the coasts of India. The fever is of a sudden onset, and ranges from 102° to 105° F. It is accompanied by intense headache, with extremely severe pains in the joints or limbs, much aggravated by movement. This primary fever lasts about forty-eight hours, and subsides by crisis. At this stage the skin may be covered with a bright red flush. During the next one to two days there is an interval of apyrexia, with freedom from pain. Then the secondary fever appears, with a return of the pains in the limbs. Both, however, are less severe than in the primary stage. A universal mottling of the skin, starting on the hands, somewhat resembling measles, though never papular, accompanies the secondary fever. As it subsides in a day or two, slight branny desquamation occurs.

Diagnosis.—Dengue is known from *scarlet fever* by its being rarely associated with sore-throat, by the severe articular pains, and by its occurring in hot weather, and, later, by its characteristic temperature. *Acute rheumatism* is rare in the tropics, has no rash, and has profuse sweats.

Prognosis.—As regards life, the prognosis is excellent; the case-mortality is extremely small. Death rarely, if ever, occurs; if so, it is from such complications as weak heart or hyperpyrexia, in the enfeebled. In most cases the patient is well in eight days. Some have painful and swollen joints for some time after the fever has gone.

The *Treatment* does not differ from the ordinary hygiene necessary in fevers. The patient should be kept in bed to avoid chill. For the pain in the limbs, belladonna, antipyrin, morphia, and in chronic cases iodide of potassium, may be given. The subsequent anæmia and enfeeblement are sometimes troublesome.

§ **360.** VIII. **Typhus** (synonyms: Contagious Typhus, Exanthematic Typhus, Hospital, Gaol, and Ship Fever; Germans call it Enteric Fever Typhus) may be defined as a contagious fever, lasting fourteen days, with an eruption on the skin consisting of subcutaneous mottlings and petechial spots, with a great tendency to the typhoid state. Its disappearance from our midst is a good illustration of the triumphs of hygiene. It is due to a highly contagious specific poison, which can be propagated only where overcrowding, deficient ventilation, squalor, and destitution exist. As these conditions have disappeared, typhus has gradually died out, though it is still occasionally met with in Glasgow, Liverpool, parts of Ireland, and other places where the poor are crowded into back-to-back houses.

Symptoms.—(1) After an incubation period, which varies considerably, but is rarely longer than twelve days, the temperature rises rapidly for two or three days

to 103° to 105° F., or more, at which it remains until the fourteenth day. It starts somewhat abruptly with chilliness, rarely with rigors. There is severe headache and extreme prostration, so much so that on the second day the patient is unable to walk or stand. Drowsiness is common, and there is a typical aspect of heavy stupidity. At the end of the first week headache gives place to delirium, and this is followed by drowsiness and coma. The temperature continues to rise until the seventh day, and then falls slightly during the ensuing week, and usually ends by crisis on the fourteenth day. (2) The spleen is enlarged and tender. (3) The eruption appears usually on the fourth or fifth day, first on the back of the hands, arms, folds of axillæ, and in front of the chest and abdomen. It has usually two elements, which vary in their proportion : (*a*) Subcuticular mottling, certain portions of the skin appearing hyperæmic, with fading margins ; (*b*) reddish-brown spots, having a definite but irregular outline, varying in size from a pin's head to three lines, very slightly elevated at first, and in the course of two or three days becoming petechial, so that they will not disappear on pressure. One attack usually confers immunity. The patient ceases to be infectious the second day after the evening temperature is normal.

Diagnosis.—(1) *Typhoid fever* was originally confused with typhus, and it is chiefly owing to the observations of Sir William Gairdner that they are now differentiated. Typhoid differs from typhus in (i.) the insidious onset ; (ii.) the course of the temperature ; (iii.) the different eruption ; and (iv.) the diarrhœa and pea-soup stools. (2) In *measles* the eruption resembles the typhus spots, and appears at the same date, but in typhus it does not start on the face, it is never preceded by catarrh, is never papular, and becomes petechial. (3) Some *malarial* fevers present considerable difficulty, but they have no eruption. (4) *Uræmia* and other causes of coma may be mistaken for it. (5) *Pneumonia*, meningitis, and other causes of the *typhoid state* may be confused with typhus. (6) *Plague* sometimes presents a rash identical with typhus, but the glandular swellings of the former are distinctive.

Etiology.—The disease is chiefly met with in middle and advanced life. It is due to a specific contagium which has never yet been isolated. This is given off in great amount, especially during the second week, from the breath and exhalations of the patient ; but the infecting distance would seem to be very short, for it is destroyed by free ventilation. Doctors frequently contract it by bending over the patient ; Dr. Charles Murchison, who did so much for the study of this and other fevers, contracted typhus twice, and thus incurred the heart disease of which he died. The poison can only be produced where there is overcrowding, deficient ventilation, and personal squalor ; and the strangest circumstance connected with this disease is that it cannot *be developed* under any other conditions, and therefore it only appears at long intervals. Thus it gave rise, with considerable colour of truth, to the theory of the origin *de novo* of infectious diseases. The malady is predisposed to by a general debility, and it is therefore commoner in times of famine and distress.

Prognosis.—Case-mortality, 10 per cent. : between the age of fifteen and twenty-five, 4 per cent. ; over fifty, 50 per cent. Thus the age of the patient greatly influences the mortality. Typhus is always a serious disease, especially in the plethoric and alcoholic. It terminates fatally in three ways : (i.) Degeneration of the cardiac muscle, which is a very common accompaniment of the disease ; (ii.) coma, from the toxic state of the blood ; or (iii.) asphyxia or hypostatic congestion of the lungs. Untoward symptoms are (i.) weak, irregular, or intermittent pulse, or other indications of cardiac weakness ; (ii.) an abundant rash, with high fever ; (iii.) early and protracted cerebral signs or protracted hiccough ; (iv.) all complications, especially pulmonary. Of the *complications* and *sequelæ*, (i.) the pulmonary are the worst, especially broncho-pneumonia and hypostatic congestion of the lungs ; œdema glottidis and pleurisy are less common. Other complications are (ii.) hyperpyrexia and meningitis ; (iii.) femoral and other thromboses ; (iv.) gangrene of the extremities from embolism, bed-sores, and pyæmic abscesses ; (v.) cardiac weakness, which may remain for a long time, on

account of the granular degeneration of the muscle; (vi.) post-febrile mania; and (vii.) paralysis of various parts.

Treatment.—Hygienic treatment is essential (§§ 386 *et seq.*), especially free ventilation. Therapeutic treatment is chiefly symptomatic. It is sometimes the practice to give an emetic at the outset. Mineral acids may assist the digestion. Stimulants in most cases are not necessary, but they must be given if the pulse is weak or irregular, or if the extremities are cold.

§ 361. **Anthrax,** or Malignant Pustule (synonyms : Woolsorters' Disease, Anthracæmia, Splenic Fever—under which term the disease is registered in the Registrar-General's returns—Charbon, Carbunculus Verus). The primary lesion consists of a solitary vesicle at the seat of inoculation. As the base of this becomes transformed into a central slough, the contents become hardened, and around this a zone of vesicles arises. Pasteur showed that it is due to the anthrax bacillus, a relatively large organism which was one of the first to be isolated.

This disease, which has a marked and prolonged vesicular stage, is most usually situated on the dorsum of the hand or arm, occasionally on the face. It affects woolsorters, furriers, felt-makers, ragsorters, and others who come in contact with animals or their hides or fur; or it may be conveyed by the stings of insects. The incubation period is twenty-four to seventy-two hours. First a papule forms at the seat of inoculation, which rapidly enlarges, and becomes on the second day a vesicle, with serous or hæmorrhagic contents. On the third day this bursts, leaving a raw exuding surface, which, on the fourth day, turns to a dry black slough, surrounded by a zone of intense inflammation slightly raised above the surface. Upon this inflammatory zone there appears, also on the fourth day, a characteristic ring of small red vesicles. The œdema extends around, and the lymphatics and the glands inflame. The pain is usually very slight, and no pus forms until about the tenth day, when the slough begins to separate. The constitutional symptoms vary considerably, and bear no proportion to the local mischief. The pyrexia may be so slight as not to interfere with the patient's ordinary avocation, and it may not come on until some days after the local signs. Usually, however, it is severe, comes on early, and soon assumes a typhoid character.

Intestinal and *Pulmonary* types are also described, according to the method of infection. In the former intense vomiting and diarrhœa occur, with great prostration and cramps, with, in some cases, cyanosis and dyspnœa, and towards the end convulsions and spasms. The spleen is enlarged. In the latter, which is caused by inhalation of diseased wool or hair (*woolsorters' disease*), there are urgent dyspnœa, and pain in the chest of sudden onset. The temperature rises to 102° or 103° F., and death may occur with profound collapse in twenty-four hours. Sometimes delirium and convulsions, or diarrhœa and vomiting, occur.

Diagnosis.—It may have to be diagnosed in the first place from the sting of an insect, from various conditions which lead to solitary vesicles or bullæ on the second day, from erysipelas (if on the face), lymphangitis, and other causes of œdema. The occupation of the patient assists us, but a diagnosis may be made by examining the serum or secretion of the sore, stained by Gram's method (Chapter XX.), under the microscope. The bacillus anthracis, which is the cause of the disease, is thus readily discovered.

Prognosis.—The mortality varies with the position of the primary lesion, being 40 per cent. when this is situated on the neck or face, and 12 per cent. when situated elsewhere.

Treatment.—The local lesion should be freely incised if seen early, and the wound irrigated continuously with carbolic lotion (1 in 20). The lotion may also, with advantage, be injected into the tissue surrounding the part, and repeated every four hours, due care being taken to watch for carbolic poisoning (carboluria, etc.). The patient's strength must be supported. Sclavo has prepared an antianthrax serum, which is still on its trial.

§ 362. **Glanders** (Synonym : Equinia) may be defined as a contagious febrile disease attended by a discharge from the nostrils, and sometimes an eruption on the skin, due to the inoculation of the bacillus mallei, in a person attending to

Horses affected with the disease. The eruption, which only occurs in Acute Glanders, consists of a general erythema, on which a crop of pustules of hemispherical shape appear in the course of a few days or hours. They vary in size between a lentil and a florin. There are also nodules of granulomatous material in the subcutaneous tissue and muscles, which usually suppurate, leaving large foul ulcers. The other symptoms are (i.) a copious discharge of viscid, semi-purulent matter from the nostrils; (ii.) pains in the limbs and joints; and (iii.) high fever, with rigors and prostration, passing on to the typhoid state.

In Chronic Glanders (Farcy) the pyrexia and constitutional symptoms are absent, and the cutaneous eruptions (erythema, pustules, and nodules which leave ulcers and sinuses). The discharge from the nose may be the only sign.

Diagnosis.—The pustules of acute glanders resemble those of variola, but they are larger, and not umbilicated, and the temperature in glanders does not fall when the rash—in those cases which present a generalised pustular eruption—comes out.[1] The pain and swelling of the joints and limbs bear some resemblance to acute rheumatism, and still more to pyæmia.

Treatment.—Vaccination with small doses of dead bacilli is advocated, and has been tried in a few cases. At present the disease is extremely fatal. In Farcy or Chronic Glanders the death-rate is 40 or 50 per cent.[2] Iodide of potash, aconite, mercury, iron, arsenic, and strychnine have all been tried, and good results have accrued from the injection of small doses of mallein.

GROUP II. CONTINUED PYREXIA.

§ **363.** In this group the pyrexia tends to assume a CONTINUED TYPE —*i.e.*, it runs a continuous course except for the slight normal diurnal variation (§ 347). This group is distinguished from Group I. by the absence of an eruption, at any rate during the first four days of the illness. It is distinguished from Group III. mainly by the course of the pyrexia, though aberrant types of one group are found in the other.

Common.		*Rare in this Country.*	
I. Enteric fever	§ 364	VII. Plague	§ 370
II. Diphtheria	§ 365	VIII. Malta fever	§ 372
III. Influenza	§ 366	IX. Yellow fever	§ 371
IV. Rheumatic fever, pneumonia, and various other inflammatory disorders, usually attended by local signs	§ 367	X. Epidemic cerebro-spinal meningitis	§ 373
V. Whooping cough ..	§ 368	XI. Relapsing fever ..	§ 374
VI. Mumps..	§ 369		

Enteric Fever, which may be taken as a type, may present no other symptoms than *the characteristic pyrexia*. The rash, when present, is ill-marked, and does not appear till the second week of the disease. In Diphtheria there is the characteristic *throat lesion*; in Influenza there are *pains in the limbs* and a more sudden advent; in Pertussis the *characteristic cough*; and in Mumps the *parotitis*. Various Microbic Reactions may aid us in the diagnosis. Cholera (§ 220)

[1] The author once notified a case of this kind as small-pox, and the case passed as such through the hands of two of the most experienced medical officers of the Metropolitan Asylums Board, the mistake not being cleared up until after death, and a full investigation had been made of the *circumstances under which the disease arose*. It was then ascertained that the patient was a stableman, attending on glandrous horses.

[2] Dr. Sims Woodhead in Allbutt's "System of Medicine," vol. ii., p. 524, 1st edit.

and Dysentery (§ 219) might also be included in this group, but the pyrexial disturbance is quite a subordinate feature compared with the intestinal manifestations.

It was formerly the custom to speak of Enteric Fever, Typhus Fever (Group I.), Relapsing Fever, and Febricula, as the "Continued Fevers of Great Britain." Of these practically only the first still prevails amongst us. Febricula is generally identical with enteric. Relapsing fever has only occurred in times of famine, and Typhus has disappeared with improved hygiene amongst the masses.

§ **364. Enteric** or **Typhoid Fever** [Synonyms: Abdominal Typhus (Niemeyer), Pythogenic Fever (Murchison)] may be defined as an acute specific fever of about three or four weeks' duration, with a tendency to diarrhœa and the typhoid state, often attended by successive crops of rose-coloured spots, and due to a specific microbe (the typhoid bacillus of Eberth, see Coloured Plate IV. and Fig. 111.). A characteristic ulceration of Peyer's patches occurs.

Symptoms.—(1) The period of incubation is usually about ten days, but it may be shorter or longer. The onset is insidious, differing in this

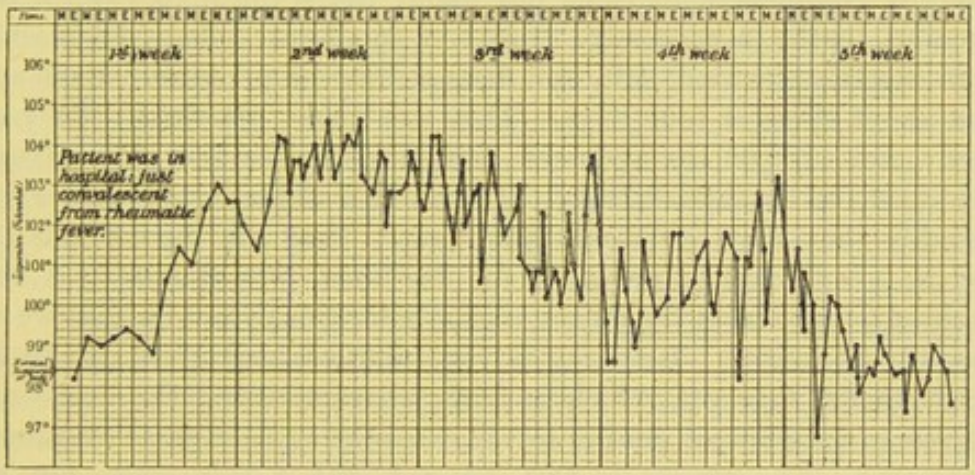


Fig. 99.—Enteric Fever (typical chart).—Henry H——, æt. 22 (under the author's care) was in hospital when he developed the enteric fever. There was apathetic mental condition, great feeling of illness and headache, watery pea-soup stools, and bronchial catarrh. The chart shows the continued character of the pyrexia in the second and third weeks, with gradually increasing remissions in the fourth and fifth weeks.

respect from the fevers in Group I. The most important early symptom is headache, otherwise there are simply malaise and lassitude, usually with constipation, and perhaps slight abdominal pain. The typical typhoid chart (Fig. 99) is the most characteristic feature of the disease, and until the discovery of the Widal reaction we were mainly dependent upon this for the diagnosis of the malady. In the *first* week it is "ladder-like," gradually rising with diurnal remissions until it reaches, about the end of the first week or ten days, its highest point (103° to 105° F.). During the second stage, which may last a week or more, it remains continuously high, the diurnal remissions being no more than those which are met with in health. As the disease progresses, these daily remissions become gradually more and more marked.

During defervescence, usually about the fourth week, first the morning temperature, and then the evening temperature, gradually become normal. These features are so constant as to afford a means of detecting the stage which a case has reached. Convalescence may be said to be established when the evening temperature has been normal for two successive nights. (2) Some diarrhœa is usually present after the first week, and the stools are of a characteristic pea-soup or yellow ochre colour. This feature is of very little value as a means of diagnosis, while a patient is on milk diet. In about half the cases there is no diarrhœa at the onset, and the bowels are confined. (3) The spleen is generally tender and enlarged throughout the disease, being frequently palpable even without the patient taking a long breath. Tympanitic distension of the abdomen is common, especially in the second and third weeks, and there is often pain and gurgling on pressure in the right iliac fossa, though great care should be used in attempting to elicit this symptom, as the intestinal wall is thinned by disease. (4) The eruption generally commences to come out about the seventh to twelfth day (average, tenth) in successive crops[1] of small rose-coloured lenticular spots, slightly elevated, soft, and disappearing on pressure. Each spot lasts about three or four days. They are never petechial. They are chiefly met with on the abdomen, sometimes on the rest of the trunk, very rarely on the face or limbs. The number of these spots varies considerably, but they are rarely abundant. They are very small, and may be overlooked or mistaken for flea-bites. (5) Malaise is a very constant feature from the outset, and it is for this symptom that we are generally consulted. Lethargy is very marked, and gives rise to the aspect (*facies typhosa*), which is fairly characteristic; the drowsiness deepens to semi-stupor, and in severe cases the typhoid state eventually supervenes. The tongue is first covered with a thin white fur, the edges and tip being red; in the second week the fur clears off, and the tongue becomes glazed and dry, or red and smooth. Shallow transverse fissures are often seen on it. Sordes collect on the teeth. Several *varieties* of the disease have been described, but they are not of much importance. Occasionally the disease commences quite suddenly, with symptoms of great severity. The "ambulatory" form is so called because the patient is able to keep about while suffering from it. Perforative peritonitis may be its first manifestation.

Diagnosis.—Until recently the diagnosis of typhoid was often a matter of excluding all other possibilities, and even then was largely a matter of conjecture. But at the present time we have a valuable test in Widal's reaction, for which purpose a specimen of the patient's blood must be procured and sent to a laboratory (see Chapter XX. for

[1] This fact may be revealed by enclosing each of the spots which appear on one day by a circle, next day by a triangle, and so on, by a nitrate of silver paint or aniline ink.

method). The diazo test is also of service, though not so certain. Undoubtedly many slight cases of typhoid are overlooked or spoken of as *Febricula* (*vide infra*). Slight cases are also apt to be mistaken for *Influenza*, which, except for the pulmonary symptoms, the more sudden advent, and brief duration, much resembles mild typhoid. The other *specific fevers* in this group may also have to be excluded. In most cases of typhoid there appears early in the disease a generalised *bronchial catarrh* and *hypostatic congestion* of the lungs, and nothing is commoner than to mistake enteric fever, in its early stages, for pulmonary congestion or bronchitis, and severe cases may be mistaken for *pneumonia*. These pulmonary disorders should be recognised by the relative absence of the prostration, and the diarrhœa, enlarged spleen, etc., of enteric. In severe cases of typhoid, early delirium may occur and suggest *meningitis ;* but the latter is recognised by (i.) the retracted abdomen ; (ii.) the irregular and sighing respiration appearing early in the disease ; and (iii.) the headache persists longer, and may concur instead of alternating with the delirium (Murchison) ; signs of intracranial pressure also supervene, such as ptosis, squint, optic neuritis, and other local paralyses. *Acute Miliary Tuberculosis* is a disease which sometimes so closely resembles enteric that, as Niemeyer[1] remarks, they can only be differentiated in the dead-house. The positive signs of typhoid are wanting, and the presence of tubercle is suggested by (i.) the intermittent character of the temperature and its prolonged course ; (ii.) the lung symptoms are much more marked ; (iii.) the rapidity of the breathing is out of proportion to the other signs of illness ; and (iv.) the pallor and lividity of the face and the rapid emaciation are also more prominent features. *Malignant endocarditis* is recognised by (i.) the intermittent character of the temperature (usually), and (ii.) the cardiac signs. *Pyæmia* is differentiated by the wide range and irregularity of the pyrexia (§ 383).

Etiology.—Enteric fever is now known to be due to a specific microbe which has been isolated. *All matters which the patient discharges from his stomach, bowels, and bladder are infective.* Most epidemics are due to the contamination of the water supply by sewage. The disease has also been traced to the eating of oysters,[2] to ice-creams, and to the milk supply. To produce the malady the microbe must be introduced into the alimentary canal ; thus, nurses and friends contract the disease by handling the bed-pans and sheets, or any other articles which have been contaminated by the fæces and urine. The excreta become more virulent after standing from twelve to twenty-four hours. The malady is most prevalent in the autumn and early winter ; and Pettenköfer has found by several years' observations that typhoid outbreaks are

[1] "Textbook of Practical Medicine." This was before the discovery of bacillus and Widal's reaction.

[2] Sir William Broadbent.

favoured by (i.) a rapid falling (after a rise) of ground water, that is to say, a well-aerated moist soil; (ii.) a certain temperature of the earth; and (iii.) pollution of the soil by animal impurities. One attack does not necessarily confer immunity, as second attacks are not very uncommon. The malady is chiefly met with in young people between ten and thirty years of age.

Prognosis.—The case-mortality varies in different epidemics from 5 to 20 per cent. The prognosis is more favourable in the young. It is always a serious disease on account of the numerous complications, prolonged course, and its exhausting nature. The usual duration is about three or four weeks, though it varies from ten days to six weeks even without relapses, which are by no means infrequent. *Untoward Symptoms.*—The height and the continued character of the fever are the best guides to the severity of the attack. Many of the fatal issues would be avoided if it were remembered that slight attacks require just as much care as severe ones, being liable to be attended by hæmorrhage and perforation if the patient does not remain at rest. The prognosis is grave when the fever remains at about 104° F. throughout the second week, and especially if the diurnal remissions do not increase, as they should do, in the third week. It is also grave when there are vomiting, except at an early stage, urgent diarrhœa at any time, severe tympanites, or hæmorrhage. A sudden fall in the temperature suggests hæmorrhage or the occurrence of peritonitis. The most common *complications* are: (1) Those of the lungs, and, as previously mentioned, bronchial catarrh and hypostatic congestion are practically symptoms of the disease. Pneumonia and pleurisy also occur. (2) Hæmorrhage, due to the ulceration of Peyer's patches, occurs in 8 or 10 per cent. of the cases. (3) Perforation. (4) Peritonitis, due either to the spread from the ulceration or to perforation, is a frequent complication, and it is sometimes peculiar in being latent—that is to say, unattended by the pain which is so characteristic of that disorder. Its occurrence can then only be recognised by (i.) vomiting; (ii.) great aggravation of the already existing prostration; (iii.) a small rapid pulse (120 to 140); (iv.) immobility and distension of the abdominal walls; and (v.) a sudden fall of the temperature. (5) Other complications are thrombosis of the femoral vein, local suppurations and inflammations, such as parotitis, periostitis, pericarditis, cholecystitis, cancrum oris, and laryngeal ulceration. As *sequelæ* multiple abscesses, dementia, peripheral neuritis, phthisis, and miliary tuberculosis may occur.

The temperature may rise again after convalescence has begun. Such a rise may be due to too liberal a diet, excitement, or constipation. It may, on the other hand, be due to a relapse. *Relapse* occurs in about 10 per cent. of all cases. There is usually an apyrexial interval of about five days, but sometimes the temperature has never dropped

satisfactorily. The second attack is usually less severe and shorter than the first. As many as five relapses may occur.

Treatment.—The microbe of enteric fever, by virtue of its special "proclivity," attacks Peyer's patches in the small intestine, which become inflamed, swollen, and ulcerated. Consequently, there are three indications: (*a*) to prevent peritonitis, hæmorrhage, or perforation by rest and suitable diet; (*b*) to maintain the strength of the patient; and (*c*) to neutralise the toxin of the microbe (by serum-therapy, see § 388). *Hygienic.*—Absolute rest is of the highest importance, and when the diagnosis has become established the patient should not be allowed to turn in bed. Grave responsibility rests upon the nurse in this respect, for perforation may occur in changing the draw-sheet. In contradistinction to the febrile diseases already described, typhoid fever patients may be treated in a general ward, but great care must be taken to keep all utensils and the thermometer apart and disinfected. The stools must be burnt or immersed in izal directly they are passed, and 1 in 20 carbolic must be added to the urine. All linen must be first steeped in an antiseptic such as 1 in 20 carbolic, or lysol or izal, for several hours, and then boiled. All pots, pans, mugs, etc., must be boiled after each occasion on which they have been used. *Diet* is also of prime importance. Milk is the staple article, and not less than 2½ pints a day should be given. It is advisable to add barley-water or lime-water to prevent the formation of large curds. The addition of 3 grains each of sodium bicarbonate, magnesium carbonate, and sodium chloride to a cup of milk or sodium citrate in the proportion of 2 grains to the ounce of milk, has a like result. If milk disagrees, give whey, egg albumen, or butter-milk. Clear soup, chicken-broth, and beef-tea may also be given. No solids should be taken on any account until at least one week after the temperature is normal. Predigested foods are of great aid to promote assimilation, especially if the tongue be heavily furred, and pepsin is said to have quite a specific effect on the disease, though it probably acts in that way. It may be given thus: Essence of pepsin, ℥ xxx.; dilute nitro-hydrochloric acid, ℥ v.; glycerine, ad ℥i. The bowels must be regulated by enemata on alternate days, if required.

Hydrotherapy.—Judging from the good results which this line of treatment has given on the Continent and in America, its use is worthy of more trial than it has hitherto received in England. It is applied in three ways: (i.) By cold or tepid sponging; (ii.) by the ice-pack; and (iii.) by the bath, which is used as a routine when the temperature is over 102·2° F. At the Johns Hopkins Hospital it is usual to give a bath at 70° F. every third hour, if the temperature is above 102·5° F. The patient remains in the bath for about twenty minutes, during which he is rubbed with the hand or a suitable rubber, and is then taken out, wrapped in a dry sheet, and covered by a blanket.

Medicinal.—Internal antiseptic remedies are largely used. Such are perchloride of mercury, carbolic acid, creosote, izal, lysol, β-naphthol. These are not of much value, and treatment by drugs is chiefly symptomatic. If profuse, the *diarrhœa* must be checked by enemata of starch and opium (℥ss. of tinct. opii to ℥iii. of mucilage of starch); or liq. morphinæ, ♏ xx., with dilute sulphuric acid, ♏ x., every three or four hours. If this fail, give acetate of lead, bismuth carbonate, or bismuth salicylate. If *perforation* occurs, laparotomy should be performed immediately. For *local peritonitis* apply heat to the abdomen, give opium in large and frequent doses, iced milk, and very small doses of brandy or champagne. If the abdomen is *tympanitic*, apply turpentine fomentations and give turpentine internally. *Hæmorrhage* should be checked by the administration of opium, and absolute rest must be enjoined. To maintain the strength, *stimulants* are generally called for, but they should not be given as a matter of routine. *The pulse is the best indication* for their administration; they must be stopped if hæmorrhage occur. Cinnamon oil has been strongly recommended by Dr. F. F. Caiger in 1 to 2 minim doses throughout the illness.

Prophylactic Treatment is based upon a knowledge of the origin of the disease and the mode of its introduction into the system—viz., by the mouth (see § 390). The incidence of typhoid in a community is a fair index of the purity of its water-supply. Preventive and remedial inoculation are now on their trial (see §§ 386 *et seq.*), but there is great promise of success.[1]

Febricula is a term somewhat loosely applied to any condition which is chiefly evidenced by a slight degree of fever and malaise. It was formerly classed as one of the four Continued Fevers of Great Britain. Murchison[2] showed that when it assumed the form of a definite illness it was in reality a mild attack of typhoid fever; and that when death, which was a very rare event, did ensue, the characteristic lesions of that disease were found in the intestines.

§ **365. Diphtheria** (Synonym: Membranous Croup[3]) is a contagious fever, characterised by a membranous exudation on the fauces, due to the Klebs-Lœffler bacillus (Chapter XX.). *Symptoms.*—The incubation period is uncertain, but it is said to be about two to six days. (1) The onset is usually gradual (extending over a day or two), but in some cases it is sudden. The fever is often high, but in others it may not exceed 102·5°, and it may even be quite normal (Bristowe) in asthenic cases. The height of the temperature is no guide to the severity of the disease. *The temperature chart does not conform to a regular type*, but Fig. 100 represents a common case. (2) Sore-throat is present from the beginning, and frequently dysphagia. On one or both of the tonsils there is a characteristic patch of creamy white, "wash-leather" like membrane surrounded by a red areola. If removed, this leaves

[1] Sir A. E. Wright, the *Lancet*, March, 1901.

[2] "The Continued Fevers of Great Britain," second edition. Murchison, 1873.

[3] Diphtheria was probably included under the term "malignant sore-throat" or "cynanche maligna" prior to the year 1855 (about).

bleeding points. As the patches extend they run together, and may spread on to the soft palate and uvula. Their occurrence on the soft palate is a diagnostic feature of great value from quinsy. The patient complains that the neck feels very stiff, and *the glands at the angle of the jaw are swollen*. This glandular enlargement dates from the recognition of the attack, or even before, and is of importance in the diagnosis. The membrane spreads to the larynx and bronchi in grave cases, and it may also spread upwards to the nose (especially in children). An ichorous discharge from the nostrils in a child lying prostrate and fretful in bed is very characteristic of diphtheria. It

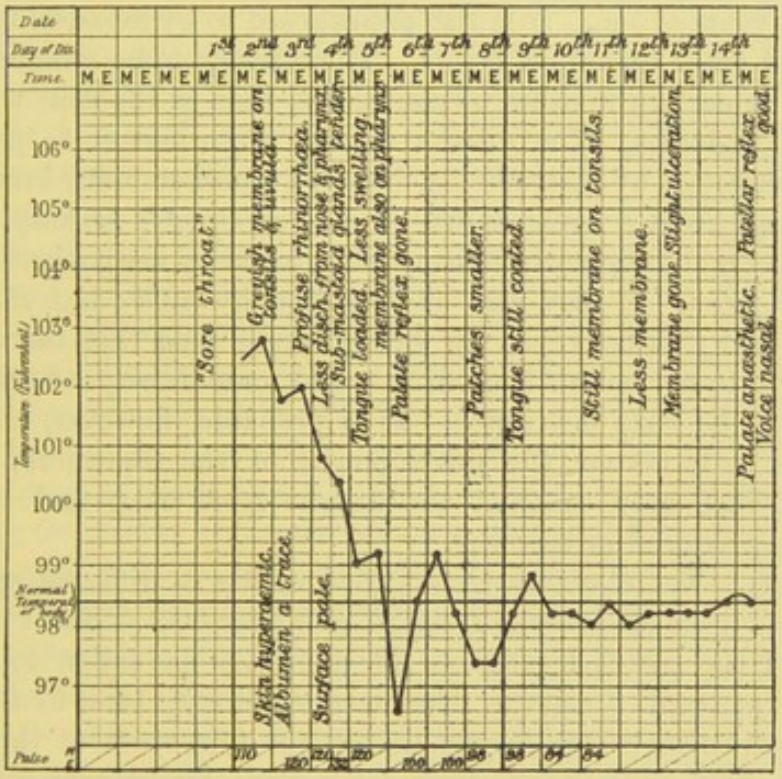


Fig. 100.—DIPHTHERIA.—Male, æt. 9. An ordinary case of faucial diphtheria without implication of larynx. The palate was still anæsthetic one month later. Not followed by paralysis. The different events are indicated on the chart, for which the author is indebted to Dr. F. F. Caiger.

may, in rare cases, involve, or start upon the conjunctivæ, genitals, or on the skin at the angles of the mucous orifices. (3) Albuminuria is present in about half the cases (Caiger) towards the end of the first week. There may be hyaline and epithelial casts in the urine which is sometimes suppressed. (4) Prostration and anæmia are very marked, but the mind usually remains clear to the end, even in lethal attacks. In the asthenic type of the disease lassitude and prostration are extreme. Eruptions on the skin are occasionally met with, the commonest being an erythema, or purpuric spots in fatal cases.

The *Diagnosis* of diphtheria may be made by finding the Klebs-Lœffler bacillus in swabbings taken from the seat of the disease. The

diagnosis of the sore-throat caused by tonsillitis, scarlatina, and diphtheria presents certain difficulties, and is given in the tabular form (§ 111). *Follicular tonsillitis* is distinguished by the absence of the wash-leather-like patches on the tonsils, nose, or larynx, and the presence of higher fever. There may also be a history of previous attacks. *Scarlatina* is distinguished by its abrupt onset, its higher fever, its rash, strawberry tongue, and generally the absence of membrane from the throat. Simple "*croup*" (catarrhal laryngitis) is distinguished by the absence of patches in the throat, and the absence of albuminuria. *Membranous croup* is always diphtheritic.

Etiology.—The disease occurs chiefly in the young, and especially under ten years of age. It is also predisposed to by measles, whooping-cough, and other infectious fevers. There seems to be a more marked tendency in certain families to contract it than is the case with other infectious maladies. The disease spreads from person to person; it may be conveyed by instruments, infected handkerchiefs, kissing, etc., and it *hangs about a house or district with remarkable tenacity.* Nurses and medical men frequently contract the disease by the patient coughing into their faces. It may also be conveyed by milk; but there is no evidence that it is conveyed by water. Some hold that the disease is predisposed to by bad air from drains, and undoubtedly a form of sore-throat may be thus developed. It is probable, however, that these conditions only favour the development of the diphtheria bacillus. Human beings may contract the disease from cats, and epidemics have been produced in this way.

Prognosis.—The case-mortality varied widely in different epidemics, but it used to be an average from 25 to 50 per cent. Since the introduction of the serum treatment the mortality has fallen to approximately 10 per cent. (Caiger). The malady is chiefly fatal by the spread of the membrane to the larynx during the first week of the disease. After the first week death may take place by asthenia, toxæmia, syncope, or complications. Pharyngeal cases are, in adults, usually mild, and recover in a week or so, but severe cases last two or three weeks. Great care is required even in the mildest cases, lest the membrane should spread, and on account of the complications (*q.v.*). The clinical *varieties* according to Caiger (*loc. cit.*), are (1) *mild faucial* cases, mostly met with in adults; (2) *severe faucial* cases, with a tendency to extension, chiefly met with in young children; (3) "*croup*" or *laryngeal* diphtheria, where the air passages are alone affected; (4) *nasal diphtheria*, where the nasal passages alone are affected; (5) diphtheria of *other parts*—cheeks, gums, tongue, lips, conjunctivæ, genitals, wounds, etc.—conditions generally associated with faucial or laryngeal diphtheria. *Untoward Symptoms.*—The prognosis is unfavourable when the temperature is low in spite of severe local lesions; when epistaxis or any form of hæmorrhage occurs, or there is much

albumen in, or suppression of, the urine ; and when the patient is young (see above). Rapid extension of the membrane is also a grave sign, especially when it extends down the larynx, leading to croupy cough, dyspnœa, and cyanosis ; and death takes place in such cases from asphyxia, unless they are promptly relieved (see below). The chief danger in the second week is cardiac dilatation and failure, and the pulse and heart should be closely watched at this time. Of the *complications*, certainly the commonest is paralysis, due to peripheral neuritis. It attacks nearly 20 per cent. of the cases (Caiger), and comes on usually about the fourth week, sometimes later. The characteristics of diphtheritic paralysis are : (i.) It starts usually in the palate, and therefore nasal voice or dysphagia is the earliest symptom, and fluids taken are returned through the nose. The paralysis is progressive, and tends to involve many, sometimes all, of the muscles of the body. Next in order we may get loss of accommodation, squint, loss of patella reflexes. Among the most serious paralyses are those of the diaphragm and intercostals. (ii.) Motion and sensation are simultaneously affected, though often the sensory symptoms are the first to be observed. The attitude assumed in marked cases is very characteristic—the little patient shambles into the room with drooping shoulders and head bent forward from weakness of the neck muscles. (iii.) The heart is frequently affected, which sometimes leads to sudden death if the patient be not kept absolutely still. The sounds are weak, and the rhythm rapid and irregular, often of the " galloping " type. Vomiting frequently accompanies such cardiac signs. (iv.) In general terms there is a tendency to complete recovery in a few weeks, though sometimes death occurs apparently from involvement of the vagus. (2) Broncho-pneumonia, so frequent formerly, only attacks about 4 per cent. under modern methods of treatment, but cardiac dilatation, probably due to myocarditis, is a frequent occurrence. (3) Nephritis and dropsy during convalescence are very infrequent, and permanent lesions of the kidney are rare. Otitis media is not uncommon.

Treatment.—The indications are (*a*) to neutralise the toxin in the blood ; (*b*) to inhibit the local process ; and (*c*) to strengthen the constitution to resist the disease. (1) Thanks to the recent advances in science, we now have a powerful *antitoxin* for the control of the disease, and if given early it is capable of completely neutralising the toxin and arresting the disease. It is a good general rule to give it in all cases, though there are three possible exceptions[1]—viz., (i.) very slight cases, when under constant medical observation ; (ii.) cases seen too late in the course of the disease, when membrane is obviously separating (the antitoxin must be given at the earliest possible moment ; doses and methods are given, §§ 386 *et seq.*) ; and (iii.) cases where there is marked irregularity and feebleness of the heart, which require caution. (2) For

[1] See also Washbourn, *Lancet*, October 14, 1890, p. 1019.

the *local* treatment, nitrate of silver, hydrochloric acid, and other caustics have been much used, but are now condemned as useless and harmful; and papain, with borax, had a reputation which no longer exists. Disinfectants are certainly useful, applied by syringing, or spraying, or swabbing every hour or so, with 1 in 10,000 corrosive sublimate, or carbolic acid (½ per cent.), but the best results have been obtained with chlorine (F. 18), formalin ½ per cent., chinosol ⅛ per cent., or sulphurous acid, used by syringing or spraying. Warm inhalations every half-hour and hot applications to the neck give much relief. When the nose is affected it must be syringed with the same disinfectant solutions. When the larynx is much affected the question of tracheotomy has to be considered. Statistics used to be very unfavourable, the mortality being 70 or 80 per cent. But in the present day over 70 per cent. of the cases operated on recover, and tracheotomy should be performed promptly whenever the breathing is difficult owing to laryngeal obstruction. The results are more satisfactory when it is *done early*, and all laryngeal cases should be closely watched for the epigastric retraction during inspiration which indicates inspiratory dyspnœa. It is then essential to keep the patient in a steam-tent. (3) The *constitutional* treatment consists of stimulating and supporting measures. In asthenic cases perchloride of iron and potassium chlorate are generally given, with wine, beef-tea, and abundant nutriment. In the inflammatory forms give diuretics and potassium chlorate, with salines and laxatives. In all cases the patient should be kept quite still in the recumbent position for fear of the heart failure, which is apt to occur, especially about the tenth to the twenty-first day.

§ **366. Influenza** is an epidemic fever attended by considerable prostration, and usually by catarrh, and a tendency to the development of local inflammations. It has been known for at least five centuries, and has occurred at various times in great epidemics, separated sometimes by many years' interval.

Symptoms.—(1) After an incubation period of one to six days the patient's temperature goes up in the course of a few hours to 102° and 104° F. The onset is frequently attended by shivering. The fever generally ends in one to five days with profuse perspiration, and is attended by the pains in the limbs which form such a characteristic feature of influenza. (2) "Catarrh" usually accompanies the fever—*i.e.*, there are redness and watering of the eyes, running at the nose, sore throat, sneezing, and tightness of the chest. (3) Malaise and prostration out of proportion to the amount of pyrexia occur. (4) Some cases have only the three symptoms just mentioned, but there is a great tendency to local complications. The *type* of the disease therefore varies according to the physiological system mainly involved. (i.) The *respiratory* tract is very frequently attacked, and in that case

bronchitis and pneumonia complicate the disease. (ii.) The *circulatory* system may be affected by endarteritis, and occasionally, but not often, other gross lesions. The neuro-vascular apparatus is, however, specially prone to suffer, causing tachycardia and bradycardia, palpitation, flushings, faintings, perspiration, dyspnœa, and the like. (iii.) Involvement of the *alimentary* tract may be evidenced by gastro-enteritis, diarrhœa, vomiting, jaundice, etc. (iv.) *Eruptions* on the skin may occur, especially urticaria, erythema, or rose-spots like measles. (v.) The *nervous* system, especially in the aged, is affected for long after the disease, and neurasthenia is particularly apt to supervene. Peripheral neuritis is frequent, and many cases of disseminated sclerosis are attributed to this disease. Depression, prolonged mental dulness, and other symptoms are met with.

The *Diagnosis* is not difficult in typical cases, especially when the disease is prevalent. The short duration of the initial symptoms and the usual absence of rash are sufficiently characteristic. The severe pains in the limbs are very typical.

The *Etiology* is still obscure, though a specific microbe has been isolated, which occurs chiefly in the secretion of the respiratory tract. It is certainly epidemic, but it is still doubtful whether it is contagious in the true sense. It appears to break out in several persons spontaneously in certain localities. One attack confers no immunity from a second. As regards predisposing causes, age has no influence, nor have seasons of the year, nor sanitary conditions. Old and young, rich and poor, all are attacked alike.

Prognosis.—The case-mortality is about 1 per cent. among the old and young together. In middle-aged and elderly people the respiratory type is very apt to end fatally with pneumonia, and undoubtedly many cases presumed to be primary pneumonia are really secondary to influenza. It is fatal only through its complications. The disease itself is usually trivial, and the patient soon recovers. Relapses are not infrequent. The *complications* consist of those mentioned above under types of the disease. The *sequelæ* are prolonged weakness, peripheral neuritis (sometimes attended by tremors), otitis, orchitis, meningitis, and mental derangement.

Treatment.—During the attack the patient should be kept in bed in view of the complications and sequelæ. Sodium salicylate, antipyrin, and antifebrin will reduce the fever, and relieve the pains in the limbs, and ammoniated tincture of quinine is a justly popular remedy. Cinnamon oil has been advocated. For the rapid heart liquor arsenicalis is recommended (℥ iv. t.d.s.).[1] It is well to keep elderly people indoors (or away from infection) during the prevalence of the disease, as they run greater risks from its effects.

[1] Sansom, "Effects of Influenza on Heart and Circulation," *Lancet*, October 21, 1899, p. 1075.

§ **367. Rheumatic Fever, Pneumonia, and other Inflammatory Disorders,** which usually present well-marked **local manifestations.**—The three fevers just described are those most commonly met with in England, in which the pyrexia may run a continued course, and which have no eruption during the first four days. But it must not be forgotten that certain inflammatory disorders may give rise to pyrexia of a continuous type, and that the usual local signs of these disorders may be absent, at the time when the patient is first seen. It will be well, therefore, to mention those which might be mistaken for an acute specific fever.

(*a*) Obscure (so-called) Local[1] Inflammatory Diseases are mostly met with as complications secondary to fevers. They can usually be detected by a thorough examination of all the organs in the body (§ 349). Nevertheless, certain cases of (1) *pericarditis*, or (2) *pneumonia* or *pleurisy*, may be latent—*i.e.*, the usual physical signs may occasionally be wanting or overlooked. (3) Various affections in or around the *throat and nose ;* (4) some *abdominal* disorders, such as perihepatitis, inflammation of the mesenteric glands or pancreas, etc. ; (5) certain rare cases of *sarcoma* and *carcinoma ;* or (6) inflammation of the *meninges*, tuberculous or epidemic, may also give rise to an elevation of temperature sometimes unattended by marked local symptoms.

(*b*) Certain obscure General Inflammatory Disorders are attended by pyrexia, which may similarly give rise to difficulties in diagnosis. (1) In *rheumatic fever* and *acute gout* the pyrexia is nearly always continuous. The joint lesions are the cardinal feature in these cases ; but it must not be forgotten that acute rheumatism may commence with inflammation of the pericardium (the structure of which very much resembles that of a joint), and that the joint lesions may not be apparent for several days. (2) There are several conditions special to infancy and childhood which are attended by continued pyrexia : (i.) *Infantile paralysis* (acute anterior poliomyelitis) is attended at its outset by a considerable rise in temperature, which may last for several days or weeks, and be accompanied by restlessness, peevishness, etc. ; (ii.) *rickets* from time to time may have a slight degree of fever, accompanied by a generalised tenderness and profuse perspiration ; and, as just mentioned, (iii.) *meningitis*, tuberculous or epidemic. (3) *A nervous or hysterical pyrexia* has been described, and I have seen the temperature go up in an erratic manner, at odd times, in nervous subjects. But while admitting that the nervous system plays a very important part in the production of fever (as witness the rigors and pyrexia which follow catheterisation), it is difficult to believe that there is not a compound cause in operation in such cases. Only a thorough *post-mortem* and bacteriological

[1] The word " local " is here used in a qualified sense. Many of these diseases with local manifestations are now known to be due to a general infection.

examination would enable us to be certain that none of the many obscure foci of inflammation above mentioned were present.

§ **368. Whooping Cough** (Pertussis) is an acute specific infectious malady, characterised by paroxysmal attacks of coughing, followed by a long noisy inspiration (the whoop). The period of incubation is from three to fourteen days (average five.) (1) The onset is marked by a preliminary catarrh, or running from the nose and sometimes the eyes, attended not infrequently by paroxysmal dyspnœa and drowsiness. This premonitory stage lasts only for twenty-four or forty-eight hours, and may be overlooked. (2) Paroxysms of coughing then set in. Each paroxysm consists of a series of short sharp coughs, followed by *a loud inspiratory "crow,"* through the narrow chink of the half-closed glottis, and it is often followed by vomiting—a diagnostic feature of value when we have to depend on the mother's account of the case. As the result of the coughing, large quantities of stringy mucus are expectorated. After some days the face remains somewhat swollen, and subconjunctival hæmorrhages may also occur. There are no physical signs characteristic of the malady, unless, as some maintain, enlarged bronchial glands can be detected by percussion or ausculto-percussion over the root of the lung. Bronchitic sounds are generally present in greater or less degree. (3) The constitutional symptoms vary considerably in severity. In many cases they are absent, the temperature being hardly elevated, and the child being apparently quite well between the attacks of coughing. In typical cases, however, there is slight pyrexia. The *Diagnosis* is not difficult, since the paroxysms of coughing are very characteristic.

Prognosis.—All the symptoms increase for the first ten days, then remain stationary for a few days, and decline during the ensuing week. It is usually one of the trivial ailments of childhood. It is severe only in very young children, in the weakly and the rachitic, or by reason of its complications, of which there are three chief ones—viz., bronchitis, broncho-pneumonia, and convulsions. Ulceration of the frenum of the tongue is common, due to the forced protrusion against the teeth in the act of coughing.

Treatment.—In view of the fact, which does not seem to be sufficiently known, that children living near gas-works and bleaching-works do not get the disease, it would be worth while to try inhalations of coal tar. Belladonna is, in my experience, the most useful amongst the drugs, though nothing seems to cut short the malady. It should be given in large doses ; children will stand 10 to 20 minims of the tincture. Antipyrin, hydrocyanic acid, carbonate of ammonia, ipecacuanha wine, conium, and the bromides have also been recommended. How long a child remains infectious is an important practical question. Infection lasts as long as the characteristic cough is present, but when, as in some cases, it is hard to say whether the attacks are typical or not, it is best to take three to four weeks from the commencement of the disease as the duration of the infection.

§ **369. Mumps** (**Acute Epidemic Parotitis**) is an acute febrile infectious disorder, characterised by inflammatory swelling of one or both parotid glands. The period of incubation is from one to three weeks.

The *Symptoms* are moderate fever (102° F.), subsiding in the course of a week, stiffness of the jaw, and difficulty of swallowing, due to swelling and inflammation of the parotid gland. One side is first affected, and is succeeded by the other in about twenty-four hours. Sometimes the submaxillary and sublingual glands are also involved. The glands may swell so as to completely prevent the patient opening his mouth more than a quarter of an inch. They are acutely tender, and disfigure the patient very much, but the malady is essentially a trivial one. The *Diagnosis* of parotitis is very simple, the swelling of the glands being unlike anything else. The only difficulty is between mumps and simple parotitis, such as occurs in abdominal diseases, after laparotomy, or in connection with oral sepsis ; but mumps is always bilateral, and never suppurates. *Etiology.*—It is almost entirely confined to children and young persons between the ages of five and twenty. It is rare in the very young and very old, but is often epidemic and runs

through a school. A patient remains infectious as long as there is any definite swelling of the glands. *Prognosis.*—Death from the disease is unknown, and the patient is generally quite well in ten or twelve days at the outside. The chief danger is the swelling of the tonsils and submaxillary glands. In delicate subjects the swelling is slow to disappear. The *complications* consist of (1) enlargement of the tonsils, and (2) orchitis and ovaritis. In these circumstances a very curious phenomenon occurs, for as the testis swells the parotitis subsides. It is the best instance of the phenomenon called "metastasis." The mammary glands may also become swollen and tender. In some epidemics the swelling of the mamma or testicle precedes or accompanies that of the parotid, and epidemics have been known in which the former were involved without any parotitis. Occasionally the affected glands may become permanently atrophied. *Treatment.*—The patient should be kept in one room. Warm anodyne fomentations may be applied, and if tension is present, leeches give relief. Diaphoretics and purgatives are useful, and nutrient enemata may be required.

The remaining fevers in this group are Plague, Yellow Fever, Malta Fever, *which are met with abroad;* Relapsing Fever, *met with only in times of famine; and* Epidemic Cerebro-spinal Meningitis, *which is very rare in this country. In* Hay Fever, Dysentery, *and* Cholera, *there is some disturbance of the temperature.*

§ **370. Plague** (Pestis Major, Bubonic Plague, Typhus Bubonicus, Oriental Plague, probably the same as the Black Death) may be defined as a highly contagious and fatal fever, characterised by inflammatory, glandular, and periglandular swellings, hæmorrhages beneath the skin and from the mucous membranes, and the typhoid state. The last great epidemic in London was in 1666. Its endemic centres in the present day are Northern India, Yunna (China), Mongolia, and possibly Uganda. Since 1894 many small epidemics of plague have arisen in several places.

Symptoms.—(1) The incubation period is from two to eight or even fifteen days (Manson). (2) There is often a prodromal stage, with depression and pains, but usually the onset is sudden, with shivering, and fever rising to 103° or even 107° F. Mental aberration is not uncommon. The prostration is very marked, and may be accompanied by vertigo, staggering gait, and lethargy, soon passing into the typhoid state. The spleen and liver are usually enlarged. In some cases the speech is halting and staccato, the expression vacant, and the eyes congested. (3) Buboes (inflamed glands) appear in one to five days, usually within twenty-four hours. They may be single, or a group may be affected in one place, usually the groin; sometimes they appear in several parts of the body at once. They may be painless or very painful, and they may suppurate about the seventh day. (4) Petechiæ and subcutaneous hæmorrhages are not uncommon. A distinctive rash is rare, but when present it resembles typhus. There are six principal *varieties*, which prevail in different epidemics: (i.) The *bubonic* variety is the commonest, glandular swellings occurring in quite 70 per cent. of all the cases[1]; (ii.) the *septicæmic* type is very fatal: the glands enlarge slightly, but they do not suppurate; (iii.) an *abortive* form, in which there are buboes without much fever, subsiding in fourteen days; (iv.) a *fulminant* form, with high fever, little glandular enlargement, vomiting of blood, and death within a few hours; (v.) a *pneumonic* form, which may be mistaken for bronchitis or pneumonia, attended by intense prostration, no glandular enlargement, and death usually on the fifth day, the pulse-respiration ratio being not so much altered as in true pneumonia; and (vi.) an *ambulant* or mild form, with chronic glandular enlargement, great anæmia, and weakness. The *Diagnosis* is not difficult if sudden onset, marked prostration, mental state, and bubonic swellings be present. Plague closely resembles typhus, both in its symptoms and etiology, but a rash is rare in the former; and the microbe of plague

[1] Mr. James Cantlie's description of the malady issued by the London County Council (October 1900), to which the author is also indebted for other items of information.

is distinctive.[1] Enteric fever may have to be diagnosed from plague, though its onset is so much more gradual.[2]

Etiology.—Plague is due to the bacillus pestis, discovered first by Kitasato, and later by Yersin. It can be readily demonstrated in the serum drawn from the periglandular tissues by a hypodermic syringe. It was observed that outbreaks of plague were often preceded by a large mortality among rats and other vermin, and it is now known that the disease is mainly due to rats. It has been proved that it is the fleas infesting the rats which convey the infection to man. Filth and overcrowding predispose to plague. Age and sex have little influence. The disease is highly contagious, but " its extension depends more on place infection than on direct transmission from person to person " (Manson).

Prognosis.—The case-mortality in the early periods of epidemics is generally 100 per cent., later on 40 per cent. Amongst the white population in India it was only 35 per cent. (Cantlie). In the usual course, death occurs before the sixth day ; or, if the patient is to recover, convalescence starts between the sixth and tenth day. Prolonged suppuration of the glands may delay convalescence considerably. The course of the disease is very difficult to forecast. Hæmorrhages usually herald death. The *sequelæ* include boils, pneumonia, dropsy, partial paralysis, and mental disorder.

Treatment.—The hygienic and therapeutic treatment are as in typhus (see also §§ 386 *et seq.*). The injection of carbolic acid into the glands has been practised with some success, and large doses by the mouth are also recommended. Immunisation is now obtained by inoculation of serum (Yersin) if commenced in the early course of the disease.

§ **371. Malta Fever** (Synonyms : Mediterranean Fever, Gibraltar Fever, Febris Undulans) arises only in the countries around the Mediterranean Sea, chiefly in the summer months, but it recurs in England in patients who have left those districts. It is predisposed to by bad drainage and unhygienic conditions. It is now known to be due to the micrococcus melitensis, which is apparently conveyed by the milk of the goats which abound in Malta. It begins insidiously, with langour and increasing debility. The temperature stands about 102° to 104° F. for a week or a fortnight ; then it falls. In mild cases this may be all, but more often, after an interval of a week or so, the fever returns ; then it falls again. The pyrexia runs a most indefinite course, neither continuous nor intermittent, though it may be either. In two cases I have recently seen the temperature resembled pyrexia more than anything else. The most characteristic feature of this disease, however, consists in the *attacks of pyrexia with marked prostration, separated by intervals of comparative health.* These attacks may be repeated over several months. The spleen becomes enlarged, and there is usually a certain amount of gastro-intestinal disturbance. Among the symptoms complained of are rheumatic pains and sometimes swelling of the joints and limbs. The weakness may amount almost to paralysis. Thrombosis is fairly common.

Diagnosis.—This fever is very apt to be mistaken for *enteric fever*, as the gradual onset is common to both. But in typical cases of enteric there is a more prolonged and characteristic course, and the agglutination reaction (*q.v.*), which is obtainable in both diseases, affords a certain means of distinction (Sir A. E. Wright). Some cases may be mistaken for *malaria*, but the malarial parasite is not found in the blood.

The *Prognosis* as regards life is good. The case-mortality of Malta fever is only 2 to 3 per cent. But when a patient is attacked with Malta fever at the beginning of the summer months, and cannot be removed from the Mediterranean at once,

[1] The Code of regulations for searchers of the plague, issued by the Royal College of Physicians in 1665, mentioned a rash as a means of recognising the disease, but probably typhus was confused with plague. Murchison held they were probably identical, but the chief fact which disproved this was their seasonal incidence. Plague was a summer disease, most prevalent in July and August. Typhus was most prevalent in the winter.

[2] See also the *Lancet*, October 27, 1900, p. 1197.

he may have a long series of relapses, with consequent anæmia of great intensity. So long as the tongue remains coated a relapse may be feared. The usual complications are excessive sweating, orchitis, diarrhœa, and cardiac failure. The most severe complications, however, are pneumonia or "abscess of the lung," and hyperpyrexia, the latter being the usual cause of death.

Treatment.—It is most important, if the disease occurs in the beginning of the summer, to immediately remove the patient to a cooler climate. Drugs should be avoided. Antipyretics are dangerous. If the fever is high, cold sponging is preferable (see § 392). Sir A. E. Wright has prepared a vaccine.

§ **372. Yellow Fever** is an acute specific fever peculiar to hot climates and seaport towns, accompanied by jaundice, black-vomit, and the typhoid state.

Symptoms.—(1) The incubation period is short, probably from four to five days. One attack usually renders the patient immune for life. Yellow fever has a sudden onset, the temperature rising on the first day to 101°, 105° F., or even higher, and it remains high for three or four days. The pulse does not rise in proportion. The temperature then falls to normal, or at least remits greatly; and though it may rise again, the second fever is not so high. (2) Albuminuria is a constant sign, and occurs usually in eighteen to twenty-four hours after onset. The urine often has a green colour. (3) In most cases jaundice and vomiting appear about the second day, but in mild cases these may be absent, as may also the yellowness which has given the fever its name. In severe cases the jaundice is intense, with petechiæ, the vomit is mixed with bile, and in the later stages with blood, forming the "black vomit." Hæmorrhages may also occur from the gums, stomach, nose, and bowels. There is no splenic enlargement. The liver has a degree of fatty degeneration, and the bloodvessels supplying the stomach and intestines are in a state of degeneration, and readily rupture.

Diagnosis.—Yellow fever has to be diagnosed from the pernicious forms of *malaria*. A very important point is the occurrence of albuminuria in yellow fever. In malaria the spleen is enlarged, and the parasite is found in the blood. In *Blackwater fever* there is a green vomit, which may cause it to be diagnosed as yellow fever, but it is accompanied by hæmoglobinuria, and no blood-corpuscles are found in the urine; whereas in yellow fever, if the urine is red, it will be found that the condition is due to the presence of blood-corpuscles. *Acute Yellow Atrophy* of the liver has a more gradual onset, and is practically confined to the female sex.

Etiology.—Yellow fever is peculiar to the West Indies, certain parts of America, the Brazilian ports, and the west coast of Africa. It is found only in seaport towns. It rapidly spreads, especially in those parts which are crowded and dirty. It is always worse in the summer months, as a high temperature is necessary for the existence of the poison. A slight frost will destroy it, as at Memphis in 1879. It has been proved that it is transmitted, like malaria, by a mosquito (*Stegomyia fasciata*), but the specific germ has not yet been demonstrated. It is usually stated that the negroes do not contract the disease, but I am informed on good authority that this is not correct.[1] The disease does not spread so rapidly amongst them as amongst Europeans, probably because many of them have been rendered immune by a previous attack.

Prognosis.—The case-mortality varies in different epidemics from 5 to 75 per cent. Sometimes the patient recovers uninterruptedly after the fever falls on the fourth day; in such cases the skin is moist, there is little albumen or vomiting, and little or no yellowness. On the other hand, death may occur with "typhoid state" a few hours after the onset of disease, or from collapse after the fever remits. The prognosis is always grave when the jaundice is intense, the vomiting frequent, and hæmorrhages occur from the stomach or elsewhere.

Treatment.—As the liver is found after death to be in a state of fatty degeneration, one of the first indications is to diminish the work of the portal system by attention to the dieting of the patient. Only weak milk and water, or mutton broth may be given. No solid food must be taken for four days. Other treatment is

[1] Dr. F. B. Archer, Quarantine Health Officer, Barbadoes.

symptomatic—*e.g.*, ice and astringents for the hæmorrhage. It is important to induce free elimination by the skin and kidneys. The rest of the treatment is conducted on the same principles as typhus fever.

§ **373. Epidemic Cerebro-Spinal Meningitis**[1] is characterised by (1) fever, sometimes very irregular at the onset, becoming normal for a day or two, then rising again. It may be remittent, but not often. It is rarely over 102° to 104° F., but may be considerably raised towards the end. The pulse frequency is not always proportional to the degree of fever. (2) Symptoms of irritative intracranial inflammation, such as very severe headache, of sudden onset, with vomiting and muscular spasm. Compression symptoms may supervene later. The so-called " Kernig's sign "—*i.e.*, when the thigh is flexed on the abdomen, the leg cannot be extended because of spasm of the flexors of the thigh—is usually present. (3) There is always retraction of the head, and sometimes opisthotonos may be present, owing to the rigidity of the muscles of the back. Hyperæsthesia, especially along the spine, and severe pain in the back, may be so great that all movement is intolerable. (4) A prominent feature is the presence of some skin affection, very often occurring symmetrically. Herpes labialis or zoster may be present. On the second day a rash of purpuric spots sometimes appears on the neck and extensor aspects of the limbs. Its frequency varies considerably in different epidemics, for in some it has been a rare symptom. Urticaria and erythema may occur.

Diagnosis.—This disease has to be diagnosed from *tuberculous meningitis*, which has an insidious onset, and no eruption. From other forms of *meningitis* the best method of diagnosis in doubtful cases is by lumbar puncture, when the fluid will be found to be turbid, and to contain the specific diplococcus. When an epidemic is present, there is little difficulty in the diagnosis.

Etiology.—The disease attacks persons under twenty usually, and some epidemics have occurred chiefly among young infants, and males more than females. It never occurs in summer, and is most frequent in winter and spring. It does not appear to be contagious, although it usually occurs in epidemic form. It is due to a specific micro-organism, described by Weichselbaum, which is intracellular, non Gram-staining, and occurs in pairs.

Prognosis.—The disease has a case-mortality of 30 to 70 per cent. The usual course of the malady is three weeks ; but there are three varieties based upon the duration besides the common form above described : (i.) The foudroyant form, which kills the patient in a few hours or days ; (ii.) the typhoid form, which lasts for several weeks ; and (iii.) the form which recovers in a few days. The prospect of recovery is not good when the disease attacks infants or old people. Amongst the unfavourable signs are the occurrence of hyperpyrexia, convulsions, irregular breathing, or an unduly prolonged period of illness. The more common complications are inflammation of the joints, optic neuritis, and polyuria. A trace of sugar may appear in the urine. Amongst the sequelæ may be mentioned deafness, impairment of the vision, chronic hydrocephalus, and transient paralysis of the limbs, or aphasia.

Treatment.—Hitherto the treatment has been mainly symptomatic, for the relief of the pain and the fever. Repeated lumbar punctures have been recommended as a curative measure, but apparently do little more than temporarily relieve the pressure symptoms. Recently sera and vaccines have been employed, and give greater promise of success.

§ **374. Relapsing** or **Famine Fever** [Synonyms : Recurrent or Relapsing Typhus, Spirillum Fever (Vandyke Carter)] is a contagious fever met with in times of famine, ending abruptly on the fifth, sixth, or seventh day, and followed after an interval of one week without fever by a relapse similar to, but shorter than, the first attack. The incubation period varies from five to nine or more days.

Symptoms.—(1) The fever has a sudden onset, and rises rapidly. It frequently reaches 108° F., a range which in other diseases is not consistent with life. After

[1] The author is mainly indebted for this account to " The Etiology and Diagnosis of Cerebro-Spinal Fever " (The Cavendish Lecture, 1899), by Professor W. Osler.

remaining elevated for six or seven days, the temperature returns to normal as rapidly as it rose. The fall is preceded and attended by profuse perspiration or diarrhœa, or both. This is followed by an interval of about a week, during which the patient feels exhausted, and the pulse and temperature are subnormal. At the end of this week a relapse occurs which is similar to the first attack, but shorter, lasting three or four days. In rare cases there is a second and even a third relapse. (2) Abdominal pain and tenderness, and great enlargement of the spleen and liver, are present in almost all cases. Jaundice is also very common. Epistaxis is common, and sometimes there is vomiting of blood. Delirium is very rare, but if present is of the noisy kind, and occurs at the crisis. Convalescence is slow. (3) The Spirillum is found in the blood during the pyrexial period, but in the intervals it is only present in the spleen.

Diagnosis.—The diagnosis is not difficult, on account of the circumstances under which the disease occurs, and the course of the temperature. *Enteric fever* and *smallpox* cause rash ; rheumatic fever is associated with joint lesions. Yellow fever, which it most resembles, produces jaundice, and a diagnosis is only made by the course of the fever and the presence of the Spirillum in the blood in relapsing fever.

Etiology.—Relapsing fever is due to a specific spirillum, the spirochæte of Obermeier. The disease arises under the conditions which attend a famine, and has been noticed to accompany most epidemics of typhus, in which circumstances the epidemic begins with relapsing fever and ends with typhus. The poison is given off by exhalations and the skin ; the infecting distance is extremely short. The disease appears in seasons of unusual distress, as during strikes. It does not occur with ordinary destitution, but in times of famine, when people eat unwholesome articles, such as grass, roots, hay, etc. Overcrowding is not absolutely necessary for its production, in which respect it differs from typhus. One attack does not confer immunity from a second. As regards the *Predisposing Causes*, age has no influence, nor have seasons or occupation.

Prognosis.—The case-mortality is not nearly so great as that of typhus ; it rarely exceeds 2·5 per cent. Age has not much influence, but dissipation and debility are unfavourable. Death, which occurs generally at the height of the first attack, is usually due to syncope, from hæmorrhage or from granular degeneration of the heart. When occurring later, it may be due to complications. Untoward symptoms are : More than one relapse, hæmorrhage, suppression of urine, the typhoid state, cerebral symptoms, or indications of a weak heart. A rapid pulse, a high temperature, and even jaundice, are not unfavourable.

Remedial *Treatment* consists of the administration of salines and diuretics. At the commencement of an attack considerable relief may be given by an emetic or mild purgative. Digitalis may be required for the heart, and paraldehyde for the sleeplessness.

§ **375. Thermic Fever** or **Heat Stroke** (Synonyms : Siriasis, Heat Apoplexy, Heat Asphyxia, Sunstroke, Coup de Soleil) is one of the numerous varieties of tropical fevers about the pathology of which we know but little.

Symptoms.—The onset is usually sudden, during or after exposure to a hot sun. In some cases there are a few days prodromata, consisting of headache and malaise. Then a short stage of delirium rapidly sets in, and is immediately followed by coma and high fever (108° to 109° F.). During the stage of delirium the patient is restless, with muscular twitching and spasms. The stage of coma is marked by a very hot skin, rapid pulse, flushed face, heavy or stertorous breathing, and contracted pupils. In most cases death occurs a few minutes or hours after the onset of insensibility.

Diagnosis.—The coma of *uræmia*, *diabetes*, and *drugs* (morphia, alcohol, etc.), is known by the absence of high fever. In the coma of *cerebral hæmorrhage* into the pons fever may occasionally be present, but it would not precede the onset of coma. The comatose form of *malaria* is recognised by finding the parasite in the blood, and an enlarged spleen.

Etiology.—All ages and sexes may suffer. It is predisposed to by intemperance, fatigue, malaria, overcrowding, and weakness of any kind. The disease appears

to be endemic in certain places, and at times an epidemic occurs. Sambon[1] pronounces it to be due to a germ which requires for its action a high temperature. This would explain its frequent occurrence amongst those who have to perform long marches in the sun of tropical or sub-tropical climates.

Prognosis.—The case-mortality is about one in four. Most patients die from failure of respiration after the onset of coma. Favourable cases terminate by crisis, and make a rapid convalescence. Much depends on prompt treatment.

Treatment.—The indication is to reduce the temperature at once, if possible without the use of drugs. Lest ague be also present, it is best in malarial countries to give a hypodermic of quinine (7 grains) at once, and to repeat it every four hours. In order to reduce the temperature the patient must be laid on a stretcher, with a sheet covered with ice placed over him. Iced water should be run over him till the thermometer in the rectum falls to 102° F., or, if much hyperpyrexia be present, to 104° F. Then he should be wrapped in blankets, and stimulants given. Avoid strychnine because of the tendency to convulsions.

§ 376. **Hay Fever** (Hay Asthma), especially the constitutional variety, **Dysentery,** and **Cholera,** give rise to a certain amount of pyrexia of a continued type.

Hay Fever (§ 130) is recognised by the violent attacks of sneezing.

Dysentery (§ 219).—Acute dysentery is sometimes attended at the onset by some degree of pyrexia, but much the most important symptom of this disease is diarrhœa.

In Cholera (§ 220) the abdominal cramps, collapse, and diarrhœa are the leading symptoms. During the collapse stage the temperature may be as high as 105° F. in the rectum, although in the axilla and mouth it is subnormal. In the reaction stage, if the patient lives, there is usually a degree or so of pyrexia lasting from a week to a fortnight.

Finally, there are several diseases which in their typical forms belong to Group III., or, belonging to Group I. are seen perhaps before or after the eruption comes out, which may present pyrexia of a continued type. It is well in all cases of difficulty or doubt to remember this, and to pass in review the members of all three groups.

GROUP III. INTERMITTING PYREXIA.

§ **377.** In this group of diseases the pyrexia is of an INTERMITTENT (or remittent) type—*i.e.*, the temperature drops at regular or irregular intervals to normal (or nearly to normal). This group is distinguished from Group I. by the complete absence of eruption. It is distinguished from Group II. mainly by the wide variations of the temperature.

Common.		*Rare.*	
I. Malaria	§ 378	VII. Malignant endocarditis	§ 39*a*
II. Latent tuberculosis ..	§ 381	VIII. Lymphadenoma ..	§ 408
III. Visceral syphilis ..	§ 382	IX. Pernicious anæmia ..	§ 403
IV. Acute septicæmia ..	§ 383	X. Acute lymphatic leukæmia	§ 407
V. Subacute septic conditions	§ 384	XI. Opium habit	§ 385
VI. Enteric fever (some cases) and occasionally influenza ..	§ 364		

The clinical investigation of these diseases is often attended by considerable difficulty. Ague, which may be regarded as the type of this group, is essentially a *paroxysmal pyrexia*, each paroxysm having three stages (cold, hot, and sweating), and each paroxysm being usually *separated by one or more days' interval of health.*

[1] Discussion at the Meeting of the British Medical Association, *Lancet*, 1899, vol. ii., p. 609.

Tuberculosis and Syphilis have a daily rise and fall, and are good examples of *regular diurnally* intermitting pyrexia. Acute Septicæmia, on the other hand, is noted for the *irregular* character and wide range of its temperature and the severity of the rigors. Chronic Septic Conditions occupy a position midway between these two types—regular and irregular intermitting pyrexia. In a given case of intermitting pyrexia which has arisen in a tropical and subtropical climate ague is probable, but in England the commonest cause is probably latent tubercle. The Serum Reactions aid us to some extent in the diagnosis of this group (*vide* Chapter XVI., p. 590).

Turning to the rarer diseases, which must always be kept in mind, Malignant Endocarditis is chiefly remarkable for the *long course* it may run. In Lymphadenoma we find the enlarged *glands ;* and in Pernicious Anæmia the patient is a male, and the skin is very sallow, and the blood is characteristic.

It follows therefore that if we have a patient's temperature chart before us, and it shows definite intermissions or remissions, the disease will belong to one of three sub-groups :

A. Regular Intermittent Pyrexia, with one or two days' interval, which contains only one disease—Malaria . . . . . § 378

B. Regular Intermittent Pyrexia occurring daily, such as Tuberculosis, and Visceral Syphilis . . . . . §§ 381 *et seq.*

C. Irregular Intermittent Pyrexia, such as Septicæmia, and other pyogenic processes . . . . . §§ 383 *et seq.*

§ **378. Ague** (Synonyms : Malarial Fever, Intermittent Fever, Remittent Fever, Jungle Fever).—Ague is a non-contagious fever, occurring in paroxysms with complete intermissions, due to the malarial parasite introduced by the mosquito.

Symptoms.—As a rule the fever comes on suddenly without warning. The period of incubation varies considerably. Sometimes there may be none at all ; sometimes it is not more than a few hours ; while in some cases there may be an interval of months or years after exposure before the disease develops. Sir Patrick Manson gives two to two and a half years as the limit for tertian ague, and two to three years for quartan. A paroxysm has three characteristic stages. First there is a *cold stage*, in which the patient shivers or has a rigor, and feels cold, though the temperature is elevated three or more degrees ; the skin looks cold, sometimes livid, and the nails are blue. It lasts from one-half to two hours, or so, and is followed by the *hot stage*, in which the temperature goes up 103° to 106° F. It begins with flushing of the face, and is attended by headache, pains in the back and elsewhere. It lasts three or four hours, and is followed by the *sweating stage*, in which the perspiration is so profuse that the bed-linen may be soaked. This stage lasts one or two hours, and is accompanied by a fall of temperature. The spleen enlarges during the attack. The stages may be shorter or longer. The sweating stage is followed by an interval, during which the temperature is normal, or subnormal, and the patient is fairly well, except for great lassitude and indigestion. In the commonest type of the disease (Tertian Ague, Fig. 101), there is an interval

of about twenty-four hours between the paroxysms, which, if untreated, may recur for weeks.

Varieties of Ague.—Malarial fever may vary in two ways : (*a*) According to the duration of the interval between the attacks, or (*b*) according to the intensity ; both of which probably depend on the species of the plasmodium. (*a*) There are three types of periodicity (Fig. 101) : (i.) *Quotidian* ague, in which the paroxysm occurs daily, is rare—probably a compound of (ii.) and (iii.); (ii.) *Tertian* ague, in which attacks occur every other day ; and (iii.) *Quartan* ague, in which the attack occurs every third day. Various compounds of these occur. (*b*) The chief of the more severe forms are (i.) the *comatose*, in which the patient

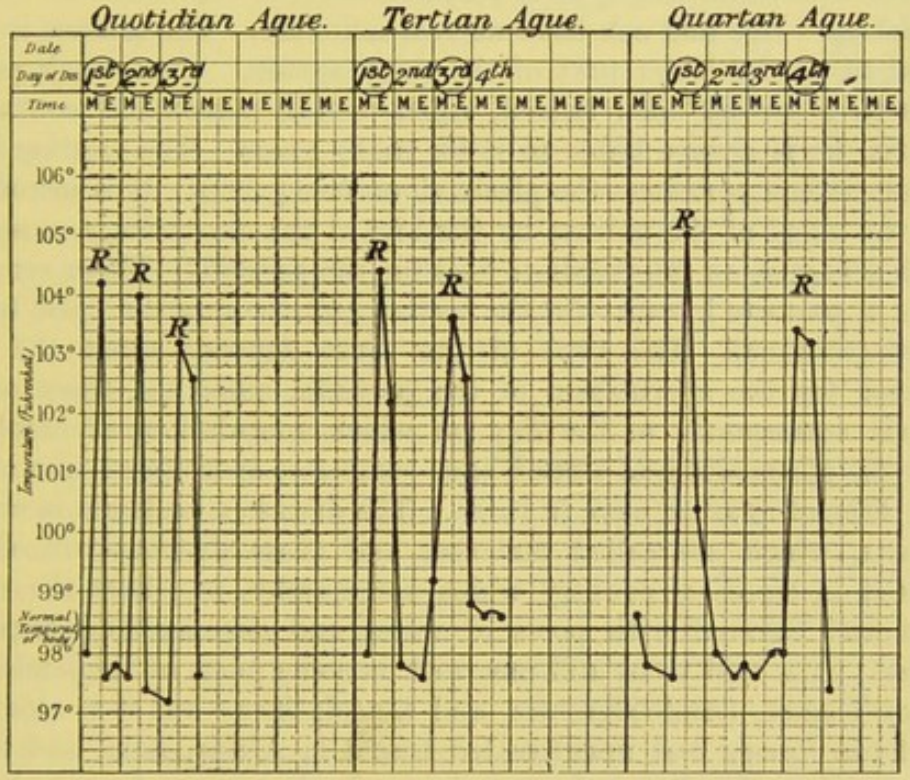


Fig. 101.—Types of Ague.—Quotidian (daily) ; Tertian (every other day) ; and Quartan (every third day). " R " indicates the rigor which ushers in the cold stage.

suddenly passes into coma ; (ii.) the *hyperpyrexial*, in which the temperature rises suddenly to 107° or 112° F., and death occurs in a few hours ; (iii.) the *algid*, which resembles the algid stage of cholera. Other varieties depending upon the intensity are referred to below—namely, Remittent or Jungle Fever, Algid, Comatose and Hæmorrhagic Malaria, and Blackwater Fever. *Æstivo-autumnal* fever is a variety of malarial fever attacking Europeans on the Gold Coast and elsewhere.

Diagnosis.—Malaria is rarely mistaken for other diseases ; but the other disorders attended by intermitting pyrexia about to be described are very frequently mistaken for ague. *Clinically*, this mistake would be avoided if it were remembered *that ague of true quotidian periodicity*—

daily recurrence—*is very rare*[1]*:* and that tertian or quartan periodicity is absolutely pathognomonic ; it occurs in no other disease. In leprosy and all the diseases mentioned below the intermission is daily. *Therapeutically*, the diagnosis may be established by full doses of quinine ; if this be hypodermically given and fail to relieve, the attacks are certainly not malarial. The *microscopic* diagnosis of the parasite in the blood requires considerable experience, but it is always possible to find it in blood-films, provided the patient has not taken quinine for several days ; this is an essential part of the examination, and is, of course, positive evidence.[2] Enteric fever and many other conditions belonging to Group II., when occurring in a malarial subject, are apt to assume malarial or intermitting type of pyrexia.

Etiology.—Age and sex have no real influence. The disease is most prevalent at the latter part of the rainy season. One attack predisposes to a second one ; indeed, when once a person has contracted malaria, he is always liable to it for many years. The *exciting* cause of malaria is a parasite—the plasmodium (see § 399). It is introduced into the blood of the patient by the bite of the *anopheles*, a mosquito, which serves as an intermediate host for the parasite. There are three forms of parasite known—(*a*) the tertian ; (*b*) the quartan ; and (*c*) the æstivo-autumnal. The type of the fever depends upon the time required for the sporulation of the parasite, since the onset of the pyrexia corresponds to the day on which sporulation is completed. Thus the plasmodium of tertian ague completes spore formation in two days, that of quartan ague in three. The disease is endemic in certain districts, which are called malarial, and these districts are always situated in tracts of country which are marshy, or where the soil is moist and covered with pools of water and decomposing vegetable matter. It is said to be still met with in some parts of England—*e.g.*, Isle of Thanet, Cambridgeshire, and Lincolnshire—but it is chiefly in the dense forests and uncultivated tracts in Africa, Asia, and South America that the disease is prevalent. It disappears from a district when the soil is drained and cultivated. Standing water, especially in puddles, seems to be a necessary condition, together with a moderately high temperature. Malaria was always observed to keep close to the ground, and its spread was known to be interrupted by a tract of water, especially salt water, or by a grove of trees. It was known that change of wind would bring malaria from a distance. Persons newly arrived

[1] The only fallacy to this statement occurs in those cases when both the tertian and the quartan parasites affect the patient at the same time, and thus give a partial semblance to quotidian attacks. But even then the pyrexial attacks are not really quotidian, as may be seen from the following diagram. Supposing "a" represent the attacks of a tertian parasite, and "b" those of a quartan parasite, there would be one day's interval after at longest three days' pyrexia, viz. :

a	b	a		b a		a	b	a		b a		

[2] Sir Patrick Manson, the *Lancet*, May 17, 1902.

in a district were especially prone to contract the disease. These and other curious data have been known for many years, but remained without explanation until it was proved by Manson, Ross, Nuttall, and others, that the disease is conveyed, and is introduced into the blood of man, by the mosquito (Fig. 102).

Prognosis. — Death usually occurs from complications, without which malaria is not a very fatal disease. The most favourable type of case is that in which the pyrexia runs a typically intermittent course. The gravest, and happily the rarest, is that form in which the pyrexia is continued or only remittent. Coma or delirium, hæmorrhage from the stomach or bowels, and choleraic diarrhœa with cramps are unfavourable signs, and if collapse sets in after the hot stage a fatal termination is usual. The chief *complications* are (i.) great weakness and anæmia, which are common results of the disease, and in time, especially if untreated, the patient develops the typical cachexia of malaria. Pigmentation of the skin is a marked characteristic of this cachexia ; the distribution of pigment is general, but is especially evident around the eyes.[1] This is doubtless accounted for by the deposit of blood-pigment granules which are so constantly found in the blood. (ii.) Enlarged spleen—" ague-cake " (§ 262, VI.)—is a usual sequence, and rupture of the organ occasionally takes place. (iii.) Jaundice, due usually to hepatitis, is one of the more serious complications, and the liver after many attacks becomes enlarged.

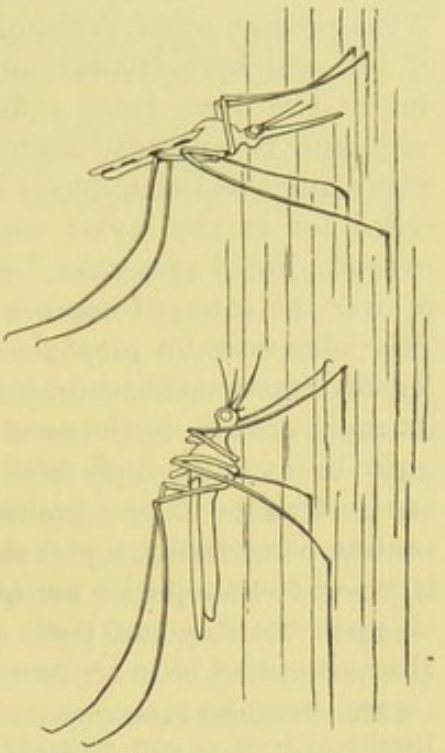


Fig. 102.—MOSQUITOES settling on a wall.—There are two chief types of mosquitoes—Culex and Anopheles—easily differentiated by their attitudes when resting upon a wall. Anopheles is the more dangerous one, and is recognised by its spotted wings and its tilted attitude. Its larvæ lie flat on the surface of puddles, and move along the surface ; whereas Culex larvæ lie more perpendicularly, and if disturbed rush to the bottom of the pool. Anopheles larvæ are found in puddles which contain algæ and which are too large to be dried up in a week (time needed for the mature insect to be hatched). They are not found in pools which contain minnows, nor in rapid streams, nor in shallow rain pools that are easily dried up. Kerosene oil (about ½ i. to a pool of 1 square yard) killed all larvæ in six hours. " Malaria Expedition to Sierra Leone," *Brit. Med. Journ.*, September 30, 1899, p. 869. See also meeting of Path. Soc. at Cambridge, Summer, 1900 ; *Lancet*, vol. ii., 1900 (Nuttall) ; and Ross and Mallerd, *Brit. Med. Journ.*, November 3, 1900.

Treatment.—Quinine is a specific in the treatment of ague. The hydrochloride or the sulphate, dissolved in tartaric acid and water, is

[1] On one occasion when I joined a passenger ship from the East this periorbital pigmentation was so marked in one of the passengers that I thought he must have been fighting, until I learned that he had been a victim of malaria.

preferred to hard and sometimes insoluble pills and tabloids. A large dose (at least 10 grains) should be given, and repeated in diminishing doses four times daily. Some advise that it should not be given in the pyrexial stage. An aperient should have been previously administered. When prompt action is required (as in the graver forms), 7 to 10 grains of the acid hydrochloride of quinine should be given hypodermically, one or two doses being sufficient. For the resulting anæmia arsenic with iron is especially useful, and Manson recommends that quinine with salines be taken once a day for two months. Stimulants are also called for in the severe varieties—ether, strychnine, or brandy *per rectum*. For "ague-cake," red iodide of mercury ointment rubbed in over the enlarged organ is said by foreign observers to be of value. The indications for *prophylactic* treatment are based upon the etiology. Marshy tracts must be drained, and of all trees the red gum-tree, from its rapid growth, is the most suitable for this purpose. The night air must be avoided, high sites selected for sleeping, and mosquito-nets used. Quinine (2 to 5 grains three times a day), given as a matter of routine, is certainly a preventative. It is now recommended that the larvæ of the anopheles mosquito should be killed by pouring kerosene oil upon the stagnant pools and puddles in the district (see Fig. 102). Mosquito-proof huts are now used.

§ **379. Remittent Fever** (Synonyms : Jungle Fever, Continuous Malarial Fever).—Remittent fever is now generally admitted to be a variety of ague, due to the æstivo-autumnal parasite, which is the cause of the more irregular forms. There is a protracted hot stage, and no apyrexial intermissions. The cold stage is either absent altogether or is only marked by a chilliness. The pyrexia continues, with slight daily remissions, for a week, two weeks, or more. In some forms there is a tendency to the typhoid state ("typhoid remittent fever"). In other forms gastric symptoms are prominent, together with marked jaundice ("bilious remittent fever"). Sometimes there is great prostration, with hæmorrhages, and this form is followed by extreme anæmia. The *Diagnosis* from yellow fever on the one hand, and enteric on the other, may be impossible without an examination of the blood. In yellow fever albuminuria is present, and the temperature falls in three or four days. Enteric gives the Widal reaction. The *Prognosis* is always unfavourable. It is especially bad when the remissions become less marked, and the typhoid state supervenes. Unfavourable symptoms are collapse, delirium, and coma.

§ **380. "Blackwater" Fever** (Synonym : Hæmoglobinuric Fever), so named from the colour of the urine, is a form of malaria possibly due to a special plasmodium.

Symptoms.—In a typical attack the onset is marked by rigors, and the temperature ranges from 103° to 105° F. The urine which is passed is of a dark colour, due to the presence of hæmoglobin ; it is scanty in amount and of high specific gravity. There is bilious vomiting, which may be extremely severe, and accompanied by intense jaundice. As the fever falls, the urine clears ; then a new paroxysm of fever may set in, with a return of the hæmoglobinuria. The liver and spleen may be enlarged. During the paroxysm there is great destruction of red blood-corpuscles, and the blood shows poikilocytosis. *Etiology.*—This fever is endemic in the tropical and subtropical regions of America and Africa, especially West Africa. What determines the onset of the paroxysm is not known. Some have thought that the hæmoglobinuria is related to the administration of quinine.

Diagnosis.—This disease may be mistaken for yellow fever, but in the latter, one attack renders immunity, whereas in hæmoglobinuric fever there is usually a history of many attacks. In yellow fever the spleen is not enlarged, and the

fever does not remit several times in succession. Malaria parasites may be found in the blood in about 60 per cent. of the cases.

Prognosis.—The case-mortality is 25 per cent. Frequent relapses are certain to occur if the patient remains in the endemic district. Even if he return home, he is liable to have attacks of hæmoglobinuria, though these may be accompanied by little or no fever. In severe cases there may be profound prostration, with all the symptoms which accompany a profuse hæmorrhage. Death may result in this way, or from syncope, or collapse. There may be suppression of urine, and death with symptoms of uræmia.

Treatment.—Manson advises the avoidance of quinine, unless the presence of the malarial parasite in the blood indicate its necessity. Otherwise the dehæmoglobinating effect of quinine will only do harm. The patient must be kept at rest, and water may be freely given. Iron is indicated.

§ **381. Latent Tuberculosis.**—Tuberculosis is said to be latent when the usual physical signs or local manifestations are wanting. In all cases of unexplained intermitting pyrexia in this country, one of the first things to be suspected is tuberculosis in some part of the body. It may be very deeply seated, but it is a useful clinical axiom to remember that *no active tuberculosis can exist in any part of the body without the occurrence of a daily intermitting pyrexia.* Moreover, the degree of the fever is a fair indication of the activity of the process. The chart is a typical one; the temperature drops each morning to (about) normal, and rises each evening one, two, or more degrees, occasionally *vice versa*. The physical signs may be altogether wanting, and the patient, perhaps, only seeks advice on account of the weakness, dyspepsia, and other vague symptoms. Such a condition may go on for weeks without any local manifestations, as in the cases referred to under Tuberculous Meningitis. The lungs, kidneys, peritoneum, and various organs may be affected. (1) The commonest locality in adult life is the *lungs*. In this case physical signs usually appear which resemble bronchitis or simple pulmonary congestion, for which diseases it is apt to be mistaken (§ 83). (2) The *meninges*, *peritoneum*, and other *serous membranes*, are perhaps the commonest positions in childhood in which tubercle may be deposited without definite signs. (3) In the *kidney*, tuberculous pyelitis may be readily overlooked, and in suspicious cases the urine should be carefully examined for traces of pus and tubercle bacilli (§ 305). (4) Tubercle may also be latent in other situations, such as the cranium, spine, intestines, and other viscera; and, finally, the tuberculous process may be generalised, and give rise to the condition known as *Acute General Tuberculosis*. In the diagnosis of tuberculosis we now have the help of certain blood reactions—(*a*) opsonic index; (*b*) Calmette's ophthalmic reaction; (*c*) Von Pirquet's cutaneous reaction (§ 405).

§ **381*a*. Acute General Tuberculosis** (Synonyms: Acute Miliary Tuberculosis, Typhoid Tuberculosis) may be of the meningeal type, usually known as tuberculous meningitis, and described under that title; of the pulmonary type (*vide* § 83); or of the typhoid type, with which we are now concerned. It is characterised by intermitting pyrexia, prostration, and a tendency to the typhoid state—due to a generalised infection of the body by the tubercle bacilli.

Symptoms.—(1) The onset is insidious. The patient complains perhaps of nothing but lassitude, which is attended by feverishness of a typical intermitting type, and perhaps bronchial catarrh. The temperature each morning may be normal, that in the evening raised one or more degrees. The inverse type—*i.e.*, a lower temperature in the evening than the morning—is said by some to be more frequent in this than in other forms of tuberculosis.[1] In very rare cases the highest daily temperature does not rise above normal. The patient complains of lassitude, which gradually increases, and in the course of a few weeks he has wandering, muttering delirium, at first only at night. Maniacal delirium is rare. The typhoid state supervenes towards the end. (2) The respiration is always increased in frequency. The pulmonary signs, which are generally present, have been mentioned (§ 83). (3) As a rule there are no marked local manifestations,

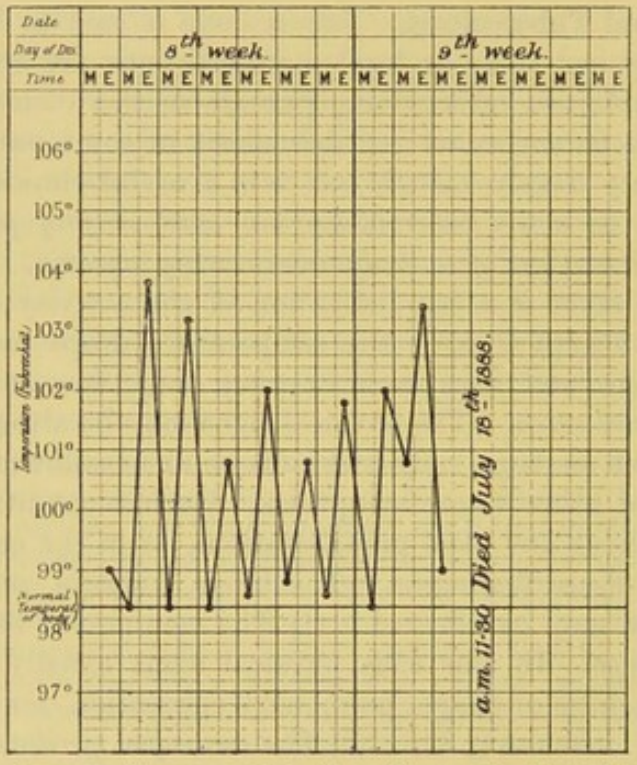


Fig. 103.—Acute Miliary Tuberculosis.—Geo. W——, æt. forty-nine, admitted to the Paddington Infirmary, July 9, and died July 18, 1888. Seven weeks' history of vague illness before admission, during which time there was profuse hæmoptysis on one occasion. The signs in the chest were very indefinite during life. After death the lungs were sparsely studded with miliary tuberculosis. The liver and peritoneum were also dotted with tiny tubercles, hardly visible to the naked eye.

but, according to the chief seat of mischief, various other signs may be elicited, such as paralysis of the cranial nerves, peritonitis, pleurisy. The spleen is nearly always enlarged.

Diagnosis.—(1) The presence of bacilli in the sputum is pathognomonic, and these should be looked for repeatedly in all cases of "*bronchitis*" attended by an intermitting pyrexia, especially in young adults. Most cases of acute miliary tuberculosis in the early stage are admitted to hospital as bronchitis, in the later stages as enteric fever. (2) The course of the disease may bear so close a resemblance to *enteric fever* that Niemeyer (before the discovery of the tubercle bacillus) stated that these disorders might be indistinguishable until the patient reached the dead-house. The Ehrlich-Diazo reaction occurs in both enteric and acute tuberculosis, but not the Widal reaction. Choroidal tuberlecs are sometimes visible on ophthalmoscopic examination, and if present settle the diagnosis.

[1] According to Reinhold (quoted by Osler, *loc. cit.*), 18 per cent. of tuberculosis cases present an inverse temperature.

Etiology.—The disease is due to a general dissemination of the tubercle bacilli throughout the body. These may have been introduced from outside, but far more frequently can be traced to some chronic or subacute focus in the patient himself, such as an old caseous or fibroid gland which appeared to be dead, or an old quiescent spot in the lungs or elsewhere.

Prognosis.—The disease is uniformly fatal in the course of four to eight or more weeks. Death occurs by coma, sometimes by pulmonary or other complications. The height and range of the temperature is a fair measure of the virulence and activity of the morbid process.

Treatment.—In such widespread mischief no treatment is of any avail. As regards prevention, it should always be remembered that convalescence from pulmonary tuberculosis should be very thoroughly re-established before treatment is stopped.

§ **382. Visceral Syphilis.**—It is now generally recognised that syphilis is a specific contagious disease like small-pox. There are two different

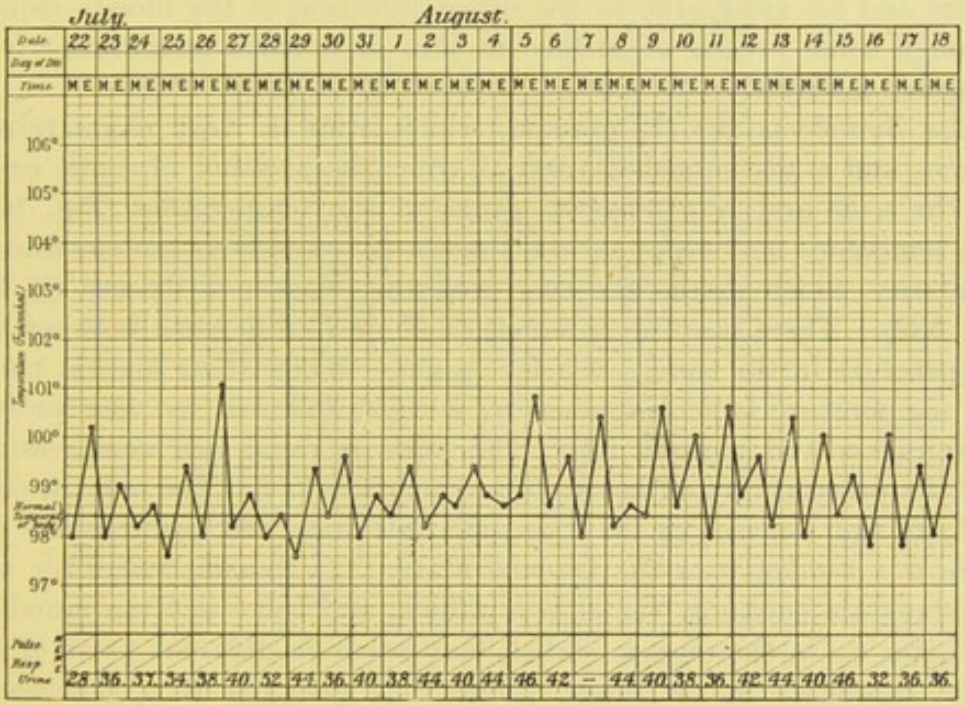


Fig. 104.—VISCERAL SYPHILIS. Annie L——, æt. sixty-six, admitted to the Paddington Infirmary, July 22, 1889 (?). The temperature subsided under iodide in large doses, but she ultimately died of exhaustion and hypostatic pneumonia. P.-M.—Gummata of liver and bones, hypertrophic cirrhosis, widespread fibrosis of organs.

stages of syphilis at which intermitting pyrexia may occur. (*a*) At the first development of the primary roseolous eruption fever there may be some elevation of temperature.[1] This is generally overlooked, but at other times it may be accompanied by thirst, loss of appetite, and shivering. It always occurs within sixty-five days of the date of the infection, and is only present if no mercury be given. (*b*) In the later secondary and tertiary stages of the disease an intermitting pyrexia

[1] This has only been generally known of late years, but it was first pointed out by Guntz in 1865, and called "general syphilitic fever." Lancereaux also pointed it out in 1866, and stated that it much resembled quotidian ague. He referred to several cases.

may occur in connection with syphilitic periostitis, or gummata of the internal organs.[1] This is a not infrequent occurrence in the course of clinical work, and syphilitic lesions of this kind are always to be suspected in cases of prolonged intermitting pyrexia, especially if it be attended by anæmia. The morning temperature is normal, but in the evening it goes up one, two, or more degrees (Fig. 104). There may also be rigors, nocturnal sweating, and paroxysms of pain in the joints, unrelieved until iodides are given; then the symptoms speedily subside. In obscure cases careful investigation should be made of the eyes, liver, ribs, clavicles, and other bones, and iodide of potassium tried.

§ **383. Acute Pyæmia** or **Septicæmia**[2] (Synonym: Ichoræmia) is a disease characterised by a wide range of temperature, accompanied by rigors and sweating, due to the direct infection of the blood—usually through some breach of surface in skin or mucous membrane—by a pyogenic microbe.

The *Symptoms* are (1) pyrexia, which runs a very characteristic course, and is distinguished from all other diseases not of septic origin by the *wide* and *very irregular* range of the temperature (Fig. 105). The remissions may occur several times a day, and have not the diurnal regularity which marks the two preceding classes of disease (§§ 381 and 382). There may be as much as 6° or 7° difference between the temperature in the course of a few hours. When at its highest point, the temperature is accompanied by a rigor, followed by very profuse perspiration and a rapid fall. The pulse is rapid and compressible, and the prostration and lassitude are very marked. The mind is clear at first, and remains so for a considerable time, but towards the end there is a tendency to the typhoid state. (2) Nausea, vomiting, and diarrhœa are common, the skin is sallow, and there is often jaundice. (3) Later on in the disease emboli may occur in different parts of the body, especially in the lungs, where they give rise to a generalised congestion and patches of pneumonic consolidation or gangrene (as in the case given in Fig. 105), and in the liver and spleen, and deposits of pus may occur in or around the joints or in other parts of the body. The serous cavities may also contain pus, constituting empyema or pyopericarditis. The occurrence of albumosuria is an indication of a focus

[1] Two cases are reported by Dr. Alfred Dufferin in the Clin. Soc. Trans., 1869. The following year a Committee reported nine cases. Dr. Bristowe reports a case of gumma of the liver in a lad of sixteen, in whom the temperature went up every evening 2° or 3°, the cause being overlooked until interstitial keratitis was discovered, and iodide was given (Clin. Soc. Trans., vol. xix.). The author has records of six similar cases in which the leading symptoms were intermitting pyrexia, anæmia, and signs referable to the liver or spleen, all of which rapidly disappeared under iodide (see also *Clin. Journ.*, December 1, 1897, p. 87.

[2] There is still some confusion in the use of these terms, but for clinical purposes they may be regarded as synonymous. In former times, when localised deposits of pus occurred, the former term was generally applied, when these were absent, the latter.

of pus in the body, and this may be an aid to diagnosis ; so also are the leucocytosis and other changes in the blood (Chapter XIV.).

Acute Osteomyelitis (or, as it used to be called, Acute Periostitis or Acute Necrosis) is a pyæmic process which may set in very suddenly, usually after an injury to one of the superficial bones, generally the tibia. In children there may be no history of injury. The diagnosis is easy when the tissues round the diseased bone are swollen, but during the first day or two of the disease pain is often complained of near a joint, and may lead one to diagnose rheumatic fever.

The *Diagnosis* of septicæmia is easy when there is an external wound or abrasion, and should never be difficult on account of the wide

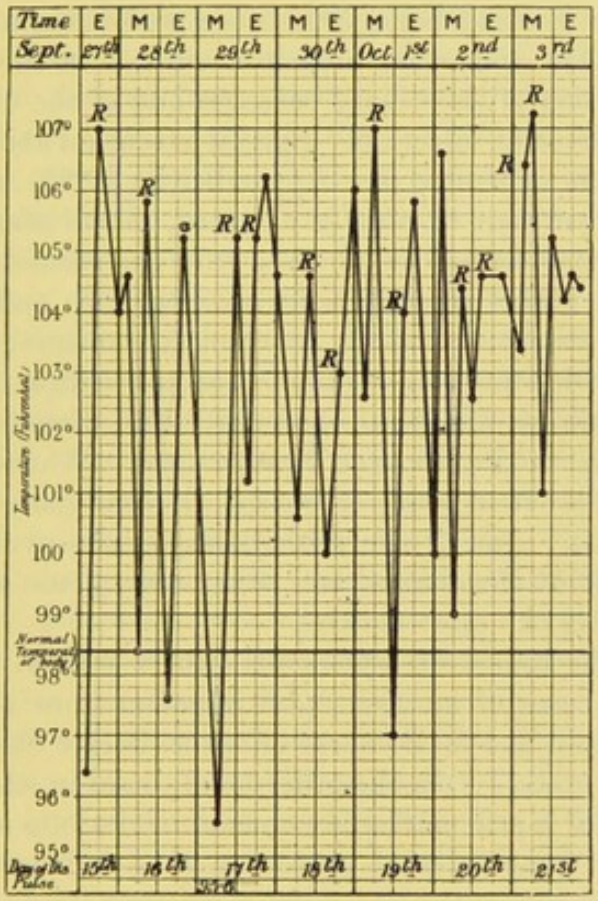


Fig. 105.—ACUTE SEPTICÆMIA (typical of an irregularly intermitting pyrexia).—Catherine W——, æt. six, admitted to hospital, September 27, 1881. She was taken ill somewhat suddenly on September 13 with shivering and vomiting. On admission she was in a condition of prostration. There were no physical signs excepting a systolic bruit over the whole cardiac area, and slight enlargement of the spleen. On the 30th there was rusty sputum with streaks of blood ; dulness and crepitations over the right back. She was delirious from time to time, and died somewhat suddenly on October 3. At the autopsy pus was found in the mastoid cells and sinus thrombosis secondary to long-standing middle ear disease, of which a history was now obtained, infarcts in the kidney and pyo-pneumothorax secondary to rupture, of one of the gangrenous-looking abscesses of the lung.

variation of the temperature, coupled with the rigors and the sweats. The chart of a typical acute case is like nothing else. When due to some internal cause, it may resemble malignant endocarditis, enteric fever, pneumonia, ague, remittent fever, and acute rheumatism. But when carefully recorded temperatures of several days are available,

and a thorough examination of the organs is made, the diagnosis should not be difficult.

Etiology.—A cause—*external* or *internal*—for pyæmia should always be carefully sought. Among *external* sources, unhealthy wounds were, before the introduction of Listerism, a prolific source of this disease, and the patients in the surgical wards were decimated by it. A mere scratch is sometimes sufficient for the introduction of the micro-organisms, and sometimes the most trivial operations are followed by pyæmia. The source of infection may arise from some *internal* condition. The internal sources are very numerous—sometimes it is caries, especially of the mastoid bone, sometimes periostitis, or osteomyelitis, sometimes an ulcer and other breach of surface in the mucous membranes. Ulceration of the biliary passages and of the urinary passages are frequent sources of infection. Special attention should be directed to the vermiform appendix (see Appendicitis, § 175) and the uterus. *Recent abortion, perhaps criminally procured, should always be borne in mind when a young woman is admitted with septicæmia.* After recent parturition, the uterus resembles an open wound, and offers a large surface for the absorption of the pyogenic organisms; hence the frequency with which septicæmia complicates parturition unless the most scrupulous cleanliness has been observed. The disease is then called Puerperal Fever, or Puerperal Septicæmia. When the poison is derived from a previous case of puerperal septicæmia it is specially virulent and fatal. Among the *predisposing causes* overcrowding, bad ventilation, want of cleanliness, and other unhygienic and septic conditions, are among the most fruitful.

Prognosis.—The *course* of septicæmia differs widely. Thus, on the one hand, some cases of intense septic infection from a *wound* or parturition run a rapid and fatal course of ten to twelve days, terminating in the "typhoid state." On the other hand, cases in which apparently small quantities of septic matter are constantly leaking into the general circulation from some *internal* source may be indefinitely prolonged over many weeks or months, the mind remaining clear the whole time. Such would appear to have been the course of the disease in the patient referred to in Fig. 106. There is, in fact, no definite line to be drawn between the *acute* septicæmia now under consideration and the *sub-acute* and *chronic* septicæmia due to pent-up pus or ulceration described below (§ 384). Acute pyæmia is a most serious and, if untreated, invariably fatal malady. Death may occur either by the intensity of the poison (typhoid state), asthenia, or complications. The *untoward symptoms* are a very high temperature, frequent rigors, or cerebral symptoms. The most frequent *complications* are (1) pneumonia, which invariably occurs in severe cases; (2) pericarditis or pleurisy, which usually become purulent, and peritonitis; and (3) suppurative inflammation of the spleen, liver, and other organs, consequent on the infec-

tive emboli ; (4) malignant endocarditis. Among the sequelæ in certain less acute cases which recover may be mentioned a destructive form of arthritis.

Treatment.—The indications are (1) to remove the cause ; (2) to inhibit the microbic toxin ; (3) to relieve the symptoms and maintain the strength. (1) If the infection is derived from a wound or some accessible purulent cavity—*e.g.*, an abscess, an empyema, acute necrosis, etc.—this should be promptly laid open, drained, and treated by antiseptic measures. Search must be made for some internal cause — *e.g.*, appendicitis — and this should, if possible, be dealt with. (2) Thanks to the researches of modern pathology, we are now in possession of an antistreptococcic serum, and several cases are on record which have been rescued from death by this means. As previously mentioned (and see § 388), several different bacteria may produce the disease, and we must identify which is in operation before we can employ the appropriate serum. The most frequent, when the source is some purulent focus or abscess, is a form of streptococcus. The great variety of forms of streptococci has proved to be the chief difficulty in the serum treatment for septicæmia. It therefore usually happens that the serum given is not antagonistic to the particular organism in operation, and a polyvalent is more likely to be successful than a monovalent serum. Recently vaccines prepared from the organism obtained from the patient's blood have been tried, with some success. (3) The administration of quinine in large doses has some controlling influence over the temperature ; antipyrin, antifebrin, and other febrifuges are also used. The internal administration of antiseptics generally has not been found of much use. Stimulants and concentrated nourishment are called for (see also §§ 391 *et seq.*).

§ **384. Subacute and Chronic Septic Conditions** (*e.g.*, **Abscess, Ulceration, etc.**) also give rise to intermitting pyrexia. The various clinical conditions met with under this heading are due to the absorption of some septic or toxic material into the circulation. The possible sources of the sepsis are very numerous, and may be grouped into two divisions —(*a*) Abscess (or pent-up pus) ; and (*b*) Ulceration (internal or external). Clinically, the former is more acute than the latter, and, indeed, the former might be called subacute, the latter, chronic septicæmia.

(*a*) Abscess (Pent-up Pus).—Pus never forms in any part of the body—*e.g.*, in the pleura (empyema), in the liver (hepatic abscess), or elsewhere—without the occurrence of " chills," " shivers," or " rigors," and an intermitting or remitting pyrexia. Before the clinical thermometer was invented, these shiverings (sometimes followed by sweating) were the chief symptoms by which the formation of matter was identified. When there is fluid in the chest, for instance, and we do not know whether it is serous or purulent, the occurrence of shivering or

sweating will often settle the question in favour of pus. The temperature in such cases presents much the same chart as that in tuberculosis, though it has not such regularly diurnal variations, and is more often accompanied by shivering or rigors. There are considerable lassitude, debility, pallor (though with a hectic flush on the cheeks), and more or less loss of flesh in course of time. Albumosuria is usually present, and is a valuable confirmatory symptom. Blood-films should always be examined, and the presence of leucocytosis with an increase in the proportion of polynuclear cells will afford strong confirmation that pus is present.

Causes.—Abscess or pent-up pus in any position may produce these symptoms, and careful search should be made for abscess of the liver,

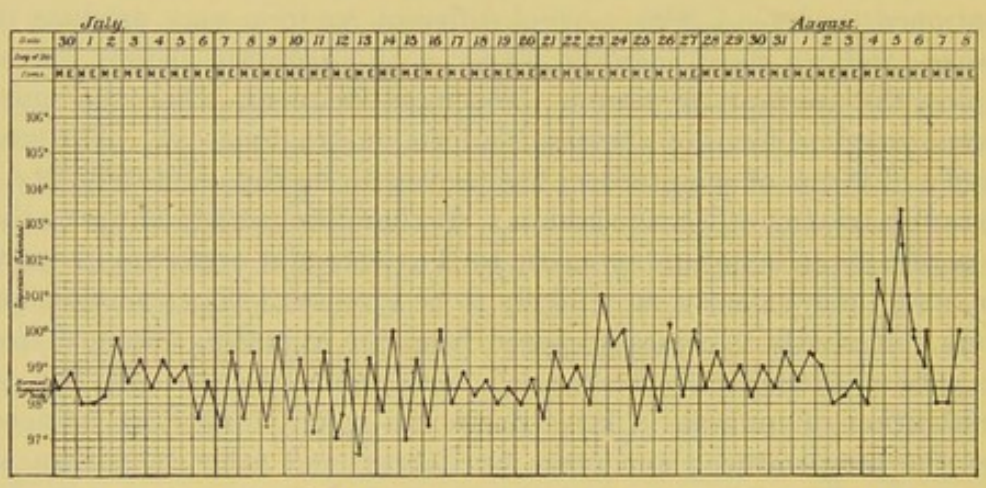


Fig. 106.—Chronic Pyæmia. Frank T——, æt. thirty-one, had had an attack of gonorrheal rheumatism two years before, from which he had recovered. The present illness had come on quite gradually a month or so before admission. Stiffness and pain in the joints being the chief symptoms, and the urethra being *absolutely normal*, it was regarded as a case of chronic rheumatism, though none of the usual remedies had any effect. The joints became progressively worse and though he complained of abdominal pains from time to time attention was not directed to that cavity. He died some two months later suddenly from perforation of the appendix vermiformis. A review of the case pointed to a chronic septic process having its origin in the appendix, and specially affecting joints which had been previously diseased.

spleen, or other organs, pelvic cellulitis, caries of the spine or mastoid bone, appendicitis (Fig. 106), intracranial abscess, empyema, pyonephrosis, etc. Pain is the chief localising symptom, but it may be wanting. On giving free exit to the pus the pyrexia should rapidly subside.

(*d*) Ulceration of an Internal or External surface (including the conditions known as "Hectic Fever," Hepatic, and Urinary Intermitting Pyrexia) is always attended by some degree of intermitting pyrexia, running a more chronic course than the foregoing. This fever also differs from the last in the usual absence of definite rigors. Sometimes the shivering may not amount to more than "chills down the spine"—thought to be ague, perhaps—and sweating which is hardly

noticed. The morning temperature is normal, or almost normal, and it is raised one or two degrees some time during the day. Anæmia and failing health are always present, and here, again, albumosuria and the blood changes just mentioned are present. This kind of fever, due to prolonged suppuration, and attended by chronic wasting, was formerly known (and is still among surgeons) as *Hectic Fever* (ἑκτικος, Greek "habitual"). When due to a discharging sinus—a sinus, for instance, connected with caries, or necrosis of a bone, or a bed-sore—the cause is obvious. But the condition may also be set up by ulceration of the intestines or any of the mucous membranes or internal passages—*e.g.*, the appendix (Fig. 106). It is called *Urinary Fever*[1] when it arises from chronic ulceration of some part of the urinary passages—*e.g.*, when a stone is impacted in the ureter, or when the patient has "stricture urethræ," or there is ulceration of the pelvis of the kidney (pyelitis). This cause may be suspected if there be a history of renal colic. Similarly, *Hepatic Intermitting Pyrexia* (ulceration of the biliary passages) may be suspected if there be a history of biliary colic. When the ulceration, due to gall-stones, is situated in the *gall-bladder*, both colic and jaundice may be entirely absent, and the patient complains of nothing but the "chills" (§ 241).

§ **385.** The **rarer causes of Intermitting Pyrexia** are fully described elsewhere, and need only to be mentioned here.

Influenza, Enteric, and other diseases described in Groups I. and II. are occasionally attended by pyrexia of an intermitting type. This is particularly so if the patient has had ague or lived in a malarial district.

Enteric Fever during the first two weeks of its course is attended by typically continued pyrexia, but in the concluding stage of the disease the pyrexia gradually drops each morning to normal, and the case may be seen for the first time in this stage. Under certain other circumstances also the temperature may be intermitting—viz. : (i.) In rare instances it may commence with symptoms of ague (Murchison) ; (ii.) in very mild cases the temperature may be intermittent ; (iii.) after lasting a few days, the fever sometimes aborts and takes on an intermitting type. For the diagnosis of the disease we now have a valuable guide in Widal's test.

Various **local inflammatory diseases,** other than the septic conditions previously mentioned, may at times be attended by intermittent pyrexia. In cirrhosis of the liver, for instance, a prolonged fever with daily oscillations has occasionally been observed.[2]

Malignant Endocarditis (Multiple Systemic Embolism) (§ 39*a*.) is always attended by pyrexia of an irregularly intermitting type, sometimes with sweatings and rigors, very much resembling the chart of septicæmia, though the temperature is usually a little more diurnally regular, and rigors are not usually so frequent

[1] The term "urinary intermitting pyrexia" is applied by some to the rigors, sometimes accompanied by fever and sweating, which follow the passage of a catheter.

[2] Dr. Frederic Taylor, Clin. Lects. on Polyorroneuritis, *Brit. Med. Journ.*, December 15, 1900.

(compare charts, Figs. 105, 107, and 14). The diagnosis of these two diseases is sometimes very difficult (§ 39*a*). Malignant Endocarditis is favoured by (i.) the existence of a loud cardiac murmur detected quite early in the case; (ii.) a history of acute rheumatism; (iii.) the secondary emboli in this disease are more frequently found in the systemic arteries, such as those of the spleen, liver, and kidneys, and they do not result in abscesses. In pyæmia the emboli occur primarily in arteries of the lungs, and from the very beginning they suppurate and form abscesses, which constitute centres of secondary infection elsewhere.

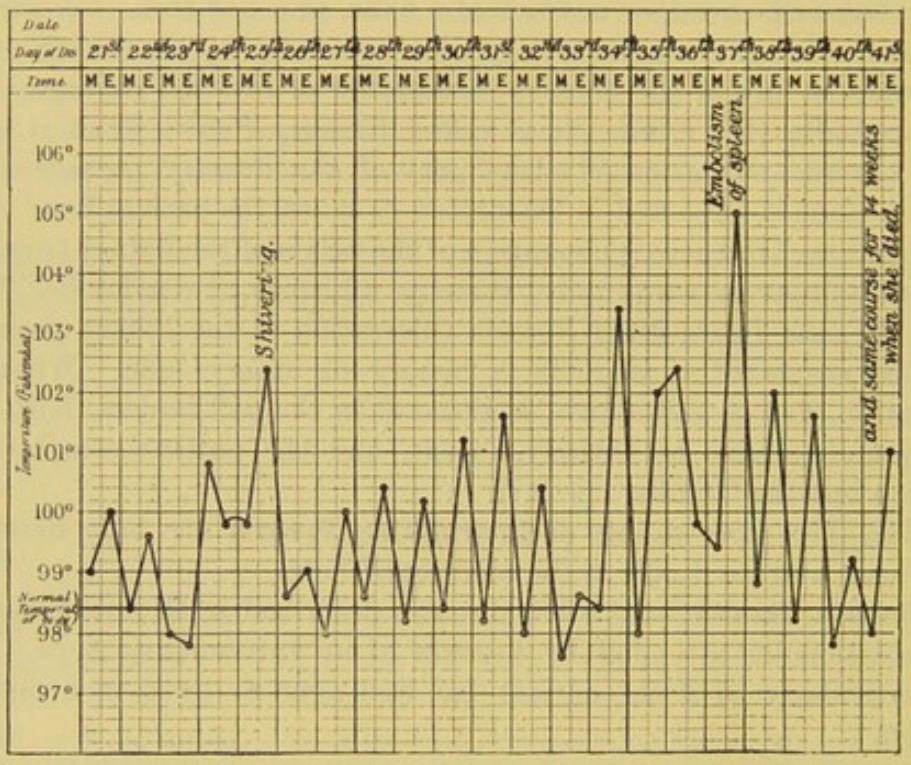


Fig. 107.—Malignant or Ulcerative Endocarditis in a female patient, æt. forty-two, who was admitted to the Paddington Infirmary in the year 1890. The three weeks shown illustrate the course of the temperature over a period of seventeen weeks, when she died. The chart of another case will be found in § 39*a*.

Lymphadenoma is recognised by the enlargement of the lymphatic glands. This enlargement is attended by pyrexia of an intermitting character.[1]

In **Pernicious Anæmia** the temperature is sometimes subnormal, but it is more frequently attended by exacerbations of fever of an intermitting type. Rigors and sweats may also occur, but they are not usual. The disease is also identified by the intense sallowness of the skin and the condition of the blood.

In **Acute Lymphatic Leukæmia** the temperature is high and irregular, somewhat resembling that of septicæmia. It can be diagnosed by the examination of the blood, when there is found to be an increase in lymphocytes.[1]

The **Opium or Morphia Habit** is attended from time to time by attacks of intermittent pyrexia, during the reaction stage, in which there are cold, hot, and sweating stages. Dr. Livenstein calls attention to this fact, and records cases where no other cause could be found, and where the attack ceased on giving opium.

[1] Forbes and Langmead, Proceedings Roy. Soc. Med., May, 1908.

THE GENERAL TREATMENT OF MICROBIC DISORDERS.

Remedial treatment has, for the most part, been given under each disease, but there are some important matters relating to all fevers in common which must now be referred to—viz., **Immunisation, Serum Therapeutics, Notification** and **Isolation, Disinfection, Diet,** and the treatment of **Pyrexia** and **Hyperpyrexia.** In the first two of these we find ourselves on the threshold of discoveries which are revolutionising the methods of treatment and prevention of infective disorders.

§ **386. Immunity.**[1]—Before entering on the treatment of microbic disorders by inoculation (serum therapeutics) it will be desirable to discuss briefly the meaning of the terms "Natural Immunity," "Acquired Immunity," "Artificial Immunity" (Active and Passive), and "Dual Immunity." A state of IMMUNITY is that in which an individual or an animal is more or less protected against contracting a certain disease. There is no such thing as absolute immunity; it is only a question of dose as compared with susceptibility. Given a large enough dose of the virus, even a hen may develop tetanus. Some of the greatest achievements in preventive and remedial medicine have recently been reached in this domain. It was a triumph of this kind which Jenner achieved, though by purely empirical means, in the latter part of the last century (1796) in the prevention of small-pox by means of vaccination. After an interval of three-quarters of a century, this important department of medicine has been enriched by the researches of Pasteur in hydrophobia and anthrax, Koch in tubercle, Loeffler (1888), Fraenkel (1890), Behring, Roux, and Kitasato in diphtheria, tetanus, and pneumonia; Haffkine in plague and cholera; Ross in malaria; Wright in enteric; and many others too numerous to mention. Some idea of the latent power for good in these researches may be grasped when it is remembered that the case-mortality of one disease alone (diphtheria) has been reduced from over 30 to under 15 per cent.

a. NATURAL OR INHERENT IMMUNITY is that form of immunity which a human being or other animal possesses at birth (or acquires during its growth), either by virtue of its species, race, or individual peculiarities. Thus, different animals are susceptible to various infective disorders in different degrees—hens are practically immune to tetanus, goats, sheep, and rats to tubercle. Again, certain races are immune to certain diseases—the negro is said to be immune to yellow fever.[2] Certain races appear to become after many generations relatively immune to some diseases—*e.g.*, measles among Europeans is now a very mild disease, but when it was accidentally introduced to the

[1] Dr. George Dean, of the Jenner Institute, very kindly read the manuscript of §§ 386 and 387 in the first edition.

[2] Compare footnote to § 372.

Fiji Islanders it became a devastating plague. Finally, certain individuals and certain families are more prone to contract infective disorders than others. I know of one family where three out of six members have had scarlatina twice, and some of the other infectious fevers more than once. Different families certainly vary in their susceptibility to infectious disorders.

b. Acquired Immunity is produced by contracting a disease in the usual way by infection. It has long been known that one attack of certain of the infectious disorders confers on the individual immunity from a second attack. The degree of immunity from second attacks differs considerably in the different diseases, and roughly one may make three groups, thus :

(α) One attack of the disease confers very strong protection against a second attack in Varicella, Scarlatina, Small-pox, Syphilis, Pertussis, Enteric, Dengue, Typhus, Yellow Fever, Mumps, and Whooping-Cough. In the first five of these, and probably in the others also, the immunity lasts practically for a lifetime.

(β) One attack confers only a moderate degree of immunity in Measles, Pneumonia, and Diphtheria.

(γ) Some confer immunity for only quite a short period—namely, Erysipelas, Cholera, Dysentery, and Influenza. However, it seems probable that *all* infective disorders confer upon the individual a certain amount of immunity—for a short time, at any rate.

From these facts the question arises, Cannot a mild attack be produced by inoculation of the infected material from a patient so as to secure immunity ? And this question was successfully answered in the case of small-pox, which was so extensively inoculated in the early part of the nineteenth century, and which was finally forbidden by law after vaccination became compulsory.

c. Artificial Immunity.—Now, what is it that creates this quality of immunity in an animal or an individual, and can this immunity be more scientifically produced ? These are questions upon which pathologists have speculated for many years, but it was not until bacteriology had become a science that a solution of them seemed possible. It is now firmly established that all infective disorders—indeed, the great majority of diseases attended by pyrexia—are due to the presence in the body of minute living organisms or bacteria, most of which can be cultivated outside the body. Some of these measure no more than $\frac{1}{10000}$ inch. It is these minute organisms which constitute the infection or germs of the several disorders. Each disease has its own particular species of microbe, which has special qualities as to size, shape, growth, life-history, pabulum, virulence, and so forth, though some have not yet been recognised. (2) Koch's four criteria, which identify a particular microbe as the specific cause of a disorder, are so important that they may be repeated here. (a) The constant presence

of the microbe in all cases of the disease ; (*b*) it can be cultivated outside the body, the cultures having constant properties ; (*c*) these cultures, when inoculated into animals, can produce in them the same disease ; and (*d*) the same microbe can again be recovered from the local lesion or from the body of the animal after death.

(*a*) *Passive Artificial Immunity.*—It is now known that the clinical manifestations and lethal effects of these organisms are due chiefly, if not altogether, not to the microbes themselves, but to the chemical products, the " TOXINS," which they evolve. The toxins and microbes can, moreover, in certain cases, be separated from one another by filtration.

The answer to the question, " On what does immunity depend ?" has been differently answered at different times. At one time it was believed to depend upon the presence in sufficient abundance of phagocytes to devour the microbes. Another theory was that an attack of the disease exhausted the store of pabulum necessary for that particular organism which was to be found in the blood. Another theory was that the microbes left behind them something which rendered the soil unsuitable for the future growth of that particular microbe. None of these were found to be quite correct, but it has now been proved that the introduction of a particular kind of microbe or its toxin into the fluids or tissues of an animal sets up a physiological reaction in the body by means of which a chemical substance (which we call ANTITOXIN) appears in the blood, which more or less neutralises the toxin and prevents its lethal action. It is the presence of this antitoxin, or the faculty of again producing it on stimulation after the bacteria have disappeared, which gives to the individual immunity from the infection of that particular disease. It follows, therefore, that if a particular microbe or its toxin can be injected into an animal, beginning with small doses, and gradually increasing them, the blood serum may be found to contain the antitoxin which antagonises the virus of the disease. In the case of diphtheria, for instance, this has been accomplished by the subcutaneous injection, at intervals of a few days into a horse (chosen chiefly because of the large quantity of serum available) of gradually increasing doses of the virus of that disease—*i.e.*, the diphtheria microbe grown in bouillon or some other suitable medium.[1] In this way it was found that the animal would gradually tolerate enormous doses of the virus—doses one-thousandth part of which would have killed the animal before the immunisation was commenced. In short, the animal in this way had become highly immunised. Next, it was found that if a relatively small quantity of the serum of such an animal were injected into other experimental animals, it protected them against many times the lethal dose of the virus. Finally, it was

[1] Some of the methods of introducing the virus are mentioned below under Active Immunity.

found that if a small dose—say 10 or 20 c.c. of the—immunised horse's serum were hypodermically injected into a human being, it rendered him similarly immune to the disease. Moreover, it was found that if the serum were injected even after the individual had contracted the disease (if given at a sufficiently early stage), it would cut the disease short, and prevent the lethal consequences. This kind of artificial immunity—produced by the injection of serum from an immunised animal—is called *Passive Immunity*.

The explanation of all this is not yet by any means certain, but Ehrlich's theory is the one now generally believed, which is as follows: The microbic *toxins* produce their lethal effects by combining with some constituents of the cells of the body for which they have a special affinity. But the corresponding *antitoxin* has a stronger affinity for the toxin than the latter has for the body cells, and therefore the antitoxin acts by combining with the toxin, and thus preventing the latter from doing harm.

(*β*) *Active Artificial Immunity.*—We have seen that Passive Immunity is that kind which is produced by the injection of the serum of an immunised animal—it is, as it were, a "borrowed" immunity. The term "Active Immunity" is applied to that kind of exemption which is acquired by the actual introduction of the microbe or its products into an animal or person whom it is desired to immunise. In the latter instance the individual manufactures his own antitoxin; in the former he receives the antitoxin which has been manufactured in the body of another.

As long ago as 1880 Pasteur began his brilliant series of experiments, showing that if animals were first inoculated with microbes weakened by age, heat, or exposure to the atmosphere, and were subsequently inoculated with the most virulent and actively growing cultures of the same organism, they had a very mild attack of the disease in question.

The following are some of the methods by which active immunity may be produced in the laboratory, the chief object being to attenuate the virus down to a suitable degree.

1. By taking some of a virulent living culture diluted with sterilised saline solution, and injecting a small *non-lethal dose*. This is followed by constitutional symptoms. When these have subsided a second dose is injected, and then a third, and so on. The blood normally possesses a certain amount of bactericidal power. It is only when too large a dose—that is to say, too many bacteria—are introduced that they become lethal, and therefore it has been found possible to immunise an animal by a series of injections of non-lethal doses of the microbe in question.

2. By the injection of a living culture of microbes, the virulence of which has been *attenuated in some way*, either by growing it in the

presence of a weak antiseptic, or in the presence of oxygen, or in a current of air. The virulence of some microbes may also be attenuated by passing them through one species (a less susceptible species, for instance) of animal, which attenuates it for another species ; or, again, by growing the culture at an abnormal temperature.

3. By the injection of *dead microbes* (killed by heat, for instance) in a series of gradually increasing doses.

4. By filtering a bacterial culture the bodies of the microbes are removed, and then the toxins alone, which remain in solution, may be injected into the animal.

§ 387. Vaccine Therapy is based on the third principle above mentioned. Sir A. E. Wright and Captain S. R. Douglas have shown by the following experiment that the phagocytic power of an individual's blood depends mainly on the serum. The white corpuscles are first separated from the blood by centrifugalisation, and are then washed free of any adherent serum. If these washed leucocytes are then mixed with bacteria and normal saline, and incubated, no phagocytosis occurs ; but if they are mixed with bacteria and *serum*, and incubated, phagocytosis results. To the hypothetical substance in the serum which promotes the ingestion of the bacteria by the leucocytes they have given the name *opsonin*. Opsonins act by preparing the bacteria for ingestion by the leucocyte ; for if bacteria be mixed with serum, and the serum be subsequently washed away, they are now able to be phagocytosed by washed leucocytes. The opsonic value of a patient's serum against a particular micro-organism can therefore be measured by the amount of phagocytosis which occurs when it is mixed and incubated with washed leucocytes and that micro-organism. To gauge the opsonic value of a patient's blood, we must compare it with the opsonic value of a normal individual as a standard, and the ration thus obtained is the *opsonic index*, which may be stated thus :

$$\text{Opsonic index of the patient's blood} = \frac{\text{Phagocytosis of the patient's serum}}{\text{Phagocytosis of normal serum}}.$$

The result of the study of the opsonic index is that treatment by inoculation of the dead virus, or *vaccine therapy*, as it is called, has been greatly extended on scientific lines, and accurate dosage can now be used at the proper time, whilst formerly the doses were usually much too great, and often did more harm than good by being employed at the wrong time. It is also of value in aiding diagnosis, for it has been shown that if the index to a particular organism is either persistently low or high, or if it fluctuates widely, that organism is present in the body.

To estimate the opsonic index, the first step is to collect a few minims of the patient's serum. This is done by means of a special U-shaped capsule (see Fig. 108). A bandage is firmly bound round the thumb to produce engorgement, and then a prick is made on its dorsal aspect just behind the nail-bed. The curved end is now applied to the small pool of blood thus formed, which runs into the capsule. The bandage may be loosened and reapplied until sufficient blood is collected to half fill the capsule. The other end is now sealed in the flame. As cooling occurs, he blood is drawn into this end, the first end is also sealed, and the sample is ready. The further details are too technical to be described in a clinical textbook, but the procedure is briefly as follows :

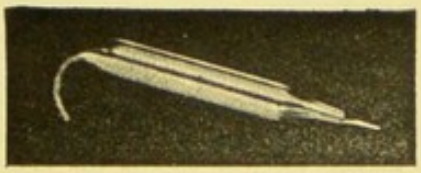


Fig. 108.—WRIGHT'S CAPSULE.

The capsule is allowed to remain until the serum has separated from the clot, and formed a layer on its surface. The end is then broken off, permitting access to the serum to be investigated. Two mixtures are made—(i.) of washed corpuscles with a bacterial emulsion free from clumps in normal saline and the patient's

serum ; (ii.) similar, but with normal serum substituted for that of the patient. Both are incubated for fifteen minutes. A film is then made of each, and stained, and the number of bacteria ingested in a hundred corpuscles counted. The opsonic index is equivalent to the ratio of the number of organisms ingested in the first film to the number in the second.

The object of " vaccination " is to keep the resistance of the blood at as high a level as possible. Each injection is usually followed by a temporary lowering of the opsonic index, and to this period the name " *negative phase* " has been given. This is succeeded by a more permanent increase of the opsonic index, or *positive phase*. If a large injection be given during the negative phase, or when the index is very low, the succeeding negative phase, by bringing the resistance to bacteria still lower, may do dangerous harm. For this reason it was at first thought essential to know the value of the opsonic index before each injection, and so regulate the time and dose. Further experience with this method of treatment has shown that in many conditions it is unnecessary to determine the index, especially in superficial lesions, where the effect can be watched. Such, for example, are staphylococcic skin lesions (multiple boils, acne), conditions which are very successfully treated by vaccine therapy. " Vaccines " for the treatment of most of the infective diseases of known etiology are now on the market, and although there are certain cases where an appeal should be made to the opsonic index, yet in its absence much good may be done by their use. As a rule, the more ill the patient is, the smaller should be the dose, and, conversely, the more chronic the disorder, the larger should it be. In the treatment of local disease means must be employed to bring the blood, the immunising power of which has been raised, to the seat of disease. This may be done by poultices, fomentations, local depletion, or the application of salt and sodium citrate. X rays and Bier's method of passive congestion act in the same way.

§ **388. Remedial Immunisation.**—General Procedure.—(1) The skin must be washed, and every possible aseptic precaution adopted. (2) A special syringe is

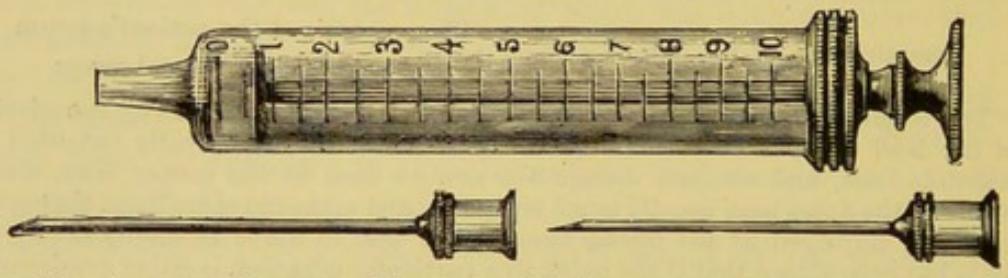


Fig. 109.—Antitoxin Syringe.—The chief qualification of an *antitoxin* syringe is that it shall be capable of thorough asepsis and that the parts can be separated for that purpose ; the piston head generally being of asbestos. Another qualification is that it shall be capable of containing the whole of one dose. The syringe here figured contains 10 c.c. The dose of a *bacterial vaccine* is generally much smaller, and an ordinary hypodermic syringe, if capable of being thoroughly asepticised, will do. It is best, if possible, to boil the parts. Failing this, thorough soaking in strong carbolic solution and afterwards thoroughly washing with sterilised water, will serve the purpose.

desirable, though any good thoroughly sterilised syringe may be used at a pinch. The best form is one in which all the parts can be taken to pieces and thoroughly boiled, because this is the best method of rendering it aseptic (Fig. 109). (3) If more than one dose has to be given, it is advisable to make the injection into a different situation each time. The most usual sites are between the shoulders, the loin or buttock, the front of the abdomen, or the back of the arm. (4) Various skin eruptions of an urticarial or erythematous type may follow the injection of any serum. (5) The treatment should be started as early as possible. If antitoxins are used, large doses should be given from the beginning, and repeated until improvement occurs. In the case of vaccination, as has been stated above, it is safer to first note the effects of small doses.

Special Methods for Each Disease.

I. Diphtheria.—An antitoxic serum has been in the market since 1895. When given early enough and in large enough doses, it has been found to be of the greatest value as a remedial agent for patients suffering from the disease (see Comparative Mortality, § 365). It has also been used as a preventive, but it is for this purpose of only limited value ; it confers immunity for a few weeks only. Some years ago the *Lancet* Commission tested several antitoxins in the market, and found many inefficient.

The *Contra-indications* for its use are given in § 365.

Method.—The remedy should be used as early as possible in the disease. A dose of at least 4,000 units[1] should be given, and repeated in half-doses every twenty-four hours until the exudation is obviously separating. Avoid injecting more than 20 c.c. at one place, if possible. The usual site for injection is subcutaneously in the flanks, but it has lately been shown that valuable time is saved if intramuscular injections are used. The best site is the intragluteal.

Effects.—In the course of twenty-four hours there should be an improvement in the patient's symptoms: the membrane ceases to extend, or perhaps begins to loosen, the swelling abates, and the rhinorrhœa is diminished. Occasional effects are urticarial or erythematous eruptions, additional rise of temperature, or joint pains and swelling.

II. Tetanus (Fig. 110).—Of late years the mortality from tetanus has been reduced by the use of an antitoxic serum. The tetanus antitoxin was first prepared by Behring and Kitasato. There is no superiority in the French antitoxin over the German and English.[2]

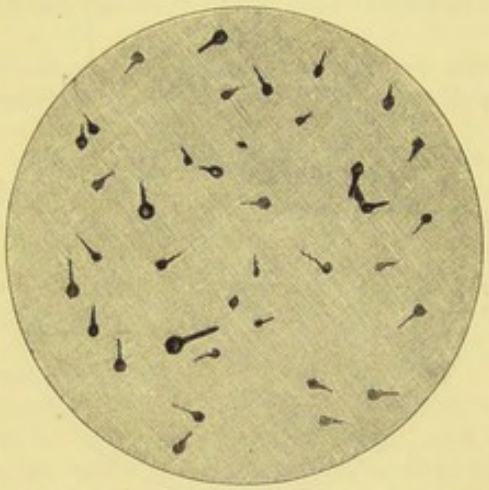


Fig. 110.—Tetanus Bacillus. — × 1,000. Cover-glass preparation. Gentian violet. Photomicrograph by Mr. Frederick Clark.

Contra-indications.—The longer the time that has elapsed between the infection through a wound and the injection of the serum, the worse is the prognosis. It is usually too late to give the serum when tetanic spasms have appeared ; yet cases are on record with recovery even after marked spasms had set in.

Method.—In a decided case of tetanus 100 c.c. of the antitoxin should be injected within twenty-four hours, at different sites, in five doses. If there be no improvement next day, give it again, and continue with daily injections of about 20 c.c. Roux has found that the serum is considerably more potent by injecting directly into the subdural space after trephining, and advanced cases have been saved by this means.[3]

Effects.—The patient should sleep well on the night following the injection, and, if fever is present, the temperature should fall considerably the next day.

III. Septicæmia and pyæmia (including Erysipelas, Malignant Endocarditis, and Puerperal Fever).—The pyogenic (pus-producing) organisms are capable of producing septicæmia—streptococcus, staphylococcus, B. coli communis (in some circumstances), etc.—and the antiserum of one will not act upon another. The antistreptococcic serum now on the market (1909) is a polyvalent serum, which

[1] In Behring's serum there are 3,000 units to 5 or 6 c.c. This same serum is now prepared at the Lister Institute, and sold through Allen and Hanburys.

[2] It is prepared in large quantities at the Lister Institute, and procurable from Allen and Hanburys, and elsewhere.

[3] Method of intracerebral injection is given in detail by Dr. Semple, *Brit. Med. Journ.*, January 7, 1899.

destroys the streptococci present in the blood in the most typical form of septicæmia.

Contra-indications.—This serum being useless to cure a septicæmia due to the presence of any pyogenic germ other than the commoner forms of streptococci, it is recommended first to examine the blood for micro-organisms. This is done by spreading some blood obtained from the patient (§ 399) on agar plates and examining the cultures.[1]

Method.—As in all cases treatment should be commenced early, and since the case may be one of mixed infection, some do not consider it desirable to wait for a bacteriological report. Start with 20 c.c., and repeat once or twice daily as long as high fever or rigors continue.

Effects.—In successful cases there should be an almost immediate fall of temperature and improvement in the condition of the patient.

Vaccine Therapy is now employed for septicæmia. A culture is made from the patient's blood, and a vaccine prepared from it. Several successful cases have been reported, including cases of malignant endocarditis, puerperal fever, and surgical septicæmia. The dose usually employed consists of from 1,000,000 to 2,000,000 organisms.

IV. Enteric Fever (Fig. 111).—In 1896 Sir A. E. Wright introduced a method of *preventive* inoculation against typhoid fever.[2] Agar cultures of typhoid bacilli are washed off with saline, killed by heating at 60° C., and the number of bacilli estimated. Two doses are inoculated, the first containing 1,000 million bacilli, the second 2,000 million bacilli, after ten days' interval. Single doses are supplied in asepticised "vaccine" tubes, with full directions as to the method of use.

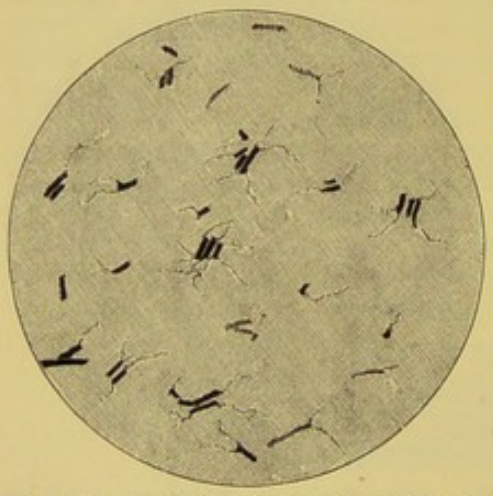


Fig. 111.—Typhoid Bacillus.—Cover-glass preparation showing flagellæ. × about 1,000. Loeffler's method. Photomicrograph by Mr. Frederick Clark.

Effects.—The immediate effects of a full dose are loss of appetite, faintness, slight fever, and restlessness, and in twenty-four to forty-eight hours the patient is well again. Locally there may be œdema; in some cases this is extensive, and Wright has found that by giving large doses of calcium chloride before the injection the tendency to local œdema is overcome. The immunity conferred probably lasts a few months; the blood serum may give the Widal reaction for this period.

An *antitoxin* for administration during an attack of enteric is on trial. It is given when the fever is remaining continuously high, and when the usual treatment appears to fail.[3]

V. Tuberculosis.—(a) Koch first introduced a "tuberculin," made by filtered bacillus cultures. This product is now known as the "old tuberculin," and is used solely for diagnostic purposes, for it only produces a reaction in an individual when he has tuberculosis in some part of his body. In this country it is used chiefly for animals.

Method.—Half a milligramme is injected, and the temperature is taken every

[1] There are numerous pyogenic organisms, the precise clinical and pathological effects of which have not yet been differentiated. The clinical effects of some of them appear to be interchangeable, as when the infection of erysipelas or scarlatina conveyed to a puerperal patient may give rise to all the symptoms of septicæmia.

[2] Wright, *Lancet*, 1896, vol. ii., p. 807, and *Brit. Med. Journ.*, 1897, vol. i., p. 256.

[3] Cases are narrated in *Brit. Med. Journ.*, 1897, vol. i., pp. 259, 578.

four hours. If during two days there is no rise, 2 milligrammes are given, and the temperature taken for two days again ; then 5 milligrammes, and the temperature again taken. If there is no elevation of temperature at any time the case is not one of tuberculosis.

(β) Koch next introduced a new tuberculin (T.R.), which consists of finely-powdered bacilli, washed free of toxins.

(γ) Lastly, he has introduced a bacillary emulsion, a suspension of finely-powdered bacilli in water and glycerine. This is rendered sterile by heating to 60° C., and is now generally employed. Sir A. E. Wright's researches have shown that far too large doses were formerly used. The dosage now ranges from $\frac{1}{50000}$ milligramme to $\frac{1}{2000}$ milligramme, and is controlled by the opsonic index, though in some cases this may be dispensed with. Dr. Latham[1] has administered tuberculin (T.R.) by the mouth with either horse serum or normal saline, with encouraging results.

VI. Hydrophobia.—The Pasteur treatment of hydrophobia has obtained a world-wide reputation. Rabbits are inoculated with the virus of hydrophobia, and their spinal cords are taken out and dried. The longer these are allowed to dry, the more attenuated is the virus contained by them. Emulsions are made of the cords, and these are injected into the patient. Weak cords, which have been dried for fifteen days, are first employed, and the virulence of the cord employed is gradually increased for ten days.

Contra-indications.—Treatment must be commenced as early as possible after the date of infection. The danger to be avoided in the treatment is a too rapid increase in the strength of the virus.

Method.—The practical points for the practitioner are (i.) to cauterise the wound at once ; and (ii.) to send the patient to the Pasteur Institution, Paris, taking with him, packed in ice, the head of the animal which bit him. Thus it can be ascertained whether the bite was dangerous or not.

The *results* of this method of treatment are very encouraging. The ordinary mortality of bitten patients, before the institution of this treatment, was about 16 per cent., but from 1886 to 1895 (17,337 cases) the mortality was 0·48 per cent.[2] A serum treatment of hydrophobia is at present on trial.

VII. Plague.—The serum treatment of plague is still on trial in India. Yersin's[3] or Lustig's[4] serum is employed. It appears to be an antibacterial serum, and is attended with considerable success. As so few cases can be obtained in time for this treatment, Haffkine[5] has introduced a method of preventive inoculation by attenuated cultures, which it is believed confer immunity for about one year.

Contra-indications.—The treatment must be commenced on the first day of the illness, because the course of the disease is so rapid and severe that later administration cannot check its progress.

Method.—From 20 to 40 c.c. are injected daily for one to ten days, according to the nature of the case.

Effects.—Cases have recovered in two days when the treatment was commenced on the first day of illness. In such cases the mortality has been greatly reduced.

VIII. Cholera.—A cholera antitoxin has been introduced, but is still on trial. Haffkine has been able to confer immunity for a year by inoculating two or three times with attenuated cholera cultures, then with more exalted virus. The results were encouraging : fewer people were attacked ; but those who were attacked did not have a modified or milder form of the disease. Hence he has more recently attempted to produce an inoculating material which shall contain antitoxic properties as well as antibacterial.

IX. Snake Poison.—Calmette introduced an antitoxic serum for the poison of snake bite, which is known as "antivenene." It is effective against the venom

[1] Proc. Royal Soc. of Med., vol. i., 1906.
[2] "Bacteriology," Muir and Ritchie, p. 472, first edition.
[3] *Lancet*, 1897, vol. i., p. 604 ; and vol. ii., p. 1546.
[4] *Lancet*, 1897, vol. ii., p. 1361.
[5] *Lancet*, 1899, June 24.

of the colubrine snakes, but often fails to neutralise that of the viperine species. The serum can be kept for a long period in a tropical climate without losing its properties.

Method.—Inject as soon as possible after the bite at least 10 c.c. of Calmette's antivenene, and repeat the dose some hours later.[1]

Effects.—The patient recovers very soon if the injection is given before unconsciousness or paralysis set in. Even if given when respiratory paralysis threatens, this dangerous symptom may not ensue, and the paralysis of the limbs usually disappears in less than two days.

X. Pneumonia.—The serum treatment of pneumonia has as yet been tried chiefly on animals. In animals remarkable results have been obtained. Thus 1 c.c. of Pane's serum protected a rabbit against 3,000 lethal doses of a living culture of the pneumococcus.[2] Good results were expected from this method of treatment in the human being, but it has proved disappointing.[3] The serum of a patient who has recovered from pneumonia protects, to some extent, rabbits from the pneumococcus. This serum is probably antibacterial. It appears that there are several varieties of pneumococci, and that one kind does not protect against another. Vaccines are beginning to be employed.

XI. Anthrax.—Animals have been inoculated with increasingly virulent doses of anthrax cultures, and the results are encouraging as a preventive. The serum of the inoculated animals is probably antibacterial.

XII. Cerebro-Spinal Fever.—Flexner has introduced an antibacterial serum. This was injected into the spinal canal during the recent epidemic in Belfast, and was thought to have considerably reduced the mortality. The dose is employed from 15 to 30 c.c.

§ 389. Notification and Isolation.—Two duties are laid upon the medical practitioner in cases of the commoner infectious maladies: (1) Notification of the case to the medical officer of health of the district in which the case arises. The notifiable complaints in most districts are scarlatina, diphtheria, "membranous croup," enteric fever, and "continued" fever, small-pox, cholera, erysipelas, typhus, relapsing fever, puerperal fever, and plague (measles, varicella, and phthisis are *voluntarily* notifiable, and the public ambulances may be used for the first two). A medical man is bound, under a penalty of forty shillings, to notify any of the maladies named "immediately on becoming aware" of its existence. (2) Removal of the patient to a fever hospital is compulsory, unless the parents or guardians can make *proper* and *adequate* arrangements for the isolation of the case at home. In some places the removal is superintended by the medical officer of health. In the metropolitan area the medical practitioner should at once communicate with the central office of the Metropolitan Asylums Board, Victoria Embankment, E.C., when an ambulance will promptly be sent for the case. Their telegraphic address is "Asylums Board, London," and the particulars required to be sent are Name, Address, Disease, Age and Sex of patient, and Severity of case.

It is far better for the patient and for his relations that he should be removed to a properly organised Fever Hospital; but to isolate a patient at home, hang a sheet, constantly wet with carbolic solution (1 in 20), across the door or passage. Carpets, curtains, and superfluous furniture should have been previously removed. Books and articles in use must be such as can be afterwards burned. Ventilation must be carried out as described on p. 551. The nurse in charge of an infectious case should wear a washable dress when on duty, and should hold no communication with others, nor should she go out of doors without having first changed her wearing apparel, and, if possible, taken a bath. An airy, quiet room *at the top of the house* having cubic space of about $12 \times 12 \times 10$ feet, is desirable. The air in this space

[1] Cases narrated in *Brit. Med. Journ.*, 1899, vol. ii., pp. 1432, 1732, and elsewhere.

[2] Washbourne, Med. Soc. Report, *Lancet*, October 13, 1899.

[3] A successful case is narrated by Dr. Harnett in the *Brit. Med. Journ.*, 1897, vol. i., p. 1279.

requires to be changed three or four times in every hour. Only the furniture in immediate use should be allowed to remain. The carpet should be taken up, and all stuffed furniture removed. The bedstead should be so placed as to be accessible on both sides. The temperature, read on a thermometer suspended near the bed, and away from draughts, should be 60° F.

Ventilation must be ample in fever cases, because of the danger of mixed infections. There are reasons for believing that the tonsils are sometimes the portal for infection, and that, perhaps, is the reason why mixed infections are more apt to arise in cases of scarlatina when there is not free ventilation and sufficient cubic space. This partly explains the higher death-rate from infectious diseases when overcrowding occurred in former days. The direction of the wind should be constantly noted, and, to avoid draught, the windows or ventilators opened on the side of the room away from the wind. A " sash-board " is an excellent contrivance for avoiding draught. It should be about 6 to 8 inches broad, and fit across the bottom of the window, so that the lower sash can be raised without a visible opening, and then ventilation takes place behind the sash-board, and also *in the middle* of the window, the air in both cases being directed upwards. The chief principle involved in all ventilation is that the current of air always takes place from a colder to a hotter medium—usually, therefore, from outside to the inside of a room. The chimney, when the fire is alight, is the only reliable *exit*. Make the window your *inlet* in preference to the door.

§ 390. Disinfection and Prevention.—Before describing the means employed for disinfection, it is necessary briefly to describe the way in which microbic disorders are propagated. Since bacteriology has become a science, great advance has been made in this direction. There are three principal ways by which infection is conveyed—by the *air*, by *water* or other ingesta, and by direct *contact* or inoculation—and microbic diseases may be thus classified.

(*a*) As regards the *air-borne* group, there is considerable variation in their infectivity, also the distance to which the contagion in an active state may be carried through the air. For instance, erysipelas and typhus probably do not spread beyond a few feet, but small-pox and scarlatina may spread for many yards, some say the former spreads to a distance of a mile or more.[1] Air-borne diseases can also be conveyed by furniture and other articles in common use. The portal by which most of these diseases enter the system is generally believed to be the lungs, but certain facts lately observed point to the tonsils, throat, and nose as possible channels for their introduction. Some of this group may be conveyed by milk, and it is possible that other ingesta may become contaminated by the contagia of these diseases. The air-borne diseases are as follows: Varicella, Scarlet Fever, Small-pox, Measles, Rubeola, Typhus, Dengue (air-borne or miasmatic), Diphtheria, Erysipelas, sometimes Influenza, Mumps, Rheumatic Fever, and Whooping-cough. Pulmonary Tuberculosis arises from the inhalation of contaminated particles.

(*b*) The *water-borne* group only comprises three diseases—viz., Enteric fever, Cholera, Dysentery. Two facts form the basis of the propagation and prevention of these diseases: (1) All matters coming from the patient's bowels and stomach are infective, in enteric the urine also; and (2) to produce the disease the virus must be introduced by the mouth into the alimentary canal.

(*c*) The *third group* comprises disorders the infection of which must be introduced into the blood or tissues of the body in order to produce the disease, either by means of a wound or a scratch which may perhaps have escaped notice. Our profession pays a penalty every year to this group of disorders when, perhaps, some overworked practitioner is called to the bedside of a *syphilitic* lying-in woman, and forgets to examine the margins of his finger-nails, where some crack or unsuspected scratch will be the means of the introduction of the syphilitic poison. Some of these disorders were formerly described as *miasmatic*—*i.e.*, dependent upon

[1] Some valuable data on this question were collected by the author from the Warrington small-pox epidemic, 1901-1902.—Appendix to the Report of the Roy. Com. on Vaccination.

some meteorological, telluric, or climatic influence, which we did not understand. *Ague* is an example of these diseases, but it is now known to be directly introduced into the blood of the patient by the bite of a mosquito. *Tetanus* and *Plague* are other examples; tetanus is introduced through a wound or scratch which has become contaminated with the soil, plague is conveyed by rats. *Septicæmia* is due to the internal or external contamination of the blood-current, and all kinds of dust probably contain pyogenic—*i.e.*, septicæmic—microbes. *Glanders* is contracted from horses by the contamination of a wound or scratch; and *Anthrax* is contracted by woolsorters and others who come in contact with the hides of animals containing the contagion, and thus inoculate a scratch or inhale the dust. *Tuberculosis* is placed under this group because it is sometimes undoubtedly inoculated into a wound, giving rise to lupus vulgaris or verruca necrogenica on the hand. *Hydrophobia* must be inoculated, generally by the bite of an animal suffering from rabies. *Gonorrhœa* is conveyed either to the urethra or conjunctiva, but whether a breach of surface is necessary or not is not known.

It follows, therefore, that the **procedure for disinfection** differs somewhat in the case of air-borne diseases, water-borne diseases, and those introduced by the contamination of a wound or scratch.

1. For Air-borne Diseases—

(i.) The *linen*, before washing, should be left to soak in carbolic solution (1 in 80). In any case, rather than leave clothes and linen exposed to the air, keep under water until they can be removed (a wineglass of carbolic acid to a gallon of water is roughly 1 in 80).

(ii.) *Clothes and Bedding.*—If a disinfecting oven (at a temperature of not less than 210° F. or more than 320° F.) or a steam-heated chamber at 212° F. is not available, they may be spread out in the room, and treated by sulphur (see below) or formalin spray. It is very doubtful if the fumigation of clothes by sulphur is of much use. *Washable articles should be plunged into a tub containing carbolic solution* (1 in 80, *vide supra*), *and then sent to the wash*, when they should be boiled.

(iii.) The *patient*, before returning to his friends, must have several warm baths, and be washed with carbolic soap. This is very necessary in diseases where desquamation occurs, and anointing with carbolised oil is recommended.

(iv.) *To Disinfect the Room.*—Close the windows and doors, and stop up all crevices. Melt some sulphur over a fire in a saucepan or small iron bucket, set it alight, and place it on an old tray in the middle of a room; then shut up the room for twenty-four hours. Use 1½ pounds of sulphur for every 1,000 cubic feet—3 pounds for an ordinary sized room. The fumes are very suffocating, but they will not hurt anything if the air be dry, excepting brass, and this may be protected by smearing it over with vaseline. A whole house may be fumigated in this way from the basement by closing the windows, stopping up the chimneys with newspaper, and opening the doors of communication. Nowadays it is recognised that a gaseous is much less thorough than a fluid disinfectant, and the walls should be washed with perchloride of mercury or saturated with formalin sprays (*Lancet*, August 13, 1898, Brit. Med. Assoc. Discussion, State Medicine). Formalin may be used as a vapour in the same way as sulphur. It is conveniently supplied as candles, which may be burnt.

2. For Water-borne Diseases—

(i.) The *excreta*, if practicable, should be burned; if not, before being removed they should be covered with chlorinated lime or carbolic solution (1 in 40, *vide supra*).

(ii.) The *underlinen*, towels, bedding, etc., must be boiled, or treated very carefully as in air-borne diseases.

(iii.) All *drinking-water* should be boiled if there is the slightest suspicion of its being contaminated by leakage, soakage (however small) from cesspools, drains, or the reckless casting of slops, etc.

List of common disinfectants: Extreme heat (200° F. or more, and preferably moist); fumes of burning sulphur (SO_2); chlorinated lime; chlorine, evolved from chlorinated lime by hydrochloric acid (spirits of salts); carbolic acid (a wineglass

of carbolic acid to each gallon of boiling water is roughly 1 in 80 solution); formic aldehyde; permanganate of potash (Condy's fluid); chinosol; lysol; sulphate of iron; sulphate of copper; creolin; corrosive sublimate; terebene; thymol; eucalyptol; sanitas.

3. Disinfection and the PREVENTION OF DISEASES INCLUDED IN OUR THIRD GROUP differs in each individual case. Thus septicæmia and tetanus almost ceased in surgical cases with the introduction of cleanliness and asepsis. Ague depends mainly upon mosquitoes, and the extermination of these is now engaging much attention.

§ **391. Diet** in fevers is a question of great importance, because all the secretions and excretions are so much diminished or modified. It should consist mainly of milk and meat juices. No more than 3½ pints of milk per diem should be given, fresh—sterilised if possible—or scalded (not boiled), in small quantities at a time; and it may often with advantage be diluted with half or a third of water, soda-water, or barley-water. If curds are passed, the milk may be peptonised, or sodium citrate may be added in the proportion of 2 grains to the ounce of milk. Lime-water may be used instead if diarrhœa be present, or if there be constipation a drachm of sodium bicarbonate to the pint. If milk is not well tolerated, whey or cream may be given, or the yolks of eggs or egg-flip. Beef-tea, chicken or mutton broth, about a pint in the twenty-four hours, should also be given, and may be supplemented by some of the many modern substitutes (*e.g.*, Liebig's or Valentine's extract, Bovril, etc.). Some methods of preparing invalid foods are given in § 212. Iced water is very agreeable, but it generally increases the thirst. Fresh lemonade may be advantageously substituted by mixing a drachm or two of bi-tartrate of potash, with a little sugar, to the pint of water.

§ **392.** The **Treatment of pyrexia and hyperpyrexia** comprises six indications:

1. *Heat production can be diminished and heat loss increased* to some extent by means of *drugs*, known as antipyretics, such as antifebrine, antipyrine, and phenacetin. The first of these is, on the whole, most efficacious for reducing temperature, but it requires care, on account of its depressing effect on the heart, and the reaction which follows some hours later. Quinine in full doses (say 5 grains every three or four hours), may be given until the temperature comes down or physiological symptoms are produced (singing in the ears, deafness, headache, etc.). Salicylates, especially in rheumatic affections, and aconite are also useful. Among the more familiar but less efficacious febrifuges and diaphoretics are liquor ammoniæ acetatis, potassium nitrate, spiritus ætheris nitrosi, and camphor; also lemon drinks, dilute acids, and salines. Kairin is said to reduce febrile temperature very rapidly, but is apt to produce profuse sweating (which may, however, be combated by atropine), or shivering (which may be combated by quinine), or collapse, if the dose be too large. Parthenine (an alkaloid derived from Parthenum histerophus (Linneus), has been known as a febrifuge for a long while to the country people of Havana, where its common name is Escoba Amarga. Febrifuge doses of 2 grammes may be given. It was tried with success in eighty patients by Dr. Ramirez Tovar.

2. *To aid the loss of heat* is a method of treatment called for in cases of hyperpyrexia (*i.e.*, when the temperature reaches above 104·5° F.), by means of the graduated bath, the wet pack, sponging, the application of ice-bags, or Leiter's Coil.

The Graduated Bath.—Place the patient in a bath one-third full of water at 90° or 95° F. Every five minutes reduce the temperature 5° until 60° F. is reached. If the patient's fever be not then reduced to 100° F. or lower, he may be left in a further quarter of an hour if his pulse be a fair strength. The pulse must be closely watched, and alcohol given if necessary.

The Wet Pack.—Take off the nightshirt and superfluous bed-clothes, and place the patient on a blanket. Moderately wring a sheet out of ice-cold water and lay it along his side. Gently roll him over on to it, and completely envelop him in it, head and all, except the face, so that it is next his skin, without creases or air, between the legs and beneath the arms. Cover these latter with wet towels. Then put two cradles over the patient, and blankets over all. Leave him thus

packed for twenty to forty minutes, until his temperature, taken in the mouth, is reduced to the required extent.

Tepid Sponging.—Lay the patient in a blanket and sponge him gradually all over with tepid water (about 75°). Do half the body at a time, the other half being covered up. Continue the process for twenty to forty minutes, until the fever is reduced.

The application of ice in large ice-bags for the head, chest, and abdomen has been used when other means are not available, but the weight of the bags and their localised application are objections to their use. *Leiter's Coil* consists of a specially made coil of metal or rubber tubing through which cold water is continually running. This coil may be applied to the head, abdomen, or chest. Neither of these two last methods are recommended for fever cases.

3. To *diminish the work done by the internal organs* is another means of combating pyrexia. This may be done by diet (*vide supra*), and by promoting the action of the skin and bowels, in order to relieve the kidneys. Saline purges fulfil the latter indication (F. 46, 51, 55, and 63, are useful).

4. In all fevers it is necessary *to watch the heart* very carefully, and, if necessary, to steady it by means of strychnine and digitalis, or to aid its flagging power by means of stimulants. The pulse should be examined several times a day in all fever cases, if only for this purpose.

5. *Symptomatic treatment* may also be necessary, but this has been dealt with in the preceding pages. The constipation must be relieved by calomel or saline purges, the thirst by lemon water in sips (not ice), and the headache by phenacetin.

6. The last indication is *to watch for and treat complications* as they arise. The chief of these are (i.) cardiac (*vide supra*), and (ii.) delirium and insomnia. If the delirium be of the *raving* kind, chloral and bromides should be given in full doses; if, on the other hand, it be of the *muttering* or typhoid variety, stimulants and ammonia are indicated. Insomnia may be relieved by the same treatment, and alcohol may be useful in this respect. (iii.) Pulmonary complications, (iv.) suppression or retention of urine, and (v.) collapse, are all dealt with elsewhere.

CHAPTER XVI

GENERAL DEBILITY, PALLOR, EMACIATION

A FEELING of general weakness and lassitude is a symptom common to a great many diseases, but we are now concerned with those in which this is the only obvious, or at least the most prominent, symptom for which the patient seeks relief. Diseases in which debility is the chief symptom may be classified clinically into two great groups according to whether they come on acutely and are attended by pyrexia or not. Debility coming on acutely with pyrexia was fully dealt with in the preceding chapter. There still remains a large group of diseases which we shall now have to consider in which the weakness is of gradual onset, runs a chronic and indefinite course, and is unattended for the most part by any notable elevation of temperature ; and these diseases may be attended by anæmia or by emaciation. Here we shall often meet with the beginnings of disease, beginnings which may, however, lead to a serious and fatal issue. It is, therefore, of the highest importance that an exact diagnosis should be made, and treatment adopted as early as possible.

The debilitating conditions mentioned in this chapter may be unattended by any other symptom, or only by the pallor of anæmia or the wasting of malnutrition, and many give rise to no characteristic anatomical changes after death. Their pathology in some instances is extremely obscure, and its elucidation in the future must largely depend upon the co-operation of the analytical chemist with the physiologist and the physician, a large proportion of them being undoubtedly due either to some kind of autotoxic, hæmolytic, hæmogenitic, or other blood changes on the one hand, or to malnutrition and a profound disturbance of metabolism on the other.

PART A. SYMPTOMATOLOGY.

§ **393. General Debility.**—Malaise, lassitude, inability to complete a day's work, are some of the terms used to describe the symptom under consideration, which is essentially chronic in its course. The weakness is generalised, and it may affect the mind as well as the body, for there

is not only a disinclination to take muscular exercise, but an inability to concentrate the attention or accomplish mental work. The weakness may vary in kind and degree from very slight malaise to a total incapacity to move. Many diseases in this category are apt to be overlooked in their earlier and more curable phases. The patient may attribute his ailment to "slight digestive derangement," or think he has "been working too hard," or "wants a change," and perhaps he calls on his doctor "as he was passing" just to confirm his own diagnosis and "give him a tonic." These cases may tax the young practitioner's skill and tact in several ways. Fresh from studying instances of marked diseases in hospital, he may regard these cases as trivial and "uninteresting"; and even if he detects the beginning of some insidious malady the patient may meet his suggestion of serious ailment not only with surprise, but even with resentment and distrust. Some tact, therefore, is required, and the practitioner may find it wise to place himself in communication with some discreet friend or relative of the patient.

Fallacies.—The distinction of general debility from *paralysis* is not usually difficult, though patients with multiple peripheral neuritis, early paraplegia, general paralysis of the insane, bulbar paralysis, and various other forms of paresis, often come to my clinique complaining simply of weakness. Cases of *malingering* offer far greater difficulty in diagnosis from general debility, for in both cases we are almost entirely dependent upon the patient's own statements. The question of motive should be considered and an exhaustive examination made by the most up-to-date scientific apparatus, but even then we may in justice be compelled to give the patient the benefit of the doubt. My experience at the Paddington Infirmary taught me in many cases that it is only by keeping the patient under daily observation, and with the aid of intelligent, experienced, and well-trained nurses, that a correct conclusion can be gained. I have no doubt that large numbers of able-bodied malingerers gain admission to the infirmaries in Great Britain as cases of general debility. *Hysteria* and *neurasthenia* may require to be distinguished from debility. The *Causes* of debility are discussed in §§ 401 and 418.

Pallor of the Skin—*i.e.*, deficiency of its normal colour—is a frequent accompaniment of cases in which debility is complained of by the patient, and the experienced observer can detect considerable variations in the different varieties of pallor belonging to several diseases which will be alluded to shortly (§ 401 *et seq.*).

Fallacies.—Slight *jaundice* may resemble some forms of pallor, but in jaundice the sclerotics are unmistakably tinged and the urine may contain bile pigment. In *town-dwellers* who suffer from a deficiency both of fresh air and sunlight, pallor of the face is common, and in certain "delicate" families a pale face is more or less normal. Euro-

peans who have lived long in the *tropics* are habitually pale and "anæmic" looking, but the blood may not reveal any changes of anæmia. On the other hand, chlorotic girls may occasionally present flushing of the face and redness of the lips, though undoubtedly suffering from anæmia.

Emaciation, or loss of flesh, may also be associated with general debility, and its presence adds considerably to the gravity of a case, for it indicates either serious organic disease such as cancer or tubercle, or definite defect in the alimentation or metabolism of the body, such as is produced by intestinal trouble or chronic Bright's disease. It is manifested to the patient by his clothes becoming looser, or his face becoming thinner, and to the physician by pinching up a fold of skin between the finger and thumb. But the only reliable test is a definite loss of weight, and it is advisable at the outset to ascertain and record the weight of all patients who come to us complaining of debility. To ascertain the net weight, one-twentieth of the gross weight may be deducted for summer, and about one-eighteenth for winter, clothes. Every consulting-room should be provided with scales. The causes of emaciation are discussed in § 415 *et seq.*

Fallacies.—A normal loss of adipose tissue may occur about the climacteric, but the reverse is quite as usual. In advancing years loss of flesh, or the reverse, is normal in some families; both are largely a question of heredity. Amyotrophy, unless generalised, is not apt to be confused with emaciation; it is usually localised. The diet a person has been taking will, within certain limits, influence his weight considerably, and one who has been taking only nitrogenous food (*e.g.*, the so-called Salisbury diet) may be many pounds under his normal weight.

PART B. PHYSICAL EXAMINATION.

§ **394.** The physical examination of cases of general debility, pallor, or emaciation, comprises (1) Examination of the Viscera; (2) Observations on the Weight, and in some cases on the Temperature; and (3) An Examination of the Blood.

1. An examination of the Viscera should be very systematically and thoroughly conducted (see Scheme, pp. 7 and 8), because we may be dealing with some incipient disease, the signs of which are obscure. Inquiries should be specially directed to the state of the digestive organs, and the urine should be carefully examined. Special importance will attach to the latter when we know more about the causes and consequences of abnormal hæmolysis (blood destruction).

2. The Weight of the patient should be noted, and, if possible, compared with previous records. It may be desirable also to take the patient's Temperature if any pyrexia be suspected, and to obtain a series of records (§ 347).

3. An examination of the Blood is generally necessary, especially in cases where any form of anæmia is suspected. This in its complete form consists of (1) estimation of hæmoglobin ; (2) blood counts of the red and white corpuscles ; (3) examination of blood-films. In most cases these three will be sufficient for a routine examination ; but in other cases it is necessary to make (4) an examination for parasites and other abnormal constituents ; and (5) certain physical and chemical properties of the blood.

Examination of the Blood.

§ **395. Apparatus and Methods**—Apparatus Required.—The cover-glasses used in blood examination must be perfectly clean and free from greasiness, and for blood films they must be specially thin. Cover-slips should be cleaned in the following manner: (i.) Drop the cover-slips one by one in an enamelled iron pot containing 10 per cent. solution of chromic acid, and boil gently for twenty minutes. (ii.) Turn the slips into a flat glass dish and wash in running tap water until all trace of yellow colour has disappeared. (iii.) Wash in rectified spirit, then transfer them by means of a clean forceps to a small beaker of absolute alcohol. (iv.) Draw off the alcohol and transfer the slips by the forceps to a wide-mouthed glass pot containing absolute alcohol in which they are to be stored. After once being placed in chromic acid the slips must on no account be touched by the fingers. They always require delicate manipulation ; a pair of spring forceps which will lie down horizontally is useful for holding them. Cover-glasses should never be laid flat on the table, but have one edge on the table, the other edge leaning on some object. Glass slides require to be cleansed from grease. Alcohol and ether are necessary for cleansing and disinfecting the skin ; my own practice is to use A.C.E. mixture.

For the *estimation of hæmoglobin* the following apparatus are in use : the Tallqvist scale, Gowers' hæmoglobinometer, Von Fleischl's hæmometer, and Haldane's and Oliver's hæmoglobinometers ; Gowers' instrument is the one most used. For *counting the blood-cells* the Thoma-Zeiss or the Gowers' hæmocytometer is employed. Diluting solutions are required for this purpose. For counting the red cells a solution of sulphate of soda of specific gravity 1025 may be used, or a mixture of sodium sulphate gr. 104, acetic acid ℥i., aq. destill. ℥vi. For counting the white cells a 0·3 per cent. solution of acetic acid coloured by methyl green is used, or Toison's fluid (methyl violet, 0·025 grm. ; neutral glycerine, 30·0 c.c. ; distilled water, 80·0 c.c. Add to this a solution of sodium chloride 1·0 grm., sodium sulphate 8·0 grm., distilled water 80·0 c.c., and filter). The instruments must be carefully cleaned before being put away, first with water, then with alcohol or ether, then with water again.

For *staining the blood* the following stains are those commonly used : (1) Eosin grm. xv., abs. alcohol 70·0 c.c., aq. destill. 30·0 c.c. (2) Lœffler's methylen blue—conc. alcoholic soln. of meth. blue, 1 c.c., caustic potash in 00·1 per cent. aqueous solution, 3 c.c. (3) Ehrlich's triple or triacid stain can be obtained from Grübler and Co. of Leipsic in the form of a powder (the Ehrlich-Heidenhain powder), and is mixed thus : Powder, gr. xv., abs. alcohol 1 c.c., aq. destill. 6 c.c. The powder contains orange G. in acid fuchsin and methyl green. (4) Jenner's stain is a specially prepared mixture of eosin and methylen blue in methyl alcohol. (5) Leishman's stain[1] is of more recent date, but is already widely used, and is supplanting other staining methods. It is particularly valuable as a stain for parasites.

Method of Obtaining Blood.—Certain precautions are necessary to obtain satisfactory results in procuring a specimen of blood for examination. A series of observations on the same patient should be carried out as far as possible always

[1] Leishman's stain is prepared by Grübler, of Leipsic. The method of preparation is described in the *Brit. Med. Journ.*, 1901, vol. ii., p. 757.

under the same conditions, because there are physiological alterations in the constituents of the blood after meals, cold baths, and exercise. Blood can be obtained from the back of the finger just above the nail, a handkerchief or string being tied round the selected finger in order to render it hyperæmic. This process is painful, and the proportion of corpuscles to the serum is slightly altered by the artificially induced hyperæmia. It is better to obtain the blood from the lobe of the ear. Cleanse the lobe with soap and water, then with ether, and in cleansing render it hyperæmic by rubbing. The skin is then lightly and rapidly pricked. For this purpose a guarded two-edged lancet is the best instrument, but a needle or vaccination lancet may be employed. The lancet should be rendered sterile by wiping with ether, not heated, as that blunts the point. The blood must not be squeezed out, or it will be diluted with lymph from the tissues.

§ 396. Estimation of the Hæmoglobin.—In estimating the quality of the blood the number of red cells is of less importance than the amount of hæmoglobin, their active constituent. Hæmoglobin may be roughly estimated by the *Tallqvist scale*,[1] which consists of a lithographed scale of tints. A drop of blood is sucked up

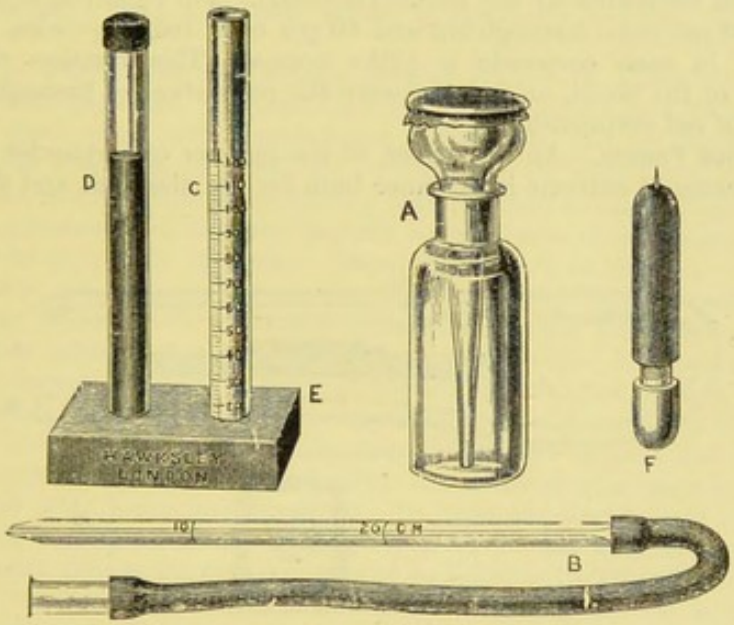


Fig. 112.—Gowers' Hæmoglobinometer.

by one of the pieces of blotting-paper supplied, and compared with the scale of tints as soon as the stain has lost its humid gloss. The figures beside the tints represent the percentage of hæmoglobin present, normal being 100. The estimation can only be performed in full daylight.

Another method of estimating the hæmoglobin is by means of *Gowers' hæmoglobinometer*, which consists of the following apparatus (see Fig. 112) : (A) A pipette dropping-bottle with rubber top ; (B) a capillary pipette marked at 20 cmm. ; (C) an open tube of the same size as D, graduated so that 100 degrees equals 20 cmm. of blood diluted 100 times ; (D) a closed tube with a solution of picro-carmine glycerine, the colour of which corresponds to that of normal blood diluted 100 times ; (F) a guarded lancet. Place a few drops of distilled water in the graduated tube ; with the pipette suck up 20 cmm. of blood, and blow this into the tube, mixing to prevent coagulation. To ensure the removal of all the blood from the pipette refill it with water several times, and blow into the tube. Hold the tubes together against the light or a sheet of paper, and add water from the

[1] The Tallqvist scale can be obtained from Messrs. Allen and Hanbury, Wigmore Street, London, the authorised agents for England.

dropping-bottle until the colours of both tubes correspond. Good daylight is required for this test. Note the number at the surface-level of mixture. This gives the percentage of hæmoglobin in the examined blood compared to the normal. The amount of hæmoglobin may also be estimated by Haldane's hæmoglobinometer, Von Fleischl's hæmometer, or by Oliver's hæmoglobinometer.

Significance of Diminution or Increase of Hæmoglobin.—The amount of hæmoglobin is always expressed in terms of a percentage of the normal standard. Thus, 87 indicates that the amount is 87 as compared with the normal of 100. A diminution of hæmoglobin is the essential feature of all anæmias, but the various forms of anæmia differ as regards the number of blood-cells. Thus, in chlorosis there is a marked diminution of hæmoglobin in each corpuscle, though their number may not be much diminished. On the other hand, in pernicious anæmia the diminution of hæmoglobin is due to the marked diminution in the number of red cells, each of which contains the normal or above the normal hæmoglobin value. The amount of hæmoglobin in each corpuscle is expressed by making a fraction, the numerator of which is the percentage of hæmoglobin present (as estimated by the hæmoglobinometer) and the denominator of which is the percentage of corpuscles (as estimated by the hæmocytometer). For example, if the examined blood has 40 per cent. hæmoglobin and 80 per cent. red corpuscles, the value of hæmoglobin in each corpuscle is $\frac{40}{80}$ths normal. This fraction expresses the *colour index* of the blood, or ratio between the percentage of hæmoglobin and the percentage of red corpuscles.

§ **397. Blood Counts.**—An estimation of the number of corpuscles in the blood is in many cases of extreme importance both for the diagnosis and the treatment

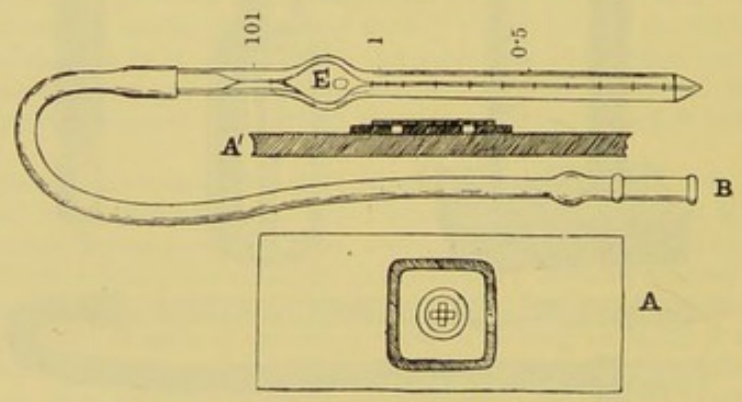


Fig. 113.—The Thoma-Zeiss Hæmocytometer.

of disease. Two instruments are in use for this purpose, the Thoma-Zeiss and Gowers' hæmocytometers. The former is more convenient, requiring a smaller quantity of blood, and having a smaller percentage of error. We shall consider first the method of estimation of the number of red cells in a cubic millimetre of blood, by means of the *Thoma-Zeiss hæmocytometer*. The apparatus consists of a mixing pipette B, a graduated counting slide A (Fig. 113), and a diluting fluid consisting of sodium sulphate in solution, of specific gravity 1025; or sodium sulphate gr. 104, acetic acid ℥i., aq. destill. ℥vi. Before starting see that all the instruments are clean and at hand. Suck up the blood from the drop to mark 0·5 (or 1·0) into the capillary pipette; if any of the blood reach the mixing chamber E the instrument must be cleansed, and the process started again, as it is necessary to be very precise in this measurement. Wipe the end of the pipette, plunge it into the diluting fluid, and suck it up to the mark 101. Holding the pipette between the finger and thumb, rotate and shake it so as to thoroughly mix the fluids in the mixing chamber; the glass ball in E greatly facilitates this process. Next blow out and discard the clear fluid from the capillary end of the tube, and also three or four drops of diluted blood; then let a fraction of a drop fall upon the graduated platform in the centre of the slide. The drop must be of such a size that

when the cover-glass is applied the blood will not run over the edge into the trench around. Place the cover-glass on the drop of blood, and if the cover-glass had been properly cleansed it lies so closely on the outer rim that Newton's concentric colour rings can be observed on the cover-glass. Set the slide aside for a few minutes to allow the corpuscles to settle.

The platform is ruled by cross lines, each enclosing a space of $\frac{1}{400}$ of a square millimetre; the depth of each square with the cover-glass on is $\frac{1}{10}$ of a millimetre. The squares are marked out into sets of 16 by double lines. Count the red cells in three of such sets—that is, in 48 squares. If the corpuscles lie *upon* the lines count those on the upper and left side lines only. Having counted 48 squares divide the total by 48 to get the average per square. The dimensions of a square are $\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000}$ of a cmm. If the blood was drawn up to mark 0·5 it was diluted 200 times. Let N equal the average number of corpuscles per square, then the number of corpuscles in a cmm. of undiluted blood will $= N \times 4{,}000 \times 200$. If the blood was drawn up to mark I it was diluted 100 times, and the last number will be 100 instead of 200. Example: Supposing that after counting 48 squares the average is found to be 6 corpuscles per square, the number of corpuscles in 1 cmm. will then $= 6 \times 4{,}000 \times 200 = 4{,}800{,}000$, which is about the average for a healthy person.

Significance of Diminution or Increase of Red Cells.—In health the average number of red cells per cmm. is about 5,000,000 in the male and 4,500,000 in the female. It is diminished with menstruation, childbirth, and by drinking much fluid. It may be increased to 7,000,000 or 8,000,000 in newly-born children, in plethoric persons, after sweating and fasting, and in those living at high altitudes. *In disease* the number is diminished slightly in all forms of secondary anæmia, though it may be normal in chlorosis. Great diminution in number—*i.e.*, under 3,000,000—occurs after severe hæmorrhages, acute septicæmia, malaria, and severe secondary anæmia. In leukæmia there may be only 2,000,000 per cmm., and in pernicious anæmia they may be as low as 500,000. In disease there is very rarely an absolute increase in the number of red cells, but there is a relative increase when a large quantity of fluid is lost from the body, owing to the concentration of the blood—*e.g.*, in the algide stage of Asiatic cholera (in which the number may reach 6,500,000 per cmm.), after the removal of a large quantity of ascitic fluid, and after persistent vomiting. In cyanosis, erythræmia, and phosphorus poisoning also there is an apparent increase in the number of red cells. In congenital heart disease the number may be as large as 10,000,000 per cmm.

The NUMBER OF LEUCOCYTES per cmm. and the proportion of white to red will next be considered. To enumerate the leucocytes the Thoma-Zeiss instrument is most convenient, and a special pipette is provided which gives a dilution of 1 in 20 (if the blood be drawn up to mark 0·5); or 1 in 10 (if up to mark 1). The leucocyte pipette is recognised by its having a mark II at the top of the mixing chamber, and the diluting fluid is sucked up to this mark. As a diluting fluid simple normal saline solution may be used, or a 0·3 per cent. solution of acetic acid coloured by methyl green. The acetic acid dissolves the red cells, and the green stains the nuclei of the white. A still more useful diluting fluid is that of Toison (*vide* § 395), which does not destroy the red cells, but stains, and thus differentiates the leucocytes. The calculation is carried out by counting the number of leucocytes in all the 256 squares. If the blood was diluted twenty times, and forty leucocytes are found, then $\frac{40}{256} \times 4{,}000 \times 20 = 9{,}200$ per cmm., which is a high normal.

Significance of Increase or Diminution of the Leucocytes.—In health the normal number of leucocytes is 7,000 per cmm. (*i.e.*, 1 white to 700 red cells). On a normal field, with $\frac{1}{5}$ English objective, and a No. 2 eyepiece, about three or four white cells are generally seen. In the newly-born there may be over 17,000 and in early childhood from 10,000 to 14,000 leucocytes per cmm. During pregnancy, after meals, cold baths and exercise, there is an increase in the number of leucocytes. The polynuclear neutrophil leucocytosis (§ 398) is the most usual form of leucocytosis both in health and disease. *In disease* the number of leucocytes is

INCREASED (leucocytosis) in the following conditions, these being the chief conditions in which leucocytosis is of use for diagnosis or prognosis :

Abscess and suppuration,
Septicæmia,
Pneumonia,
Erysipelas,
Scarlet fever,
Osteomyelitis,
Malignant endocarditis,
Tuberculous meningitis,
} in all of which the leucocytosis is due to increase of the polynuclear neutrophil cells.

In spleno-medullary leukæmia there is an increase of myelocytes, mast-cells and eosinophils,
In lymphatic leukæmia and chloroma an increase of lymphocytes,
In anæmia splenica infantum an increase of myelocytes and polynuclear cells,
In some skin diseases and in parasitic diseases an increase of eosinophil cells, and
In lymphosarcoma a slight increase of lymphocytes.

A DIMINUTION of the number of leucocytes (leucopenia) is rare ; it occurs in splenic anæmia, enteric fever, measles, and kala-azar.

Staining and *blood-films are necessary* for the estimation of the number of *different kinds* of leucocytes (the differential count), and this will now be described.

§ 398. Microscopic Examination of Blood and Blood Films.—Alterations in the shape and size of the blood-cells may be seen by examination of fresh blood-films, but for accurate examination of the structure of the red and white cells and a differential count of the leucocytes it is essential that blood-films be fixed and stained. Blood may be obtained from the lobe of the ear by the method above described (§ 395). A microscopic examination of fresh blood may be made by applying a clean slide lightly to the drop of blood, placing a cover-glass on it, and examining under the microscope. For this method any good microscope will do with a $\frac{1}{5}$ or a $\frac{1}{6}$-inch English objective (or a Zeiss's D) and a No. 2 eyepiece ; but for the differential examination of leucocytes and for bacteria a $\frac{1}{12}$-inch oil immersion lens is necessary. It is a great advantage to have a nose-piece on the microscope capable of carrying two or three objectives, so that one can first examine the specimen with a low, and then with a high power. It is well to make oneself familiar with the changes the blood undergoes in a short time after such a method of preparation. If it be desired to preserve such a specimen for some hours, ring the edge of the cover-glass with vaseline to prevent the entrance of air. In this simple way we are able to note any abnormality in the shape of the red cells, or the presence of abnormal constituents, such as particles of pigment, filaria sanguinis hominis, or the spirillum of relapsing fever. We may also note any excess of white cells, for in an ordinary $\frac{1}{4}$-inch field only two to four white cells are generally visible. Rouleaux formation is also noted in normal fresh blood—*i.e.*, the red cells run together, leaving clear the concave spaces in which blood platelets are seen. The white corpuscles are spherical, clear, and nucleated.

A film may be made upon a cover-glass or a slide ; for ordinary purposes the slide method is the easier. It is essential that the slides or cover-glass be absolutely clean and free from greasiness. Lay the surface of the slide lightly on the drop of blood exuding from the lobe of the ear, and with the smooth edge of another slide, or with a morsel of tissue paper spread out the blood in a *thin* film by pushing the drop along the surface. Care must be taken not to handle the slides too much or to breathe upon them. For those who have the necessary manipulative skill the cover-glass method gives the best results. Two cover-glasses, preferably of oblong shape, are required ; place one upon a piece of white blotting-paper ; hold the second in the forceps, and touch the summit of the drop of blood with the half of the slide farthest from the operator, so that a little blood adheres to the glass. Lay cover-glass No. 1 on cover-glass No. 2 so that their ends overlap by half of their length. Let the blood spread out evenly between the two into a thin layer; take each cover-glass edgewise between the thumb and forefinger of each hand, and slide

them quickly apart in their long axes. If successfully done each glass is covered with a *thin*, uniform layer of blood, which quickly dries, and can be taken away for fixing and staining on the same or the subsequent two or three days.

To *fix a blood-film* is a necessary preliminary to staining and mounting. If not performed correctly the corpuscles will shrivel up or break, and will not take the stains properly. Fixing may be performed in several ways : (i.) By immersing the film in a mixture of absolute alcohol and ether for a quarter of an hour and allowing to dry ; (ii.) by immersing in a saturated solution of corrosive sublimate for one to three minutes, and then washing very thoroughly in water ; or (iii.) fixing by the aid of heat. This last method is rarely employed except when it is desired to stain the film subsequently with the Ehrlich-Biondi stain. Experienced workers obtain good results by passing the film rapidly through the Bunsen flame twenty times, but this plan cannot be recommended to the novice. Another method is by placing the slide or cover-glasses on the centre of a copper plate, one end of which is heated by a Bunsen burner. The heat is regulated by placing a drop of water at the other end ; when it steams the plate is too hot.

In the choice of *stains* we employ different dyes according to whether we wish to stain the protoplasm, or the granules of the cells, or the nuclei ; the resulting differentiation of the structure and shape of the cells is of extreme importance in the diagnosis of disease. It is usual to stain first with eosin, then with a nuclear stain such as methylene blue. Stains are acid or basic. The common acid stains are eosin, aurantia, and acid fuchsin ; the basic stains are methylene blue, hæmatoxylin, and gentian violet. The protoplasm of the red cells takes up acid dyes only ; normal nuclei take up basic stains. The granules met with in the protoplasm of the various leucocytes take up different stains ; some have an affinity for acid stains such as eosin, and are known as oxyphil or eosinophil granules ; some take up basic stains such as methylene blue, and are called basophil granules. The granules occurring in the ordinary polynuclear leucocyte were at one time supposed to take up both acid and basic granules, and hence were named neutrophil granules. It is now known that these granules take up faintly acid stains, though the cell is still named polynuclear neutrophil for purposes of description and differentiation. The three methods of staining which were in common use—viz. the eosin and methylene blue stain, the eosin and hæmatoxylin stain, and the Ehrlich-Biondi or triple stain—have been now largely superseded by Leishman's method. Filter every stain before using.

Leishman's Stain.—Place the slide (film uppermost upon a horizontal table, filter enough stain on the film to cover it evenly, and leave for one minute, covering with a watchglass to prevent evaporation or precipitation of the stain. Pour on distilled water, drop by drop, in quantity equal to the stain, leave for another five minutes, and then wash thoroughly with distilled water ; blot dry, and examine. The red corpuscles are stained bright pink, the nuclei dark reddish purple.

Eosin and Methylene Blue Method.—Place the film in a solution of eosin ($\frac{1}{2}$ per cent. in 60 per cent. alcohol) for two minutes ; wash by holding the glass under a gentle stream of water running from a tap ; dry by blotting-paper, and then pass rapidly once through a Bunsen flame, taking care that the film does not become hot ; place in a solution of methylene blue for one or two minutes ; wash and dry as before ; mount in Canada balsam. The red corpuscles are stained pink, the nuclei of the leucocytes blue, the granules of the polynuclear cells show as small faint pink granules ; the eosinophil cells show coarse red granules, degenerate red cells take up some of the basic (blue) stain also. The protoplasm of the leucocytes usually remains unstained, but in some of the mononuclear cells fine blue granules are seen at the periphery of the cell ; in others the protoplasm is diffusely stained a pale blue without granules. The mast cells show coarse granules of a violet blue. For the *eosin and hæmatoxylin* method the same steps are taken as in the eosin and methylene blue method ; Delafield's hæmatoxylin being used instead of the latter (Coloured Plate III.).

The *Ehrlich-Biondi stain* requires films which have been *fixed by heat.* For accurate results by heat-fixing a small copper oven, from which a thermometer

projects, is almost indispensable. The cover-glasses or slides are wrapped in paper and placed inside the oven ; a Bunsen burner is placed 10 to 12 inches below. The films are then baked one to two hours at a temperature of 120° C., or ten to fifteen minutes at a temperature of 130° C. It is useful for the differentiation of the leucocytes and the detection of degenerative changes in the red cells. Stain from one to six minutes, wash thoroughly, dry and mount. If the film has been properly prepared and heated the red cells appear bright orange yellow ; if underheated they look brown ; if overheated, pale yellow. The neutrophil granules show a pink or pale violet hue ; the eosinophil granules are copper ; the nuclei of the leucocytes are a greenish blue ; the nuclei of the nucleated red cells green or black. As the triple stain is not a basic stain none of the mononuclear cells show granules.

Variations of the Red Blood Corpuscles in Disease may consist of (1) variability in form (poikilocytosis) ; (2) variability in size ; (3) nucleation. Normal red cells are circular, bi-concave, non-nucleated discs measuring in size 6 to 7 μ, or $\frac{1}{3200}$ of an inch.

(1) *Poikilocytosis* (Fig. 114 and Plate III.) is a variability in the shape of the red cells. They may resemble a flask, a pear, or a kidney. This change used to be regarded as a pathognomonic of that serious disease pernicious anæmia ; but the change is also found in leukæmia, splenic anæmia of children, and in severe secondary anæmia such as occurs in cancer, nephritis, and malaria. With variation in shape the red cells in any form of profound anæmia appear to undergo degenerative changes—clear hyaline spaces (vacuolation) are seen inside the corpuscles when the specimen is examined just after removal from the body. It must be remembered, however, that such spaces as these may also be seen in normal blood about an hour after it leaves the body. Other more definite changes are seen in the staining of the degenerate cells, for whereas normal red cells take up only acid stains, these degenerate cells take up both acid and basic stains, and their substance stains irregularly. This property is known as polychromatophilia.

Fig. 114.—Poikilocytosis.

(2) *Variability in Size.*—The normal red corpuscle measures about 7 μ ; red cells measuring 3 to 5 μ are termed microcytes, and those measuring 9 to 12 μ macrocytes. Both variations are met with in pernicious anæmia, leukæmia, the splenic anæmia of children, and in severe secondary anæmia. They are also seen in severe cases of chlorosis.

(3) *Nucleation*, if present, is made more evident on diluting a drop of fresh blood with a drop of $\frac{3}{4}$ per cent. saline solution coloured with methyl violet. It is also well brought out by the triple stain (*vide supra*, and Plate III.). Nucleated red cells are found in all cases where there is a great diminution in the number of the red corpuscles—as, for example, in leukæmia, pernicious anæmia and severe secondary anæmia. They are very rare in chlorosis, but have been seen. These nucleated red cells must be distinguished from lymphocytes, which resemble them approximately in size. The nucleated red cells differ in having a wider rim of protoplasm round the nucleus and the more homogeneous staining of the nucleus. The nuclei may show karyokinesis, or may be degenerate. There are three distinct forms of nucleated red corpuscles : the normoblast, about the same size as a normal red cell ; the large form or megaloblast, which is about three to five times the size of an ordinary red cell ; and the microblast (or immature red cell), which is smaller than the normal red cell. The presence of the megaloblast in the blood is generally of grave import, indicating serious need for rapid regenerative changes in the blood. These cells are usually polychromatophilic (*vide supra*). They occur in large numbers in pernicious anæmia.

In *Primary Anæmias* (p. 573 below) the essential blood change is a diminution of hæmoglobin, accompanied in the majority of instances by a greater or less diminution in the number of red cells, and various degenerative and regenerative changes.

In *Secondary Anæmia* Dr. Cabot[1] describes four stages in the blood alteration. These stages hold good except in anæmia after malaria, hæmorrhages, and acute septicæmia, when the third may precede the second stage. (1) Diminution of the hæmoglobin and lowered specific gravity, without diminution in the number of red cells; (2) diminution in the hæmoglobin, loss of rouleaux formation, slight diminution in the number of red cells, with degenerative changes in their shape and size—poikilocytosis, microcytes and macrocytes, vacuolation and polychromatophilia; (3) diminution of the hæmoglobin and the number of red cells (under 3,000,000); (4) in addition to the above there are regenerative changes—microblasts, normoblasts, and megaloblasts. It will be observed that differences between primary and secondary anæmias are not at the present time sufficiently characteristic for differentiation by the microscope, but the prognosis and treatment may depend upon the result of microscopic examination.

Variations in the Leucocytes (Plate III., Fig. 1) may occur in regard to their absolute number (blood count, *vide supra*), their structure, and the relative number of one kind or another (differential count). There are several kinds of leucocytes, and it is possible to identify the cause of an increase in the leucocytes by the predominating variety present. For this purpose and in order to make a differential count to ascertain the relative proportion of the several varieties, it is necessary to employ the staining methods given above (*e.g.*, eosin and methylene blue, or Leishman's stain.

The varieties of leucocytes found *in healthy* are as follows:

In 100 leucocytes:	
Polynuclear	60 to 70
Small mononuclear leucocytes (lymphocytes) ..	30 to 20
Large mononuclear leucocytes	5
Transitional forms	3
Eosinophil cells	2
	100

(1) In the *Polynuclear Neutrophil Leucocytes*, which form 60 to 75 per cent. of all leucocytes in the blood, and have an average diameter of 13·5 μ, the nucleus is long and lobed, giving the appearance of being multipartite, and the protoplasm of the cell contains fine neutrophil granules. The so-called neutrophil granules are really faintly acid; with the Ehrlich-Biondi stain they show fine reddish or lilac granules. This cell originates from the spleen and bone-marrow, and is actively amœboid and phagocytic (microbe devouring). (2) *Lymphocytes*, 20 to 30 per cent., are small leucocytes, without granules, with one large nucleus and very small amount of surrounding protoplasm: coming chiefly from the lymphatic glands and adenoid tissue. They are neither amœboid nor phagocytic. (3) *Large mononuclear* or "hyaline" cells, 4 to 8 per cent., have one large spherical nucleus and a larger amount of protoplasm than the lymphocytes. With the Ehrlich-Biondi stain these cells show no granules, but with a basic stain, such as methylene blue, some of them show a few fine blue granules. These cells are supposed to come from the bone-marrow, and are slightly amœboid. (4) *Eosinophil polynuclear* leucocytes, with coarse, eosin-staining granules, 2 to 4 per cent., coming from the bone-marrow, are slightly amœboid, but not phagocytic. (5) *Basophil polynuclear* leucocytes, or mast cells, $\frac{1}{2}$ per cent., some with fine, some with coarse basophil granules seen by prolonged staining with a basic stain. (6) *Transitional forms* between 3 and 1 above are found. Their nuclei are faintly stained, and of a horse-shoe shape.

Variations of Leucocytes in Disease.—Variations in the number of the leucocytes in health have been referred to (p. 561), and a list of causes of simple increase in number of leucocytes has been given. Polynuclear neutrophil leucocytosis is the commonest form of increase of the white cells. When leucocytosis is present a differential count is desirable; the disease may often be diagnosed by the predominant variety present. Several hundred leucocytes must be counted, the

[1] "Guide to the Examination of the Blood," by R. Cabot, 1898.

number of each variety noted on a piece of paper, and the percentage of each calculated. In many cases it may be necessary to make this count daily—*e.g.*, to watch the steady daily increase of polynuclear neutrophil cells, which may denote a perityphlitic or other obscure abscess within the body. Except in leukæmia, leucocytosis rarely passes beyond 100,000 per cmm. (normal about 9,000 per cmm.).

(1) The *Polynuclear Neutrophil* cells constitute, as just mentioned, the majority of the white cells in the blood. They are greatly increased in infective diseases, such as pneumonia, septicæmia, erysipelas, scarlet fever, and in local inflammations or abscesses such as appendicitis and osteomyelitis. So true is this that an unfavourable prognosis in croupous pneumonia can be based upon a small degree of leucocytosis. In typhoid fever the onset of a suppurative complication may be diagnosed by the presence of an increased number of polynuclear neutrophil leucocytes. A diagnosis may be made between typhoid fever and tuberculous meningitis from the fact that in meningitis there is leucocytosis, but there is none in typhoid fever uncomplicated by abscesses. Again, so long as there is leucocytosis present in convalescence from any acute inflammation a patient should be kept in bed. If leucocytosis be found, even without definite physical signs pointing to an inflammatory condition, the onset of inflammation can almost with certainty be predicted. In this way blood examination comes to be of the highest importance in the diagnosis of obscure abdominal cases, as a polynuclear increase is rightly regarded in such cases as an indication for operation. The blood signs of deep-seated suppuration are increasing leucocytosis, with a high percentage of polynuclear leucocytes, increase of blood platelets, increase in the fibrin, and glycogenic degeneration of white cells—*i.e.*, staining with iodine. In cancer of the stomach it has been found that there is often no increase of leucocytes one hour after meals, as would occur in health or in simple ulcer of the stomach. In all severe stomach trouble leucocytosis after digestion is diminished or absent. After profuse hæmorrhages, in malignant cachexia, gout and chronic renal disease leucocytosis is present. The subcutaneous injection of irritants also produces leucocytosis.

(2) The *increase of eosinophil* cells occurs in one form of leukæmia (see below), in several skin diseases, notably pemphigus and psoriasis. In asthma they may be increased to 25 per cent. or more, and this may be an aid to its diagnosis from cardiac disease and mediastinal tumour. Local accumulations of eosinophils also occur in the bronchial secretion of asthma and around cancer growths. It is found also in those suffering from trichinosis and ankylostomiasis; indeed, in all parasitic diseases (*vide infra*).

(3) *Lymphocytosis* (increase of lymphocytes) occurs in children suffering from gastro-intestinal disorders, rickets, and syphilis, and lymphatic leukæmia. In adults it occurs in lymphatic leukæmia (90 per cent.), and less markedly after some infective diseases and secondary syphilis. The diagnosis of Hodgkin's disease from lymphatic leukæmia may depend upon the examination of the blood. In the former there may be no leucocytosis, or if present there is an increase mainly of the polynuclear leucocytes; in the latter the lymphocytes are greatly increased.

(4) *Other forms of leucocytes* which make their appearance in disease are (i.) myelocytes, very large cells, mononuclear, some with fine neutrophil, some with coarse eosinophil granules, coming from the bone-marrow. They are not amœboid or phagocytic. Myelocytes occur in the spleno-myelogenous form of leukæmia in large numbers (30 per cent.), and in anæmia splenica infantum. They occur in smaller numbers in bone-marrow sarcoma and severe anæmia after hæmorrhage. (ii.) Pseudo-lymphocytes of various sizes, with fine neutrophil granules, may be seen in similar cases, but are rare.

Melanæmia is a term applied to certain pigment granules in the blood which occur after ague, relapsing fever, and some melanotic tumours. They appear either in minute black lumps, or are enclosed within the cells. They are probably disintegrated blood pigment.

Blood Platelets.—As yet no clinical importance has been attached to the blood platelets. They are frequently not observed in the ordinary methods of blood examination. In order to find them a cover-glass must be placed on a slide, with

the edges of both corresponding. Place the edges near the forming drop of blood, which by capillary attraction will immediately spread out into a film. Stain with equal parts of normal saline solution and glycerine saturated with dahlia. The blood platelets are seen with a $\frac{1}{12}$-inch oil immersion lens as irregular bodies, small, apt to run together in clumps. They number about 300,000 per cmm. These bodies are increased in leukæmia, grave anæmias, and pyogenic conditions, and diminished in purpura, hæmophilia, typhus, erysipelas, and malaria.

Blood Dust is a term which has been given to a number of small clear bodies in the blood, with a vibratile motion, about $\frac{1}{2}$ to μ in size. They are supposed to be granules extruded from neutrophil and eosinophil cells.

§ 399. Parasites found in the Blood.—The MICRO-ORGANISMS which can be detected in the blood (such as glanders, anthrax, septic microbes, the spirillum of relapsing fever, the typhoid and influenza bacillus), and their methods of detection, as far as it is applicable to clinical work, are referred to in Chapter XX.

The chief PARASITES which have been found in the blood are the filaria sanguinis hominis, distoma hæmatobium, the malaria parasite, the protozoa of kala-azar, and the trypanosoma.

The PARASITE OF MALARIAL FEVER is a protozoon, inhabiting the red corpuscles, which it destroys, but it does not invade other tissues. There are three well-marked varieties of the parasite, distinguished from each other by their intracorpuscular development, and these varieties correspond to the three types of malaria known as benign tertian, quartan, and malignant tertian fever. The life-history of the protozoon runs through two stages: (i.) The asexual or intracorpuscular stage in man; and (ii.) the sexual form within the body of a mosquito belonging to the genus anopheles (Fig. 102, p. 529). The tertian parasite, which gives rise to the *benign* form of malaria, is the least virulent (Fig. 115). It is first seen within the corpuscle as a small, clear, ovoid body about 2 μ in diameter, possessing active amœboid movement. It gradually increases in size, and after the lapse of a few hours becomes ring shaped, with very finely granulated pigment collecting about its centre. In the benign tertian form there is at the same time marked enlargement of the corpuscle, with a striking decrease in its colour. At the stage of full growth the parasite occupies nearly the whole of the enlarged corpuscle, and now it may follow either of two lines of development: (i.) The pigment gathers as a solid mass in the centre of the parasite, the protoplasm divides by a radial arrangement into well-marked rosettes of from fifteen to twenty segments; these disintegrate as spores, and masses of insoluble pigment enter the blood stream. This phase is known as " segmentation " ; it is complete in about forty-eight hours, and corresponds clinically to a fresh paroxysm of the fever. The intracorpuscular development just described is asexual. (ii.) The sexual development takes place in the female mosquito, which sucks the blood of a malarial patient, and takes into its stomach the protozoon in the phases above mentioned. All die except the male and female gametocyte (which are crescentic in the malignant tertian type), the female being distinguished by the pigment round the nucleus. The male gametocyte develops within the mosquito into a flagellated body, and one of the

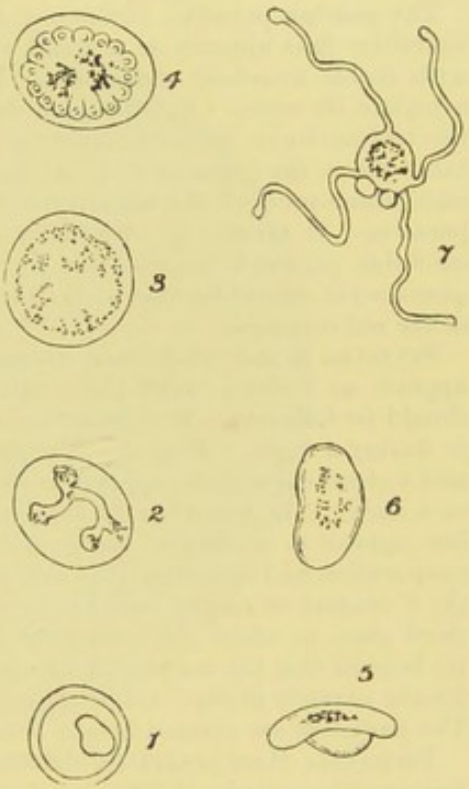


Fig. 115.—PARASITE OF MALARIAL FEVER. 1 to 4 = stages in benign tertian; 5 = crescent body; 6 = oval body; 7 = flagellated body.

flagella unites with the female gametocyte to form an oval-shaped body with pointed ends. This projects from the stomach wall of the mosquito into its body cavity, and becoming spherical it subsequently divides into numerous cells, and these again into curved, needle-shaped bodies. These are carried by means of the salivary gland of the mosquito into the blood of the person bitten, and so into his red blood-corpuscles.

The parasite of the *malignant tertian* fever is first seen in the red blood-cells as a tiny, unpigmented, hyaline body, forty-eight hours being needed for its development. At first it exhibits energetic amœboid movements, but ultimately settles into a bright, colourless, ring-like form, with one or two pigment granules contained therein. There is frequently multiple infection of the red corpuscles, which at first enlarge, then shrink, and become a brassy hue. The rosette or sporulating stage is rarely seen. In about a week (during the period of remission) characteristic crescent bodies, containing masses of coarse pigment granules, begin to appear, and increase in number rapidly. They are incapable of sporulation, and represent the sexual form—the gametocyte.

The *quartan* parasite, the easiest form for the beginner to study because of its visibility, first appears as a small, round, clear speck, resembling a vacuole, but with feeble amœboid movement. It takes from sixty to seventy-two hours to complete its cycle. By the third day pigment, coarser and blacker than that of the tertian form, gathers round its periphery. On the fourth day segmentation takes place, the pigment flows in towards the centre, and here forms the radiating lines which produce the beautiful "daisy rosette" so characteristic of the quartan parasite. It breaks up eventually into eight to ten spores, and these with the insoluble pigment become free in the blood stream. The development of the gametocyte resembles that of the benign tertian variety. There is no enlargement of the red corpuscle.

Staining is not absolutely necessary for the detection of the parasite, which appears as a clear ovoid body within the red cell. Sir Patrick Manson's rules should be followed. It is best to examine the blood unstained at first, just before or during a rigor. *Method.*—The first drop or two of blood must be wiped away, and a clean cover-glass applied to the summit of the next drop, then lightly placed on a slide. The blood should then show areas with several zones; if these zones do not appear it is simply waste of time to examine the specimen. To prevent evaporation and crenation it is well to ring the glass with vaseline. The zones are: (1) A central or empty zone; (2) a zone of isolated or scattered corpuscles; (3) a third zone, in which the corpuscles lie closely together, the "single layer" zone; (4) beyond this the corpuscles lie upon each other, the "heaped up" zone. Look for the parasite in the "single layer" zone. To stain, employ Leishman's method. The parasites are stained bright blue, with nearly black granules.

Bilharzia Hæmatobia (or distoma hæmatobium) is a trematode giving rise to recurrent hæmaturia (§ 300), inhabiting the blood of most of the Fellah and Coptic population in Egypt. The ova are extremely plentiful in the urine, and are very characteristic; they measure 12 μ long by 4 μ broad, are spiked at one end (Fig. 89, § 300), and occur in the blood and in some of the organs; the parasite itself is found in the portal system, especially in the venous plexuses about the rectum and bladder.

Filaria Sanguinis Hominis is a parasite which occasionally produces elephantiasis, chyluria, etc. The method of revealing the embryo is to allow a thick drop of blood spread upon a slide to dry. Stain half a minute in a 2 per cent. solution of methyl blue. Decolorise if necessary in dilute acetic acid (4 drops in 1 ounce of water), and examine with low power (see Fig. 116). Six species of filaria are known to infect man.

(1) The *Filaria Bancrofti* vel *Filaria Nocturna* very rarely causes any pathological symptoms in the embryonic state. The embryos may be found in 20 per cent. of apparently healthy residents in Barbadoes and other tropical climates. The embryo comes into the peripheral blood at night (from 6 p.m. to 10 a.m.); their maximum number is usually found about midnight. It may be necessary to make repeated examinations at intervals of two hours to be able to find them.

During the day the filaria go into the lung bloodvessels where they are usually found in autopsies of people who had them, and died in the daytime. Should a victim of the parasite alter his usual habits, and sleep during the day, the filaria periodicity is reversed. The adult filaria inhabit the lymphatics, where they give birth to immense numbers of embryos, a large number of which must in some unknown way disappear, or else the blood would contain them in incalculable numbers. Embryos show a very active movement, but this is within their sheath, so that there is no locomotion properly speaking. It is this motion of their sheaths which keeps the blood corpuscles moving away from the parasite. Sometimes these embryos are actually seen escaping from their sheath, and then they become locomotive. The parasite is about $\frac{1}{80}$ of an inch in length by $\frac{1}{3000}$ of an inch in diameter. The sac prevents the creature from piercing the walls of the blood-vessels, and thus gives it the chance of gaining access to the body of a mosquito, where it undergoes a metamorphosis, resulting in the formation of a mouth, alimentary system, and a trilobed tail. It grows until it becomes $\frac{1}{16}$ of an inch in

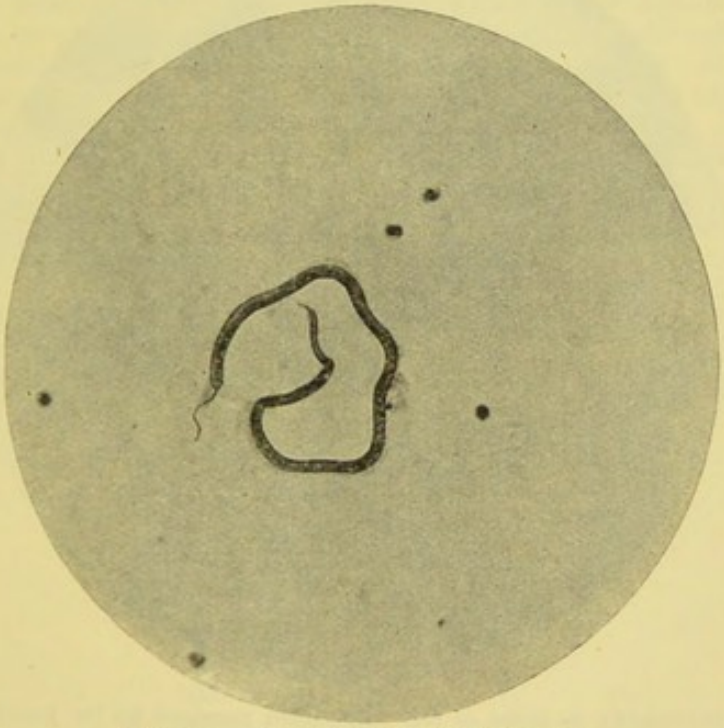


Fig. 116.—Filaria Sanguinis Hominis.

length, re-enters man, and grows to the mature stage of its existence. Adult parasites after their death cause well-marked symptoms—viz., various forms of elephantiasis, lymphscrotum, hæmatochyluria, chylous diarrhœa, and ascites, usually related to their blocking of the lymphatic circulation.

The other species are : (2) *Filaria Perstans ;* (3) *Filaria Demarquaii ;* (4) *Filaria Ozzardi ;* and (5) *Filaria Loa* (probably the adult form of Filaria Diurna), which are found in the connective tissue, subcutaneous or subperitoneal, and are not known to give rise to symptoms. (6) *Filaria Diurna* is of the same dimensions and anatomical characters as filaria nocturna, but it is found in the blood of patients during the day, and not in the night.

Trypanosoma.—The parasite of *trypanosomiasis* (a disease of West Africa characterised by irregular pyrexia, erythematous rash, and enlargement of the liver and spleen) and of *sleeping sickness* (a disease indigenous to tropical Africa) is a flagellated protozoon (Fig. 117). It is a minute worm-like organism found free in the blood, moving with a screw-like progress. One end of the parasite is drawn out into a whip-like process, the flagellum ; the other end is bluntly conical ;

the body itself is short and thick, and its substance granular. Attached to one side is a transparent, flange-like process, the undulating membrane. At its posterior end is a highly refractile spot, the vacuole. The length of the parasite, including the flagellum, is about 18 μ to 25 μ. It is best stained by Leishman's or Romanowsky's stain. In sleeping sickness the organism is found chiefly in the lymphatic glands; it causes a polyadenitis, which accounts for the glandular enlargements which invariably accompany the disease.[1] When the trypanosoma gains access to the cerebro-spinal fluid (although it may have previously existed for years in the blood) the patient develops *sleeping sickness*. There are three stages in this disease: (1) Hectic fever and lassitude; the trypanosomes are present in the spinal fluid. (2) Tremors of the tongue, lips, hands, and legs occur; the intellect becomes more dull, the face is puffy, and the gait shuffling. (3) Drowsi-

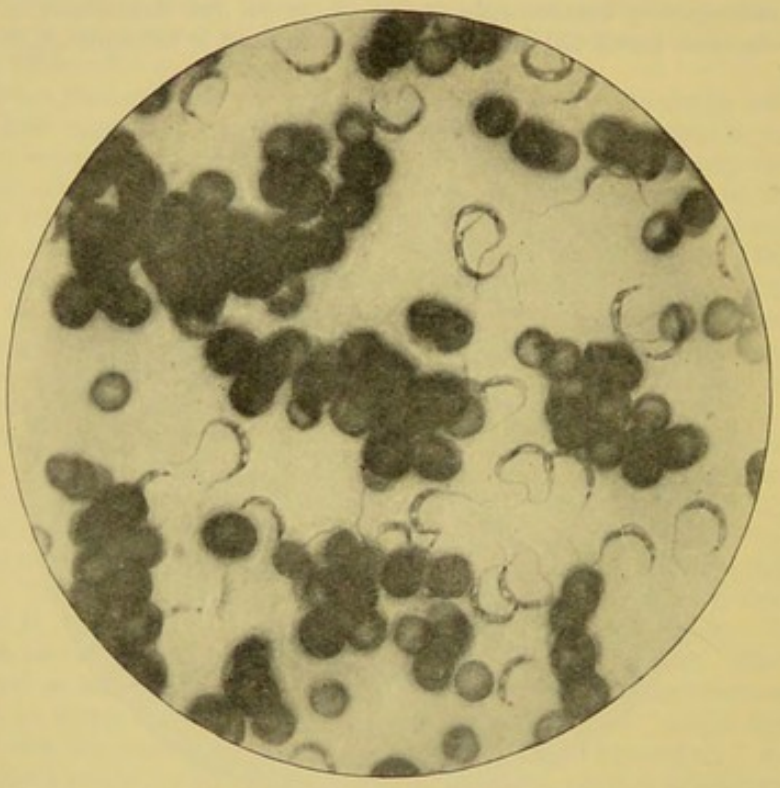


Fig. 117.—TRYPANOSOMA in blood of rat. Specimen prepared by Dr. Emilio Eccheverria photomicrograph by Mr. Fred. Clark.

ness, increasing to profound lethargy and subnormal temperature, ending in death. The disease is transmitted by the tsetse fly.

The protozoa of KALA-AZAR are found in the spleen, liver, bone-marrow, and in the lymphatic glands from the mesentery. They have been chiefly studied in blood and pulp withdrawn from the spleen, or preferably the liver during life. The commonest form found is a small ovoid body longer than it is broad, below 2 μ in diameter, measuring about one-fifth of a red corpuscle in its longest axis. It contains two nuclei; one is small, rod-shaped, and stains deeply; the other is larger, rounded, and stains less deeply. Other forms met with consist of small groups of similar bodies clumped together, resembling a quartan sporulating malarial parasite; at times these are seen breaking up into the simpler forms. A still earlier stage of this sporulisation is seen, in which pairs of unequal sized nuclei are grouped within a single cell, but with no signs of division apparent.[2] They stain faintly with methylene blue; but the best method of staining them is by

[1] *Brit. Med. Journ.*, 1902, vol. ii., p. 881; 1904, vol. i., p. 1252.
[2] *Brit. Med. Journ.*, vol. i., 1904, p. 1249; 1903, vol. i., p. 1252.

Leishman's or Romanowsky's stain. The same bodies have been found in Delhi boil and Dum-dum fever. They are known as Leishman-Donovan bodies, and are now known to be an early stage of a trypanosoma. Leonard Rogers has succeeded in obtaining trypanosomes by culture of these bodies.

§ 400. Physical and Chemical Properties of the Blood.—The ALKALINITY OF THE BLOOD can be tested by methods which can only be performed in a laboratory.[1] The blood is never acid to litmus, but the degree of its alkalinity varies. It is greater in men than in women and children ; it is diminished after violent exercise and the prolonged use of acids, and it is increased at the beginning of digestion, and after the prolonged use of alkalies. In disease there is diminished alkalinity found with leukæmia, pernicious anæmia, anæmia, diabetes, cancer, great cachexia, poisoning with carbon monoxide and with acids, high fevers, and various toxic processes. In chlorosis it is little if at all diminished.

The normal SPECIFIC GRAVITY of the blood is approximately 1055. It may be estimated by mixing chloroform and benzol till the specific gravity of the mixture reaches 1055, and adding to this mixture a drop of blood from a pipette. If the drop remains without rising to the surface or falling to the bottom the specific gravity of the blood is 1055. If it sinks, continue to add chloroform drop by drop, shaking the mixture the while, until the drop becomes suspended. If it floats add benzol until the drop is suspended. Then take the specific gravity of the mixture, and this is the same as that of the specimen of blood.

Significance of Altered Specific Gravity.—The amount of hæmoglobin can be estimated by finding the specific gravity of the blood, because it has been found that the specific gravity varies in proportion to the amount of hæmoglobin present. The following table[2] is of use :

Sp. Gr.		Hæmoglobin.
1030	=	20 per cent. ±
1035	=	30 ,, ,,
1038	=	35 ,, ,,
1041	=	40 ,, ,,
1042·5	=	45 ,, ,,
1045·5	=	50 ,, ,,
1048	=	55 ,, ,,
1049	=	60 ,, ,,
1051	=	65 ,, ,,
1052	=	70 ,, ,,
1053·5	=	75 ,, ,,
1056	=	80 ,, ,,
1057·5	=	90 ,, ,,
1059	=	100 ,, ,,

This method is of no use in cases of (i.) dropsy, (ii.) leukæmia, where the specific gravity is relatively higher than the hæmoglobin because of the weight of the leucocytes ; and (iii.) pernicious anæmia, where the hæmoglobin is 20 per cent. higher than that gauged by specific gravity because of the high colour index of each corpuscle.

SPECTROSCOPIC EXAMINATION of the blood.—The instrument chiefly used for clinical purposes is Browning's spectroscope. It is used by holding up a glass containing a very dilute solution of blood, and looking through it at the light, or at a white cloud with a spectroscope placed between the blood solution and the eyes. Hæmatoporphyrin (Fig. 118) has been found in cases of sulphonal poisoning. The discovery of methæmoglobin in the blood may be a means of warning the physician of the near onset of coma in diseases such as uræmia or diabetes. Methæmoglobin is formed in amyl nitrite and potassium chlorate poisoning ; and carboxyhæmoglobin in coal-gas poisoning.

[1] Dr. Robert Hutchison ; see the *Lancet*, March 7, 1896.

[2] Cabot, " Clinical Examination of the Blood," 1898. Modified from Schmaltz, " Pathologie des Blutes," Leipsic, 1896.

The Coagulability of the Blood is estimated by Sir A. E. Wright's coagulometer.[1] It has a number of fine tubes, into which blood is drawn at definite intervals, and at varying times the operator blows down the tubes. When the blood cannot be blown out it has coagulated. The coagulation time is thus readily calculated. Coagulation depends upon the presence of lime salts and fibrinogen, which are contained in the blood plasma, and of nucleo-proteid, which is contained in the substance of the leucocytes and blood platelets. The nucleo-proteid is liberated when the leucocytes are disintegrated, and this may occur when infective toxins circulate in the blood, when the bloodvessels are diseased, and in cachectic states, and thus thrombosis may in some cases be explained. Coagulation within the body is also hastened by the addition of certain salts, such as calcium chloride. Coagulation within the body is diminished in certain infective diseases, and in urticaria, prurigo, and allied erythematous conditions, and the administration of

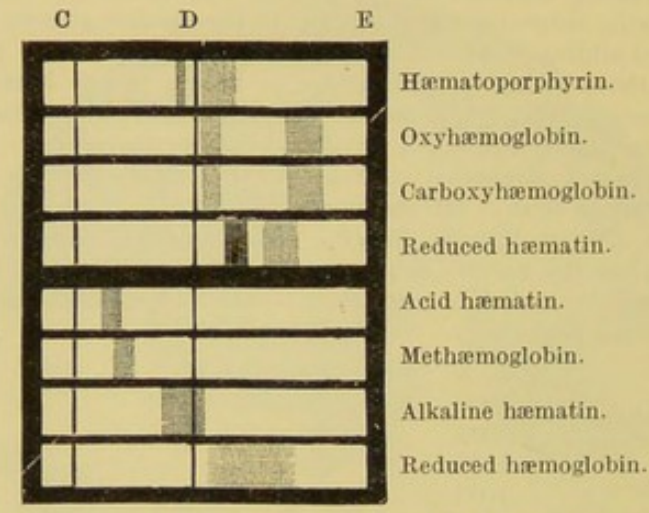


Fig. 118.—Blood Spectrum (from Waller's "Human Physiology").

calcium chloride in 20-grain doses, as the author has shown,[2] is of great use in alleviating prurigo and urticarial conditions.

The Freezing-Point of the Blood is normally 0·56° C. It is lowered to 0·60° C. in cases of renal disease, in which an excess of urea and salts is present, and reaches 0·8° C. in cases of impending uræmia.

Choline is found in the blood in increased quantity when nerve cells degenerate (Halliburton and Mott), and may be found by laboratory methods in 10 c.c. of blood in disseminated sclerosis, myasthenia gravis and transverse myelitis, but not in hysterical affections which are apt to simulate these diseases.

PART C. DISEASES WHICH GIVE RISE TO GENERAL DEBILITY, WITH OR WITHOUT ANÆMIA AND EMACIATION: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **401. Routine Procedure** and **Classification.**—Here, as elsewhere, we have three points to investigate:

First, the Leading, and perhaps the only, Symptom complained of by the patient will be debility, or pallor of the skin, or loss of flesh.

Secondly, the History of the Illness, its date and mode of onset and mode of evolution. Often these data are vague, but special inquiries should be directed to the condition of the digestion in times past, and any other points relating to nutrition.

[1] Hawksley supplies the instrument, with directions.
[2] The *Lancet*, August 1, 1896.

Thirdly, the PHYSICAL EXAMINATION of the patient, commencing with that physiological system to which the results of our previous inquiries have directed attention, and then going through all the systems seriatim. An examination of the blood should be made in all anæmic or doubtful cases—viz., blood counts, hæmoglobin, and films.

Classification.—If anæmia is suspected or PALLOR OF THE SKIN is the leading symptom, turn first to Group I., below.

If LOSS OF FLESH is most prominent, turn to Group II., p. 602.

If GENERAL DEBILITY (without obvious pallor or loss of flesh) is most prominent, turn to Group III., p. 609.

GROUP I. ANÆMIC DISORDERS.

Anæmia is an alteration in the composition of the blood, the leading character of which is a deficiency in the hæmoglobin. The disorders giving rise to **pallor of the skin** are :

COMMONER.

I. Primary anæmia (chlorosis).
II. Pernicious anæmia.

Constitutional Conditions.

III. Constitutional syphilis.
IV. Lead poisoning.
V. Tuberculosis.
VI. Carcinoma.

Visceral Disorders.

VII. Gastro-intestinal conditions.
VIII. Chronic aortic disease.
IX. Chronic renal disease.
X. Chronic hepatic disease.

Recognised by History.

XI. Hæmorrhage, hyperlactation, and other causes of long-continued drain.
XII. Post-febrile anæmia.
XIII. Chronic suppuration, septic processes, and lardaceous disease.

RARER (*in this country*).

With Signs in Spleen and Lymphatic Glands.

I. Leukæmia.
II. Hodgkin's disease, or lymphadenoma.
III. Splenic anæmia.

With Signs referable to Skin.

IV. Scurvy.
V. Hæmophilia.
VI. Addison's disease and other maladies mentioned in Groups II. and III.

With Typical History.

VII. Malaria and other tropical and parasitic diseases.

Anæmias of Infancy.

I. Primary anæmias.
II. Secondary anæmias.
III. Congenital anæmia.
IV. Infantile scurvy.
V. Anæmia splenica infantum.

The first two disorders given in the list are called primary, essential, or idiopathic anæmias, because they are not known to be preceded by any other disease (such as tubercle, malaria, etc.) ; all the others are called secondary anæmias. The division into idiopathic and secondary anæmias is somewhat artificial, and idiophatic anæmia, the anæmia of unknown causation, will probably disappear with the march of science just as idiopathic peritonitis has done. This, like all the other anæmias, is the result of a defect of the hæmogenic or the hæmolytic functions of the body, about which we shall probably know more in the course of a few years.

Among the various members of the list, the pallor may present to the experienced observer a difference in kind and degree in the several affections. Thus, the

greenish-yellow colour of chlorosis, the lemon yellow of pernicious anæmia, the earthy tint of carcinoma, the sallowness of aortic disease and interstitial nephritis, the pasty white of parenchymatous nephritis, and the transparent waxy look of lardaceous disease are very suggestive to the careful observer. The microscopic examination of the blood also reveals differences which are mentioned below. Among the *commoner* anæmic disorders it will be observed that after the two primary anæmias come four constitutional conditions, then four visceral disorders, and finally the three conditions which are readily recognisable by their history. The age of the patient will often give us a valuable clue.

Among the (in this country) *rarer* disorders, leukæmia, lymphadenoma, and splenic anæmia present physical signs referable to the spleen or lymphatic glands and leucocytosis; scurvy, hæmophilia, and Addison's disease present signs referable to the skin; and the remainder reveal a history of tropical disease or parasites.

The blood shows marked **diminution of hæmoglobin** *and other changes, and there is* NO DISCOVERABLE PRIMARY ORGANIC LESION. The disease is probably CHLOROSIS or PERNICIOUS ANÆMIA; the age and sex of the patient and the colour-index of the blood being very different in the two diseases.

§ **402. I. Chlorosis** ("green sickness," "poverty of blood," sometimes spoken of as primary, essential, or idiopathic anæmia) is a chronic malady in which the main feature is a diminution of the colouring matter of the blood, unaccompanied by any gross lesion, not preceded by any primary or causal disease, and occurring mostly in young women.

The chief *Symptoms* are (1) failing strength of vague onset and considerable duration, accompanied by pallor of the surface, but unaccompanied by wasting. There is, indeed, often an excess of adipose tissue. The pallor is marked in the lips, gums, and conjunctivæ (as may be observed by pulling down the lower lid), and the sclerotics have a bluish colour. The skin sometimes presents a greenish hue, hence the name chlorosis (χλορος, green). These patients generally present themselves on account of either cardiac or digestive symptoms, headache, or amenorrhœa. (2) Cardio-vascular symptoms, such as dyspnœa on slight exertion, palpitation, and a tendency to syncope. In a fair proportion of the cases dilatation of the heart can be made out. Hæmic murmurs are heard, especially over the pulmonary area. In marked cases the "bruit de diable" is to be heard—a continuous hum heard when the stethoscope is gently placed over the jugular vein in the neck. Œdema of the ankles at night is common, and venous thrombosis may be met with.

Hæmic or Anæmic Murmurs may be either systolic or double in rhythm, never diastolic alone or presystolic. They are usually soft and blowing, but may be extremely loud and rasping; loudest in the pulmonary area, but they may be heard all over the precordium, very rarely in the axilla; often louder when the patient is lying down or has rested, and apt to vary from day to day. As regards their causation, my own belief tallies with those who hold that they are due partly to increased fluidity of blood, and partly to a flabbiness or enfeeblement of the cardiac wall. The wall, therefore, yields to the intra-ventricular pressure, and the resulting dilatation of the orifices leads to regurgitation.

(3) Disturbances of digestion are generally present, such as deficient or capricious appetite, and discomfort after food; and there may be atonic dyspepsia, gastric atony, and gastroptosis. Prolonged and intractable constipation almost invariably precedes and accompanies the affection, and is said by some to be its principal cause. (4) Symptoms referable to the nervous system, such as headache, neuralgia, vertigo, defective attention, nervousness, irritability or depression of spirits, spots before the eyes. (5) Amenorrhœa is usual, dysmenorrhœa not infrequent; menorrhagia sometimes precedes and accompanies chlorosis. (6) Some authorities say that chlorosis may be attended by elevation of temperature from time to time, but adequate proof in the post-mortem room should be forthcoming that such cases have not been due to some undiscovered syphilitic, tuberculous, or septic lesion (*vide* p. 534, footnote). In ordinary cases the temperature is subnormal. (7) *Blood changes.*—There is usually but little diminution in the number of red corpuscles, though a great diminution in the hæmoglobin which they contain. The red corpuscles appear pale, and the total hæmoglobin in the blood is sometimes as low as 30 per cent. of the normal. In severe cases, however, there is also a considerable diminution (down to 2,000,000) in the number of red corpuscles. Poikilocytosis, leucocytosis and nucleated red cells are uncommon except in severe cases (see § 398); chemical analysis shows a marked diminution of the iron in the blood. There is an excess of the watery constituent of the blood or "hydræmic plethora."

The *Diagnosis* is not, as a rule, difficult, by reason of the age and sex of the patient. The pallor of the skin, though it may have a yellowish-green tint, is usually quite different from that of jaundice or the cachexia of malignant disease. It has at times to be differentiated from that due to any of the conditions mentioned in the succeeding sections below, and especially from gastric ulcer. The chief danger is lest an early stage of chronic tuberculosis should be overlooked; and to avoid this a thorough examination of all the organs should be made, the principal physical signs in chlorosis being the murmurs and the pallor. The pallor of renal disease is an ivory white, and is attended by albuminuria, tube-casts, and in some forms by generalised dropsy. Chlorotic girls may have œdema of the ankles, but there is no albumen in the urine. The diagnosis from organic heart disease is given under Cardiac Murmurs (§ 47).

Prognosis.—The disease is rarely fatal, but it is extremely liable to relapse, and the symptoms are sometimes so grave as to necessitate strict confinement to bed. It is essentially a chronic condition, and its course always extends over many months, unless checked by treatment. Gastric ulcer is a frequent sequel. Thrombosis of the femoral vein may occur, and, occasionally, of the longitudinal sinus. "Spurious hæmoptysis"—*i.e.*, hæmoptysis without any pulmonary

mischief — probably coming from the mouth or throat, occurs occasionally.

Etiology.—The disease is practically confined to young women between the ages of fifteen and twenty-five, who have suffered from chronic constipation, and have not sufficient out-of-door exercise or fresh air. It is a curious and unexplained circumstance that more blondes than brunettes are affected. The disease has at different times been thought to be due to a small aorta, the onset of puberty, chronic constipation, or the wearing of tight corsets. It has been suggested that in constipation the iron in the food does not reach the system, owing to its decomposition in the alimentary canal and its combination with sulphur to form sulphides. The marked benefit derived from respiratory exercises suggests deficient oxygenation.

The *Treatment* of chlorosis consists, first, in correcting any digestive trouble which may be present, or constipation, or intestinal sepsis (*e.g.*, by naphthol or salol); secondly, in the administration of iron; and thirdly, in adopting hygienic measures, especially such as promote oxygenation. It is well to remember that at least three months are required to effect a cure, and the patient should be warned of the danger of relapse. Iron should be administered in sufficient quantities and in gradually increasing doses (*e.g.*, one Blaud's pill, F. 86, thrice daily for the first week, two for the second, three for the third, and so on for five weeks, then decreasing the dose). Liquor ferri perchloridi is a very valuable remedy, if the stomach will tolerate it, on account, it is said, of the amount of free chlorine given off, which acts as an intestinal antiseptic. It may be combined with magnesium sulphate. Ferri sulphas (alone or with aloes), 1 grain thrice daily for the first week, increased to 2 grains for the second and 3 grains for the third, continuing at 9 grains per diem for three months, will seldom fail to effect a cure. If nervous symptoms be present, syrupus ferri phosphatis is useful; while ammonio-citrate or peptonate of iron or reduced iron may be given to patients with delicate stomachs. When menorrhagia is present, treatment directed to this condition should be adopted (§ 322). If the case resists iron in one or another form, the diagnosis should be revised, and tubercle or one of the other conditions mentioned below considered. Other remedies include arsenic, cod-liver oil, lacto-phosphate, malt extract, manganese preparations, dilute acids, and the administration of oxygen. Hypodermic injection of iron is much in vogue on the Continent, and good results are reported therefrom. Rest in bed is necessary in severe cases, and complete rest alone in many cases is of great benefit, for the calibre of the arteries is enlarged thereby and dilatation of the heart is prevented.[1] The curative effects of fresh air must in nowise be forgotten. The patient should sleep with window open, and be always in the open air. For mild cases gentle exercise

[1] See Dr. G. Oliver's book on "Pulse-Gauging," p. 135.

PLATE III.

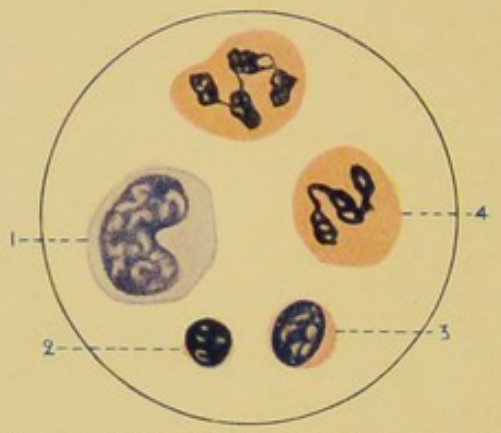


FIG. 1.—FIGURE TO SHOW VARIETIES OF NORMAL LEUCOCYTES.

(*Obj. oil immers.* $\frac{1}{12}$—*Eyepiece* 3.)

1, Large mononuclear leucocyte; 2 and 3, lymphocytes; 4, polynuclear leucocytes.

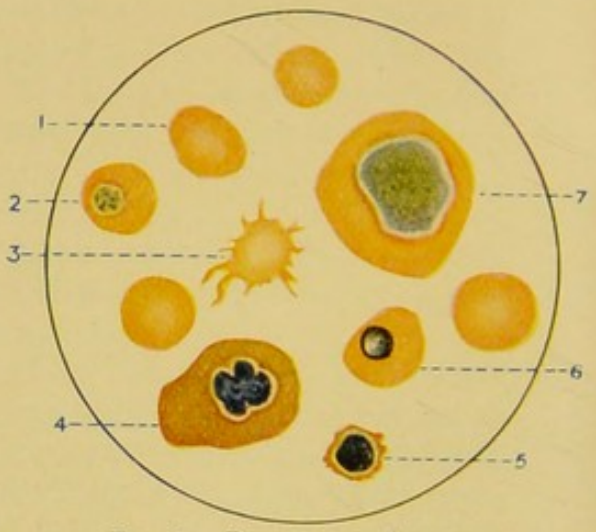


FIG. 2.—PERNICIOUS ANÆMIA.

(*Obj. oil immers.* $\frac{1}{12}$—*Eyepiece* 3.)

1, Normal red cell; 2 and 6, nucleated red cells, normoblasts; 3, cell with ragged protoplasm; 4, old megaloblast with dividing nucleus; 5, microblast; 7, megaloblast.

Fig. 2 is stained by the *Ehrlich-Biondi* method; this shows the copper colour of the coarse eosinophil granules, and the lilac colour of the fine neutrophil granules which occur in polynuclear leucocytes and myelocytes. Red corpuscles stain a yellowish-brown

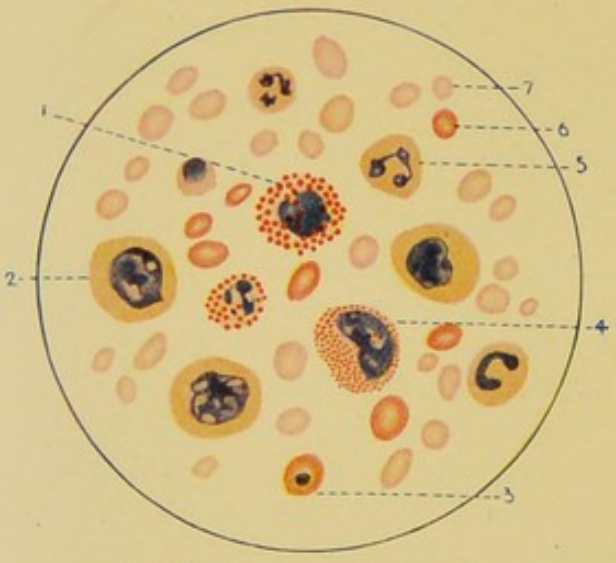


FIG. 3.—SPLENO-MYELOGENOUS LEUKÆMIA.

(*Obj.* $\frac{1}{8}$—*Eyepiece* 2.)

1, Eosinophilic myelocyte (coarse granules); 2, myelocyte; 3, nucleated red corpuscle; 4, myelocyte with fine eosinophil (oxyphil) granules, intermediate between ordinary myelocytes and and eosinophilic myelocytes; 5, polynuclear leucocyte; 6, normal red corpuscle: 7, microcyte (small red cell).

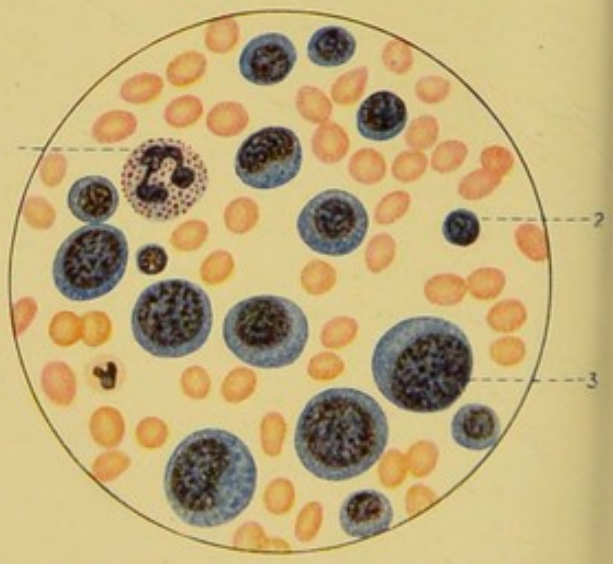


FIG. 4.—LYMPHOCYTHÆMIA (LYMPHATIC LEUKÆMIA).

(*Stained with Leishman's Stain.*) × 500.

1, Polynuclear leucocyte; 2, small, and 3, large, lymphocyte-like cells.

Fig. 3 is stained by the *Eosin* and *Hæmatoxylin* method. Nuclei are stained blue; protoplasm pink; eosinophil granules, red. Note the increased number of myelocytes. The neutrophil granules of the polynuclear cells and myelocytes are not shown by this method.

for both mind and body should be prescribed. Late hours, tight lacing, overstudy, worry, and idleness are to be forbidden. To enlarge the capacity of the chest prescribe respiratory exercises. The diet must be liberal, especially in regard to nitrogenous food in the form of butcher's meat (which should be taken at least once daily), and milk. Green vegetables and fruit are useful. The patient should avoid drinking much at meal times; half a tumbler of fluid is sufficient. Two to three hours after food a tumbler of hot water may be taken. The food should be carefully masticated, and the teeth attended to if necessary.

§ 403. II. Pernicious Anæmia (Synonyms: Idiopathic Anæmia of Addison, Addison's Anæmia) is a relatively rare variety of primary anæmia first described by Addison (who called it idiopathic anæmia), and now known to be associated with certain other signs of toxæmia and of hæmolysis, chiefly affecting men in the second half of life, and running a chronic and generally fatal course. The three principal changes constantly present after death are: (1) The heart exhibits fatty degeneration; (2) the bone marrow is unduly red, and contains a great number of nucleated red cells, especially gigantoblasts; and (3) the liver is fatty, sometimes enlarged, and contains an excess of iron deposited within it, as shown by the ferrocyanide reaction. Extensive atrophy of the gastric mucosa may also be found. Changes have been discovered in the sympathetic ganglia, and in the posterior columns of the cord by Lichtheim and by Burr.

The *Symptoms* may be classed in several groups—viz.: 1. General weakness and anæmia of insidious onset, with their usual effects—*e.g.*, palpitation, dyspnœa, a tendency to syncope, hæmic murmurs, and other symptoms as in chlorosis (*q.v.*). There is little if any wasting; there may, indeed, be much subcutaneous fat. 2. General toxic symptoms, such as lassitude, irregular pyrexia from time to time (though the temperature at other times may be normal or sub-normal), tingling of the extremities, and other vaso-motor symptoms. 3. Oral sepsis and gastro-intestinal symptoms—*e.g.*, sore tongue, dental necrosis, and suppuration of the gums are often associated; gastric pains, and diarrhœa. The spleen is not usually enlarged. 4. Symptoms due to hæmolysis—*e.g.*, urobilinuria, excess of uric acid in the urine, and the lemon tint of the surface of the body. 5. There is a marked tendency to hæmorrhage from the mucous membranes into the internal organs, the retina, and sometimes into the skin. 6. The *blood changes* are very characteristic (§ 398 and Plate III.). (*a*) The red corpuscles are much reduced in number, sometimes falling to less than one-fifth of the normal. (*b*) The colour index is high—*i.e.*, the percentage of hæmoglobin in each corpuscle is increased, but the total hæmoglobin in the blood is diminished—*e.g.*, to 15 to 37 per cent. of the normal. (*c*) The most characteristic alteration in the blood consists of irregularity in size (megalocytes and microcytes) and shape of the red corpuscles (poikilocytosis); nucleated red corpuscles (megaloblasts and normoblasts) are present, with granular degeneration of the protoplasm. It has been suggested[1] that the changes may be due to the arrest of development of the red cells from the erythroblasts (nucleated red marrow cells). (*d*) The pale watery blood of chlorosis coagulates rapidly, but that of pernicious anæmia very slowly, and in the latter the corpuscles show no rouleaux formation. (*e*) Leucocytosis is not present except in the very severe forms.

The *Diagnosis* of pernicious anæmia is effected by considering the age and sex of the patient, and the characteristic blood changes—viz., a marked alteration and diminution in the red corpuscles without corresponding diminution in the hæmoglobin value of the blood—the converse of chlorosis. The diagnosis from some of the diseases given below, and particularly from latent cancer of the pylorus, is often extremely difficult, and can only be accomplished by an exhaustive review of the case.

[1] *E.g.*, Dr. Sydney Martin, the *Lancet*, 1900, vol. i., p. 1006.

Prognosis.—The disease is slow but progressive, and almost invariably fatal. The patient often rallies, and hopes of recovery are entertained, but he almost always relapses. Progress may be estimated by examining the blood from time to time. A preponderance of large red cells is an untoward sign. The main complications are visceral hæmorrhages, cerebral or spinal hæmorrhage, and degeneration in the spinal cord, chiefly affecting the posterior columns. The nervous system towards the end is considerably disturbed. Headache, nervousness, and prostration are fairly constant, but the intellect is usually clear to the end. Sometimes convulsions and coma may occur. A form of paraplegia may supervene, due to a special kind of sclerosis of the spinal cord (subacute combined sclerosis), as Dr. James Taylor has shown.

Etiology.—Pernicious anæmia chiefly attacks males from twenty-five to forty-five, occasionally women of the same age ; it is very rare in the young. It comes on insidiously, without apparent cause. It has been ascribed to (*a*) defective hæmogenesis—*i.e.*, blood formation (Stephen Mackenzie, Rindfleisch, etc.), or to increased hæmolysis—*i.e.*, blood destruction (Stockman and Hunter). The latter theory is strongly upheld by Hunter,[1] who believes the disease is due to a gastro-intestinal sepsis resulting usually from some morbid condition of the mouth, such as dental caries, or suppurative gingivitis. This, he says, leads to absorption of toxins, which destroy the red corpuscles (hæmolysis), and thus give rise to deposition of iron in the liver, urobilinuria, anæmia, and other symptoms.

Treatment.—Complete rest in bed is necessary. Antiseptic mouth-washes should be used, and carious teeth extracted or stopped. The diet should be nutritious, and contain plenty of nitrogenous constituents, such as raw-meat juice. Milk and farinaceous foods are also beneficial. Arsenic has a greater control over the disease than any other drug, but it has often been noted to be of more use in the primary attack than during a relapse ; the dose should be gradually increased until a drachm of Fowler's solution is being given daily. It may be administered hypodermically as the cacodylate of soda (½ to 1 grain), or in the form of atoxyl (¾ grain). Naphthol, salol, and other intestinal antiseptics have been used with advantage. Red marrow, transfusion of blood, oxygen inhalations, and subcutaneous injections of antistreptococcic serum[2] have also been tried.

The patient is PALE, *but the anæmia does not quite conform to the preceding types and is* **not readily amenable to treatment by iron.** The disease is probably some LATENT CONSTITUTIONAL CONDITION (syphilis, plumbism, tubercle, or carcinoma), or some LATENT VISCERAL DISEASE.

§ **404.** III. **Syphilis** (Synonym : Hunterian or Constitutional Syphilis) is a constitutional malady, due to a microbic infection, which starts as a superficial ulcer (chancre) at the seat of inoculation, runs a prolonged and indefinite course, is liable to break out anew during the whole lifetime of the patient without fresh infection, even after many years of quiescence, and in its later stages produces granulomatous deposits in various parts of the body, particularly in the skin and nervous system.

Symptoms.—Syphilis leads to a degree of pallor which may simulate chlorosis or other forms of anæmia very closely. When no history of primary syphilis is obtainable, and no physical signs can be discovered, the diagnosis from other forms of anæmia may be difficult. For the sake of convenience, the symptoms of syphilis are divided into three stages, but it must not be forgotten that their mode of appearance is

[1] Dr. William Hunter; the *Lancet*, 1901, vol. i., pp. 473, 930, and 1903, vol. i., p. 283.
[2] See the *Lancet*, 1901, vol. i., p. 930.

extremely variable, and that the three stages may even appear simultaneously in certain patients. *Primary Stage.*—The latent period—period of incubation—generally lasts about *three weeks*, but it may vary from ten to forty-six days. The initial manifestation appears as a superficial ulcer (the hard or Hunterian chancre) at the site of inoculation. It is usually single, and occurs most commonly on the prepuce or glans penis in the male, and the labiæ and nymphæ in the female. It originates as a flat, elevated, painless papule, which slowly enlarges, and may desquamate without breaking down, or superficial erosion or ulceration takes place with a slight serous discharge, thus differing from the deep excavated ulcer of the "soft sore" or non-Hunterian chancre. The underlying induration of the tissues is always a marked feature, hence its name "hard sore." The lesion after a time cicatrises, and usually leaves behind it some slight discoloration or induration, or both, which mark the site. Sometimes the primary sore is so slight as to be overlooked, and appears to be wanting, especially in the female. About the same time, or within one or two weeks, the associated lymphatic glands, usually in the groin, become enlarged and hardened. Even thus early in the disease, the red blood discs are generally diminished to 3,000,000 per cubic millimetre or even less, and there is pallor and weakness, conditions which increase as the disease progresses if untreated. The glandular enlargement may become generalised, and may persist for months or years, and this hardness of the lymphatic glands may thus serve as an aid to diagnosis at any time.

The *Secondary* symptoms make their appearance about three weeks after the first appearance of the chancre (four to twelve weeks after inoculation). In typical cases a faint generalised dusky macular rash (which may be brought out more distinctly by a warm bath) appears chiefly on the chest and abdomen, nearly always attended by sore throat, and often attended by malaise, pains in the limbs, anæmia, and slight pyrexia. The rash takes about three weeks to mature and three weeks to decline. The pyrexia, which is generally overlooked, has already been described (§ 382). The sore throat is usually of an indolent, ill-marked kind, with whitish secretion resembling snail tracks on the tonsils (§ 113). The eruptions which may appear now and hereafter are of many different kinds—macular, papular, scaly, pustular, tubercular, practically never eczematous or vesicular. The characteristics of these (see also § 489) are their reddish-brown colour, generalised or symmetrical distribution, grouping in segments of circles, and their preference for the forehead and flexor surfaces, their polymorphism and absence of itching. The hair may fall out, and the nail-beds be affected with an indolent inflammation. Moist "mucous patches," with a highly contagious secretion, are apt to appear at the corners of the mouth and other mucous orifices. The diagnosis of the skin symptoms (§ 489) and the lesions of the mucous membranes (§ 148) are dealt with

elsewhere. The eyes may become affected by iritis, choroido-retinitis, and the bones with periostitis in which the pain is worse at night, the nervous system and the viscera with gummata, and the joints with synovitis. Any of these symptoms may crop up again and again during the ensuing months or years.

Later Stages (so-called tertiary symptoms) and *Varieties* of syphilis.—In practice it is convenient to recognise two broad varieties of syphilis. In most cases of a BENIGN type, *adequately treated* there is no recurrence of symptoms after the second stage above described; in short, there is no tertiary stage. But in other cases the disease assumes a MALIGNANT type either by reason of the intensity of the virus or the predisposition or debilitated state of the individual, combined perhaps with inadequate treatment in the earlier stages, and such cases are characterised by severity of the initial symptoms and a tendency to recurrence at intervals throughout life. It is in such cases more especially that one meets with what are known as "tertiary" symptoms. Malignant or tertiary lesions, as exemplified, for instance, in the skin, are characterised by having a greater and deeper infiltration, a greater proneness to suppuration, ulceration, and scarring, and by being followed by more loss of tissue than the benign lesions. All the same skin symptoms noted in the secondary stage may recur, but they are more apt to be localised and asymmetrical in distribution, serpiginous in outline, lenticular or nodular in shape, and pustular or ulcerating in character than the corresponding secondary symptoms. Nodular or infiltrating gummatous deposits followed by scarring, and perhaps by ulceration, may affect the mucous membranes, particularly in the oral cavity and its diverticula, the liver and other abdominal organs, and the cephalic and the genito-urinary organs, and lead to fibroid degeneration, stricture, or destruction of the proper tissues and functions of the parts. The bones are often attacked by gummatous periosteal deposits, leading in the case of the hard palate to perforation, and in the other flat and the long bones to the formation of "nodes." The arteries may become thickened and arterioles blocked; partly for this reason, partly by the proneness of the virus for the nerve-tissues and meninges, the nervous system is specially apt to be involved. But even this list does not complete the account of this insidious, prolonged, and terribly far-reaching disease, for it is, as mentioned in previous chapters, one of the two causes of lardaceous disease of the liver, spleen, kidneys, and intestines. More or less anæmia is a symptom throughout the disease, and in untreated or malignant cases of syphilis the cachexia may sometimes be fatal, as in patients referred to on p. 581 and elsewhere.

PARA-SYPHILITIC ($\pi\alpha\rho\alpha$ = derived from) is a term applied to certain diseases which are not due to definite syphilitic deposits or infiltrations, but are nevertheless due to the indirect effects of the syphilitic poison on the system, or to its after effects. Thus locomotor ataxy is due to

the after-effects of the virus on the muscle sense neurons. Paralytic dementia and certain types of epilepsy are also examples of para-syphilitic disorders.

Hereditary or Congenital Syphilis.—We have seen how ubiquitous the consequences of syphilis may be, but it is upon the children of such parents that the heaviest nemesis falls. Happily, the mother very often aborts, syphilis being one of the commonest causes of abortion, or the child dies and decomposes within the uterus, or, being born alive, it dies in the first twelve or eighteen months of life of marasmus or its complications. Thus a series of miscarriages or still-births, or a heavy mortality among children in the early months of life, imply a strong probability of syphilis in the parent. If the child be born alive the primary chancre is of course wanting, but the symptoms conform more or less to the secondary symptoms above described. The infant is sometimes healthy at birth, but in a few weeks it develops "snuffles," or a ham-coloured eruption on the buttocks, flexures, palms, or soles, and marasmus sets in and is followed (if active treatment is not adopted) by any of the other secondary symptoms above mentioned. The child is fretful, the cry is hoarse, and the bones are tender, and gastro-enteritis, bronchitis, or pneumonia may complicate matters. If the child survives the first twelve or eighteen months of life, a long period without fresh syphilitic manifestations ensues, excepting perhaps in the rapid decay of the temporary teeth and stunted growth of body. About the seventh year, however, the permanent teeth appear, and usually present the pegged shape and notched border described by Sir Jonathan Hutchinson (Fig. 3, p. 21). Again, there may be an interval of quiescence, but about the fourteenth year or puberty, interstitial keratitis, deafness, periostitis, or synovitis may appear, the skin, viscera, and nervous system only rarely being affected. From this time onwards the evidences of hereditary syphilis consist of the consequences of the previous lesions on the general development, the skin, the mucous orifices, the malformation of the bones, the eyes, ears, and teeth, which are summarised in Table XXIV., pp. 582, 583.

The *Diagnosis* of the Hunterian chancre will be found in surgical works. The diagnosis of syphilitic symptoms and lesions in the skin, nervous system, liver, and other parts, will be found in the appropriate chapters of this work. The existence of hard shotty glands as an aid to diagnosis has already been referred to. In regard to the diagnosis of syphilitic from other forms of anæmia undoubtedly mistakes may easily be made. I remember the case of a very anæmic lad of fifteen in whom the only other symptom besides those of anæmia was a slight rise of temperature in the evening, which was diagnosed as one of pernicious anæmia; after death, however, gummatous deposits were found in the meninges in the frontal region and elsewhere. A somewhat similar case of gumma of the liver was reported by the late Dr. J. S.

Bristowe.[1] Syphilis should always be suspected in obscure cases of anæmia, and very often the amenability to treatment by iodide and mercury will be a revelation. The presence or history of an eruption should be noted, and the viscera, the bones, and the eyes very carefully examined. Wasserman has introduced a test for the blood of a syphilitic patient, but it is too complicated for ordinary clinical use.

Prognosis.—Syphilis is never fatal (except, perhaps, in the case of the fœtus) by the intensity of its toxæmia, like small-pox or scarlatina; but it may kill in infancy by marasmus. In adults it only kills, usually after a life of invalidism, by its complications or by involving some vital part. Benign cases of the disease adequately treated, if the patient lives a temperate, hygienic life, may give no after trouble, and many such persons live to old age and have perfectly healthy children. Nevertheless, it behoves even these patients to be constantly on their guard, for once syphilitic means that they are always liable to the possibility of recurrence, even to the end of their days. Malignant types of the disease are sure to recur, and to require active treatment on and off throughout life. The severity and duration of an attack of syphilis are influenced by a number of circumstances, some of which are hard to gauge. The habits and mode of life (especially as regards intemperance), age, occupation, exposure, privation, pre-existing disease (especially tuberculosis and renal disease), all doubtless influence the course of the malady. The disease is often said to prevail in a particularly virulent form in some naval and military stations. But of all factors, the one which influences the prognosis of syphilis more than anything else is adequate and continuous treatment during the earlier phases of the malady.

Table XXIV.—Hereditary Syphilis.[2]

A. INFANTILE MANIFESTATIONS (three weeks to three months).

I. May be born quite healthy. Then symptoms resembling acquired secondary syphilis appear—symmetrical, transitory, etc.

II. Mucous Membranes { Snuffles. / Condylomata around anus or mouth.

III. Marasmus, leading to "senile aspect"; very marked wasting, often fatal.

IV. Skin { Papular . / Scaly . / Pustular . / Bullous . / Polymorphic } Always symmetrical, transitory, ham-coloured; on buttocks because of urine and fæces; in flexures because of perspiration. Patches of peeling erythema about face, nates, neck, etc.

V. Iritis.

VI. Definite Periostitis—Tenderness of bones and "rheumatic" pains, epiphyseal abscesses, or caries of long bones. Skull—thinning in one place, thickening in another. Skeletal deformities and nodes.

[1] Clinical Soc. Trans., vol. xix., p. 249.

[2] This Table is after Fournier, modified.

B. ADOLESCENT MANIFESTATIONS (commencing about puberty).

Which come on after an interval of quiescence of some years, if the child survive the first year of life.

I. Nebular Keratitis—first one cornea, then the other appears like ground-glass—between tenth and twentieth year. Ultimately quite clears up under treatment (Fig. 3).

II. Deafness—between puberty and twenty-third year—comes on with noises in ears, but without pain or otorrhœa—terminates in recovery or complete incurable deafness.

III. Periostitis of long bones (rarely skull)—generally causes overgrowth, sometimes bending, or nodes, occasionally suppuration.

IV. Synovitis (painless)—knee or other large joint.

V. Skin, viscera, and nervous system rarely affected at this stage.

C. LATE MANIFESTATIONS (from fifteen years upwards).

Note.—All of these, being the results of infantile syphilitic inflammations, are absent if syphilitic manifestations have been previously wanting.

I. *Constitutional Effects.*
- *Infantile build.*
- *Retardation* of development, of growth, of dentition, of the catamenia.

II. *Tegumentary System.*
- *Skin*—Peribuccal cicatrices radiating from the mouth; Parrot's cicatrices.
- *Eruptions* (very rare)—Lupoid ulceration, gradually spreading, may appear.
- *Mucous membranes*—Cicatrices of the throat, palate, and round the mouth. Hole in palate, etc.

III. *Osseous System.*
- *Cranial malformations* — prominent frontal eminences, natiform cranium, asymmetry, hydrocephalus.
- *Nasal malformations* —"Duck-nose," depressed septum, "opera-glass nose."
- *Tibial deformities*—"Sword-blade" tibia; or curving with shortening; or increased length.
- *Joint lesions*—Chronic painless effusions, and distorting arthropathies.

IV. *Hutchinson's Triad.*
- 1. Eye
  - Ocular malformations.
  - The remnants of interstitial keratitis (striæ in cornea), iritis, or choroidal atrophy.
- 2. Ear — Cicatrices of the tympanum, deafness.
- 3. Teeth
  - Underhung or displaced jaws, irregularities or absence of teeth.
  - Dental dystrophies — microdontism, amorphism, "pegged teeth" of Hutchinson (Fig. 3).

V. *Family History.*
- Miscarriages and still-births in series.
- Heavy mortality among children in first three months of life.

Etiology.—The specific microbe has now been identified as a feebly staining spirochæte, to which the name *Spirochæta pallida*—Schaudinn (*Treponema Pallidum*) has been given. It can be obtained not only from

the primary sore, but in abundance from condylomata, and also from the viscera in secondary, tertiary, and congenital syphilis. The organism is a corkscrew-like spirillum, having from eight to twelve curves, one end of which is filamentous, the other thicker and more deeply staining. It is differentiated from a commonly occurring spirillum, the *Spirillum refringens*, in that the latter has fewer and less delicate curves, stains equally deeply at both ends, and has no thickening at one end.

Syphilis resembles the specific fevers in having a period of incubation followed by a fairly characteristic eruption, and in the fact that one attack renders a person immune to a second attack, with very rare exceptions. It differs from other specific fevers in the extreme length of its course, which may last many years, in the long intervals which

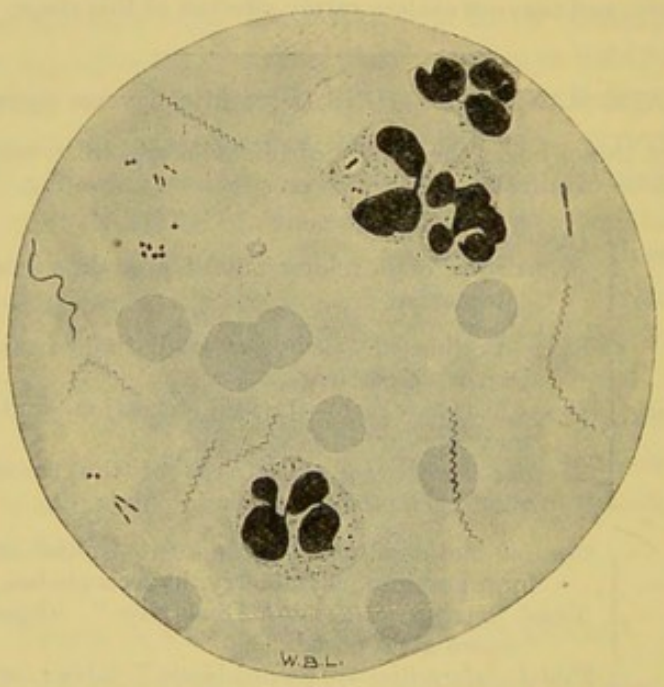


Fig. 119.—Spirochæta Pallida (Treponema Pallidum) of Syphilis, magnified about 500 diameters. Illustration lent by the courtesy of Colonel W. B. Leishmann, R.A.M.C. The organism is of a spiral form like a long corkscrew.

may separate its various manifestations, and above all in its curious liability to recur without fresh infection. Inoculation can only take place through an abrasion of the skin or mucous membrane, and may occur in three ways : (*a*) Usually it is by direct contact with an infected person, generally during sexual intercourse, but in some cases (*e.g.*, in doctors and midwives) as the result of examining diseased persons, by suckling (as in wet nurses), kissing, etc. ; (*b*) occasionally by the use of contaminated articles—*e.g.*, spoons, cups, pipes, towels, surgical instruments, or (?) the seat of a w.c. In the first two stages the blood and the *moist exudations of all the lesions are certainly contagious.* In the later stages some difference of opinion exists as to the contagiousness of the blood and secretions. My own experience is that all moist lesions

in all stages (and therefore the blood also) are contagious, and I well remember a patient of mine who had contracted the disease thirty-three years before, and who conveyed the disease to his sister by kissing, he having at the time only a small fissure at the angle of his mouth. In regard to marriage, I see no reason to modify the rule about to be stated, however old-standing the disease may be. The use of human vaccine lymph, even when free from blood, for vaccination purposes from arm to arm, has undoubtedly been the occasional means of propagating syphilis, but the frequency of the occurrence has as certainly been exaggerated. (*c*) To the offspring syphilis may be conveyed by hereditary transmission of the virus from either parent. If, as frequently happens, the mother becomes infected shortly before she becomes pregnant, or during the early months of pregnancy, the child seldom escapes the disease ; if after the seventh month, however, the child may be healthy. It is said that the child may be infected from the father, and that the mother may nevertheless escape ; but this is extremely doubtful, for under these circumstances the mother never contracts the disease from her child after its birth (Colles' law). The date when marriage is permissible is a most important one. It should under no circumstances be sanctioned within two full years after infection, even in the mildest case. Subsequently, if the patient has been *free from any syphilitic symptoms* for at least twelve months (Jonathan Hutchinson), and has been *undergoing anti-syphilitic treatment* during that time, there is every prospect that the offspring will be healthy. It is essential to insist on this interval of quiescence as a minimum, combined with this period of treatment, before sanctioning marriage or possible conception.

Syphilis has, there is little doubt, prevailed from very ancient times, and has occasionally occurred in the form of widespread and severe epidemics, particularly when introduced into previously healthy communities. Individuals of all races and ages are subject to it. Metchnikoff and Roux[1] and Neisser have succeeded in inoculating apes with syphilitic virus and reproducing the disease in them ; the anthropoids are more readily infected, and show more characteristic lesions than the lower apes.[2] Extremes of temperature seem to intensify its virulence. Tuberculous and otherwise debilitated subjects usually incur syphilis in a severe and malignant type.

Treatment.—Fortunately, in mercury and iodine we have efficient remedies for controlling this serious and far-reaching disease. The most effectual remedy in the *earlier stages* is mercury which should be given continuously for at least eighteen months, whether symptoms be present or not (Mr. Hutchinson gives eighteen months, Dr. W. Osler two years), and subsequently, if symptoms are still present, until at

[1] *Ann. de l'Inst. Pasteur*, October, 1906.
[2] Neisser, *Brit. Med. Journ.*, October 10, 1908.

least three months after all evidences of the disease have ceased. It may be given by mouth, by inunction or fumigation, and by hypodermic injections into the muscles or veins. The precautions to be observed in all cases, in addition to the duration of the treatment just mentioned, are : (i.) The mercury must be gradually increased until the gums become tender, then regulated until toleration is established. It is well to get rid of decayed teeth at the very outset, and a mouth-wash of potassium chlorate and weak carbolic acid may be necessary. (ii.) The patient should be seen once or twice a week, so as to watch for salivation, diarrhœa, gastric disturbances, and to regulate the treatment. If the mercury be temporarily stopped, 5 grains of iodide of potassium should be given thrice daily. Debilitated subjects and subjects of renal or visceral disease require smaller doses and extra caution. (iii.) The patient should absolutely avoid alcohol and tobacco while under treatment, and take plenty of milk and light food. The primary sore, according to Neisser, should always be excised, although the virus may be demonstrated in the internal organs of apes before the sore appears. This is a further argument for the earliest active treatment of the disease both locally and constitutionally. The open-air treatment, as for tuberculosis, is given at Aix-la-Chapelle. Sea air and sea voyages are specially beneficial.

Mercury is ordinarily administered by the mouth, liquor hydrargyri perchlor. (with or without potassium iodide) being given thrice daily after meals, gradually increased ; or hyd. c. cret. gr. i. or ii., or pil. hydrargyri gr. i. or ii. may be given with opium, gr. $\frac{1}{10}$, twice daily. In private practice a convenient way to give mercury is by means of a pill of hydrargyri iodidi viridi, gr. $\frac{1}{2}$ to 1, with opii, gr. $\frac{1}{4}$ to $\frac{1}{3}$, twice daily. Sarsaparilla and guaiacum, when added to medicines containing mercury or iodide, appear to increase their effects in some cases. The inunction of ung. hydrargyri is another method, a piece the size of a Barcelona nut being well rubbed into one or other axilla or groin every night. It is a dirty method, and does not admit of exact dosage. In infancy the favourite methods are the administration of hyd. c. cret., gr. i. or ii., once or twice daily, or the wearing of a broad flannel bandage on which ung. hydrargyri is spread daily. For adults a cleaner and prompter method than inunction is the mercurial vapour-bath (F. 4) given daily at first.

The hypodermic introduction of mercury—which is clean, convenient, and does not upset the stomach—dates from the time of John Hunter and has come into vogue again during the last few years, many different preparations having been used. I have been well content with a solution of perchloride of mercury (F. 24), to which 1 or 2 per cent. of cocaine may be added to prevent pain. A sterilisable hypodermic syringe is used, the needle being made of platino-iridium (which resists the action of the mercury) and being of rather larger bore than ordinary

hypodermic needles. The skin and the hypodermic needle having been rendered aseptic, the solution is injected into the substance of a muscle, preferably the muscle of the buttock. Start with a small dose, about $\frac{1}{10}$ to $\frac{1}{5}$ grain daily, or every other day, until the gums become affected; gradually increase the dose, and then administer about $\frac{1}{4}$ grain once or twice a week, or every second week, according to the state of the gums. Some prefer the succinimide (F. 24), peptonate, cyanate,[1] cyanide[2] or other soluble salt of mercury, and some recommend a solution of bin-iodide in oil.[3] The insoluble preparations—calomel, metallic mercury, or salicylate of mercury suspended or emulsified in paraffin and injected by means of special syringe—have been largely used in France and Germany,[4] but I have found the soluble salts to be more convenient, more rapid in their action, and more exact in their dosage. L'huile grise (mercury suspended in oil) is said to be sometimes cumulative in its effects. The intravenous injection of mercurial preparations is advocated by some, but is not free from the danger of thrombosis.[5] Syphilis has been treated by the injection of serum derived by a cantharides plaster from patients with tertiary symptoms; 10 to 40 c.cm. were injected every third or fourth day for one or two months. Refractory cases are said to have improved, and the improvement to have been more lasting than after a course of mercury or iodides. Neisser recommends that arsenic in the form of atoxyl should be combined with the mercury. It may be used as a 10 per cent. solution for injection into the gluteal region, 60 cgm. being injected for two consecutive days in each week for from twelve to fourteen weeks in one cycle of treatment. Care should be taken not to inject it into a vein. The atoxyl must be pure.

It is in the later *secondary* and in the *tertiary stages* of syphilis and particularly in the internal manifestations, that potassium iodide finds its chief use, and in a large proportion of these cases this alone will promote absorption, if given in large enough doses. My belief is that the doses generally given are not large enough, because I have known many cases which fail with 5 or 10 grains succeed with larger doses. My practice is to start with 20 grains, and rapidly increase the dose to 60 grains thrice daily after meals, followed by a glass of milk. If the patient has running from the nose and eyes, it may often be relieved by doubling the dose. The largest dose I have given was 90 grains thrice daily. Iodide spots may sometimes be relieved by 3 drops of liquor arsenicalis, and as the iodide lowers the arterial tension, spiritus

[1] Max Stern, *Munich Med. Woch.*, July 2, 1901.
[2] Bonzitat, *Thèse de Paris*, July, 1902.
[3] Melis, "Archives Belges," 1901, p. 21, and *Brit. Med. Journ.*, 1901, vol. ii., "Epitome," p. 20.
[4] Roux, *Jour. de Med. and Chir. Prat.*, May 10, 1902; Dr. Leredde, *Medical Press*, October 29, 1902; Max Stern, *Munich Med. Woch.*, July 2, 1901; see *Brit. Med. Journ.*, May 30, 1903, p. 1258.
[5] Mendel, "Medical Annual," 1904, p. 22.

ammoniæ aromat. or other stimulants may be called for. If iodide of potassium disagrees, even when followed by milk, substitute the sodium and ammonium salts. After the symptoms have disappeared the doses may be lowered, but on no account should the iodide be stopped until at least three months later. Mercury may with advantage be added in bad cases or if an insufficiency of the metal has been taken.

The recent experiments of Metchnikoff and Roux with virus, attenuated by passage through lower apes, gives reason for hope that by this means a vaccine may be prepared for man.

§ **405.** IV. **Plumbism** (Synonyms : Saturnism, Chronic Lead Poisoning). —Chronic anæmia, usually associated with a number of other symptoms, results from the slow absorption of lead into the system, due to the contamination of drinking water or to the occupation of the patient.

Symptoms.—(1) The anæmia is very marked ; the red corpuscles may be reduced to 50 per cent. The pale, pasty appearance of house-painters is well known. The anæmia of lead, as in the two preceding causes, is not very amenable to iron. (2) The gums, with but very few exceptions, show the so-called " blue line," a peculiar livid line on the gums close to the teeth. It is due to the formation of lead sulphide. A few medicinal doses of lead, copper, and other metals may produce it. Dr. Thomas Oliver[1] says the true blue line is incapable of removal by medicinal treatment under from eight to twelve weeks. (3) Very obstinate constipation is usual in the subjects of chronic lead poisoning, and is sooner or later associated with (4) severe intestinal colic (§ 172). This colic is very apt to recur, and its recurrence is an aid to its diagnosis. (5) Lead is usually found in the urine, and chronic plumbism is frequently associated with slight albuminuria. Ultimately chronic Bright's disease supervenes. The arterial tension is high, and arterial sclerosis supervenes relatively early in life. (6) Lead has a special tendency to attack the peripheral motor nerves. It has a special proclivity for the musculo-spiral nerve, and thus the typical lead palsy is a " wrist drop " due to paralysis of the extensor muscles of the forearm. It is important to note that the supinator longus generally escapes, and therefore supination is preserved. Both limbs are invariably affected, though one side may be worse than the other. The muscles rapidly waste, and present a typical reaction of degeneration. Other recognised types of paralysis are the brachial (or scapulo-humeral) the Aran-Duchenne (resembling chronic anterior polio-myelitis), the peroneal, and the adductor laryngeal. Sometimes all the limbs are affected, so that a complete generalised paralysis, including perhaps the diaphragm, may supervene. Muscular tremor is not very common in the lead poisoning due to drinking water, but is met with sometimes in the pre-paralytic stage ; it is, however, very usual when the lead is introduced into the system by inhalation—*e.g.*, among lead miners and

[1] " Dangerous Trades," London, 1902.

glass-blowers. Optic neuritis, neuro-retinitis, amaurosis, and amblyopia are met with. Cerebral symptoms (lead encephalopathy) are met with, such as acute mania, convulsions, coma, and occasionally insanity.

The *Diagnosis* of lead poisoning is not difficult when once our suspicions are aroused. Each of the above symptoms is fairly characteristic, and when all are met with together there can be no doubt. In doubtful cases of anæmic pallor the urine should be carefully examined for lead. The " blue line " is very characteristic.

Prognosis.—The prospect of recovery varies with the duration of the disease, the severity of the symptoms, their amenability to treatment, and the condition of the kidneys. Slight palsies may be completely restored, but when there is extreme muscular atrophy and persistent absence of faradic reaction recovery is rare.

Etiology.—(1) Numerous occupations afford opportunities for the introduction of the poison either into the alimentary canal or by inhalation into the lungs, such as painters, lead workers, plumbers, pewterers, glazed card makers, pottery glaziers, file cutters, chromic dye workers, enamellers, glass workers, lead foil makers, shoe finishers, bleachers of Brussels lace, printers' compositors, and lead miners. (2) The disease may occur in epidemics owing to the consumption of contaminated water which has been stored in leaden cisterns, or has passed through leaden pipes. A specially soft water is apt to dissolve a certain amount of lead carbonate usually found inside leaden pipes. In other epidemics, such as that of Newcastle in 1900, the water has been found to be slightly acid near its source. (3) Various articles of food or drink are apt to become contaminated when stored in lead, lead-glazed, or pewter vessels, such as tinned provisions, beer, cider, or wines. Cases still occur in men who have drunk the beer first drawn off in the morning which has lain some hours in a pewter or leaden pipe. (4) Rarer causes are the sleeping in newly-painted rooms, and occasionally in susceptible persons the external application of a lead lotion may produce poisoning. The internal administration of lead as an abortifacient is said to be common in some Midland towns (Ransom). Any age or sex may be affected, but women appear to be more susceptible than men. A first attack may not occur until after exposure for many years. Alcohol predisposes.

Treatment.—The first indication is the avoidance of the cause, and those who are exposed to the poison by reason of their occupation should observe the greatest personal cleanliness. The face, hands, and teeth should be cleansed before meals. The ventilation of the workroom should be supervised, and a respirator worn if the air contain much dust. Dr. Thomas Oliver has a poor opinion of the prophylactic value of sulphuric acid lemonade, inasmuch as the sulphate of lead is hardly less soluble than the carbonate. Fruits and alcohols should be avoided. Saline aperients and small doses of iodide of potassium should be

administered, but the latter should be increased with caution, so as to avoid flooding the blood with a soluble lead salt. The treatment of the constipation, colic, paralytic and other nervous lesions will be found in their appropriate places.

V. **Incipient Tuberculosis** is generally attended by anæmia, pallor, weakness, and loss of flesh. The anæmia is often very marked, and if a young anæmic patient is not amenable to treatment by iron latent tuberculosis should always be suspected (Trousseau). The disease may be entirely latent in the sense of being unattended by any physical signs in the lungs or elsewhere. It is useful to remember that a tuberculous process, no matter where it is situated, is always attended by pyrexia of an intermittent type, though this is apt to be overlooked. Inquiry should therefore be made for sweatings or "chills," careful temperature readings should be procured, and the sputum and urine should be examined for the bacillus. The early diagnosis of tubercle in the lungs, meninges, kidneys, peritoneum, and other parts has been given in their appropriate places. Early spinal caries may also be overlooked, and the only symptoms present may be slight pain in the hypogastrium.

Certain bacterial tests are now employed as methods of diagnosis in tuberculosis: (1) *The Opsonic Method.*—The opsonic index is estimated daily. If it is unduly high or unduly low, or if it fluctuates widely, a focus of tubercle is present; between 0·8 and 1·2 is normal. (2) *Calmette's Reaction.*—If a glycerine free solution of tuberculin be dropped into the conjunctival sac of a tuberculous patient, a characteristic reddening of the conjunctiva occurs, accompanied by lachrymation, and the formation of a fibrinous exudate; this reaction does not occur in non-tuberculous subjects. Symptoms are noticeable in three to eight hours, increase to a maximum in from six to fourteen hours, and disappear usually in from twenty-four to thirty-six hours. Sometimes severe conjunctivitis persists. A solution containing 1 part of tuberculin in 200 is commonly used, one or two drops being placed in the eye. In some severe cases of tuberculosis no result follows. (3) *Von Pirquet's Reaction.*—The skin of the arm or thigh is scarified as for cow-pox vaccination, and the prepared tuberculin placed on the abraded surface, with the usual aseptic precautions. In the tuberculous subject inflammation appears at the site of the inoculation in from ten to fifteen hours. On about the second day small vesicles form, which subsequently fade, and the sore is normal in eight to ten days. It is a useful test for children, but fallacious for adults.

VI. Incipient or **Latent Carcinoma** and sarcoma are also attended by pallor, weakness, and emaciation; they form the essential parts of cancerous cachexia. The pallor does not yield to iron. Emaciation is, however, usually the most constant and most prominent feature, and therefore malignant disease will be considered fully under that symptom (§ 415). I have twice mistaken cases of scirrhus of the pylorus for examples of primary anæmia.[1] They were cases in which the pyloric end of the stomach was drawn up under the liver and entirely escaped detection during life. The diagnosis is all the more difficult when it occurs, as it did in these cases, without vomiting, and in comparatively young women, aged twenty-eight and thirty-five respec-

[1] *Clinical Journal*, February 13, 1895, vol. v., p. 251.

tively. There may be hæmic murmurs, and on rare occasions intermitting pyrexia. Another case of anæmia which came under my notice for a severe persistent neuralgia of the third sacral nerve, lasting many months, eventually proved to be carcinoma of the prostate. Repeated careful examination should be made of all the abdominal and pelvic organs.

VII. **Dyspepsia, Constipation, Colitis,** and various other disorders of the **alimentary canal** frequently come under our notice for pallor. Indeed, dyspepsia and confinement indoors are perhaps the commonest causes of pallor among hospital out-patients, and it must not be forgotten that decaying teeth associated with pyorrhœa alveolaris may, as Dr. William Hunter[1] has shown, be a potent cause of intense anæmia. Deficient food, and particularly deficient nitrogenous food, may also act in a lesser degree. In dyspeptics the pallor is of a peculiar kind, in which the skin loses its lustre and may even be scurfy, and there are dark rings under the eyes. In colitis and other intestinal affections the same may be observed, and it is wonderful what an improvement in the patients' appearance is made after a course of treatment by salicylate of bismuth, naphthol, or other intestinal antiseptics, combined with purgatives. Many cases in this group are auto-toxæmias leading to deficient production or increased destruction of hæmoglobin, or both.

VIII. **Aortic Valvular Disease** often presents a sallowness which may be mistaken for the pallor of primary anæmia. The patient is usually an adult at or past middle life. In aortic regurgitation the cardiac murmur and the pulse (§ 47) are sufficiently characteristic for the detection of the disease on examination. But in aortic obstruction the most experienced auscultators may fail to detect or may misinterpret the signs of the lesion.

IX. **Chronic Renal Disease** is sometimes accompanied by a pallor which may readily be mistaken for primary anæmia. This is especially the case in chronic parenchymatous nephritis, which is apt to affect young people. The pallor, however, is of an ivory whiteness, is usually accompanied by a certain amount of dropsy, and the urine reveals a definite amount of albumen and tube casts. Chronic interstitial nephritis is usually met with in older people; it is generally attended by sallowness, but is mentioned below under progressive asthenia, which is its more constant and striking symptom.

X. **Cirrhosis of the Liver** may be attended by an anæmic pallor; but it is usually attended also by dilatation of the venous capillaries in the face, which are very characteristic. The signs and symptoms of this disease may be very obscure.

Certain other secondary anæmias are readily detected by their history—viz., Hæmorrhage, or long continued drain on the system; Chronic Suppuration, or other septic processes; Rheumatic and other febrile conditions.

[1] The *Lancet*, 1901 and 1902.

XI. Anæmia may be due to **hæmorrhage**—profuse and sudden, or small and frequent; hyperlactation, profuse chronic diarrhœa, and other debilitating conditions. Such cases are generally discoverable by a history of the cause, such as menorrhagia, post-partum or ante-partum hæmorrhage, bleeding piles, melæna, recurrent epistaxis, hæmatemesis, hæmoptysis, hæmaturia, etc. The treatment resolves itself into attacking the cause and applying the same measures as for chlorosis. But in severe cases of post-hæmorrhagic and other forms of intense anæmia, and whenever collapse is present, transfusion or infusion may be called for.

§ **406.** Transfusion of Blood or Infusion of Saline Solution may be indicated in the presence of shock, collapse, or intense anæmia, arising in three groups of conditions: (1) Acute anæmia, resulting from abortion, post-partum, and other causes of uterine hæmorrhage, gastric or intestinal ulceration, operations attended by profuse bleeding, or internal hæmorrhage after abdominal injuries. (2) Collapse consequent upon surgical operations, cholera, or severe diarrhœa due to any cause. (3) Blood poisoning—*e.g.*, in uræmia, puerperal eclampsia, diabetic coma, or poisoning by carbon monoxide, carbon dioxide, illuminating gas, carbolic acid, strychnine, or phosphorus. *Transfusion of blood* from one person to another is at the present time not so much employed as in the past. It is indicated where the nutritive value of the blood is required. It is described in surgical text-books. *Infusion of saline solution* can be much more rapidly and easily performed, and skilled assistance is not necessary. As a mere diluent or to make up the volume of the blood it is of great value, and has almost entirely superseded transfusion. In cases of blood poisoning (Group 3 above) venesection should be performed to eliminate the poison before the saline solution is administered. Sodium chloride (a neutral salt) is used to bring the specific gravity of the fluid to be injected up to that of the blood plasma. The normal saline solution (0·75 per cent.) is prepared very readily by dissolving 1 drachm (or to be exact 66 grains) of common salt in a pint of *boiled* water. The infusion may be made (1) into a vein; (2) into the subcutaneous cellular tissue of the thighs, anterior abdominal wall, below the clavicle, or mammæ; or (3) into the rectum. For injecting the solution into the subcutaneous tissue an ordinary exploring needle connected to a rubber tube 5 to 7 feet long, with a glass funnel or a douche-can—all rendered aseptic—and a thermometer, complete the necessary apparatus. The needle is inserted after cleansing the skin, and the operator standing on a chair pours the solution (at a temperature of 99° or 100° F.) from a height of 5 to 7 feet above the needle slowly into the funnel. Three to six ounces may be injected at intervals of ten to fifteen minutes. If necessary, more than one puncture may be made. Usually ½ to ¾ pint is sufficient, but 2 to 3 pints may be needed. The puncture must afterwards be sealed with collodion. The danger of abscess or sloughing is very slight in young or middle-aged persons if the infusion be slowly done, and not too much in one place, and if aseptic precautions are thoroughly carried out. To inject saline solution into the rectum a similar apparatus with a rectal tube is employed. This method is simple, yet often of great service, particularly in the collapse after operations.

XII. **Chronic Suppuration** (with or without amyloid disease) and other septic processes and causes of a prolonged drain on the albuminous materials of the blood are potent sources of anæmia. Prolonged lactation also may act in this way. Mention of the leucocytosis, which is characteristic of suppuration or abscess, has already been made (§ 398), but in addition the hæmoglobin and the red cells may be deficient.

XIII. **Post-febrile Anæmia,** associated nearly always with weakness and emaciation, may ensue after rheumatic fever, enteric fever, varicella, malaria, and indeed after any of the acute specific fevers.

XIV. Finally, in various conditions referred to in Group II. below (Emaciation), or Group III. (Debility), pallor may be the symptom which first attracts our notice, for these three important symptoms are so often associated. Early myxœdema (§ 419), is one of these, and

the puffiness of the eyes, the failing memory, loss of hair, and bodily weakness may for a time escape observation, or be attributed to other causes.[1]

Rarer Causes of Anæmia.

The patient is **pale** *and anæmic ; there is* ENLARGEMENT OF THE SPLEEN, *or the* LYMPHATIC GLANDS, *or both, and characteristic* CHANGES IN THE BLOOD. The disease is probably LEUKÆMIA, HODGKIN'S DISEASE, SPLENIC ANÆMIA, or a SEQUELA of MALARIA.

§ **407.** I. **Leukæmia** or **Leucocythæmia** is a comparatively rare disease characterised by progressive anæmia, a large and persistent increase of white corpuscles, a slight diminution of the red cells, accompanied by enlargement of the spleen. There are two varieties of the disease : (i.) The *spleno-medullary* or myelogenous type (splenic leukæmia), and (ii.) *lymphatic* leukæmia. In the former the chief changes are in the spleen (which is enlarged) and the bone-marrow, and the blood contains myelocytes which are derived from the bone-marrow. In the lymphatic variety the chief changes are in the lymphatic glands, which are enlarged, and the blood contains an excess of lymphocytes derived from those glands.

The *Symptoms* are (1) anæmia, with its concomitant symptoms, such as breathlessness, enfeeblement, etc., which are generally the first symptoms to be complained of. In the early stages the pallor may not be very noticeable, but anæmia and the characteristic blood changes continue throughout, and in the advanced stages there is no disease which gives to a patient such an extremely pallid appearance. (2) Enlargement of the *spleen* occurs early in the disease, and the patient may even first come under our care complaining of abdominal swelling. The enlargement is less in the lymphatic than in the spleno-medullary forms of the malady, when it may extend as far as the umbilicus, or even into the pelvis, and weigh as much as 15 pounds. The enlargement is uniform and the consistence hard. It is due to a parenchymatous increase of all the tissues, though especially of the Malpighian bodies. The liver also undergoes moderate enlargement in about two-thirds of the cases. The disease is accompanied by gastric disturbance, diarrhœa, and vomiting at irregular intervals. (3) In about one-third of the cases the lymphatic glands are enlarged ; this is especially a feature of the *lymphatic* variety of the disease. In lymphatic leukæmia the thymus may also be enlarged, leading to dulness over the manubrium, and in some cases all the adenoid tissue of the body is increased. (4) *Hæmorrhages* may occur from time to time from the nose, stomach, or elsewhere, and into the skin or retina. (5) *Pyrexia* of an irregular type occurs in three-fourths of the cases, especially towards the end. (6) The urine generally shows an excess of *uric acid*. (7) In the spleno-medullary form tender areas may be detected along the long bones, due to thinning from local marrow overgrowth, and rarely actual tumours may be felt.

(8) The *blood changes* are pathognomonic, and are different in the spleno-medullary and the lymphatic varieties of the disease. In *both varieties* there is (i.) an increase in the white cells—one to five or ten red cells, instead of one to 500 as in normal blood, and (ii.) a slight diminution in the number of the red cells. The blood changes in the *spleno-medullary* form of leukæmia are (i.) the presence of large mononucleated cells with neutrophil granules, which do not occur in normal blood nor in other forms of leucocytosis, and are regarded as special to this type of leukæmia. They are known as " marrow cells " or myelocytes, and constitute about 30 per cent. of the white corpuscles present ; (ii.) the eosinophile cells are increased in number, and may be larger than normal ; (iii.) the normal polynuclear leucocytes may be slightly increased ; (iv.) " mast cells " with coarse basophilic granules are present ; (v.) nucleated red cells are common, and megaloblasts are

[1] Cases of this kind are referred to by Dr. Charles Chapman, the *Lancet*, September 30, 1899, p. 876.

found sometimes; (vi.) the blood platelets are often increased. The myelocytes and normal polynuclear cells form the bulk of the leucocytes present. In severe cases the blood may resemble pus under the microscope; the proportion of the white to the red corpuscles being 1 to 10, and sometimes even 1 to 1. In blood slides Charcot's crystals separate out, and the hæmoglobin crystallises readily. The blood changes in the *lymphatic variety* are: (i.) The small mononuclear cells (the lymphocytes) are increased, and the most characteristic feature of the disease is that they form the bulk (sometimes as much as 99 per cent.) of the leucocytes present. In some cases the large instead of the small mononuclear cells are found to be increased. (ii.) The eosinophil and polynuclear cells are normal in number; (iii.) there are rarely any nucleated red corpuscles, and myelocytes are generally absent; and (iv.) the disproportion between the red and white cells is never so great as in the spleno-medullary variety. (See Plate III.)

The *Diagnosis* is not difficult as a rule. Splenic leukæmia is the only form of anæmia accompanied by enlarged spleen and the changes in the blood described above. Lymphatic leukæmia clinically resembles Hodgkin's disease (*q.v.*), but has different blood changes.

TABLE XXV.—THE DIAGNOSIS OF LEUKÆMIA.

	Spleen.	*Lymphatic Glands.*	*Leading Blood Changes.*
Spleno-medullary Leukæmia.	Spleen greatly increased.	Not usually enlarged.	Leucocytosis marked, chiefly due to myelocytosis. Eosinophil cells also increased. Red cells markedly deficient.
Lymphatic Leukæmia.	Spleen moderately increased.	Moderately enlarged.	Leucocytosis marked, due to increase of lymphocytes. Red cells show progressive anæmia.
Hodgkin's Disease.	Spleen slightly enlarged in ⅓ of the cases.	Greatly enlarged.	Slight leucocytosis in some cases; moderate anæmia.
Splenic Anæmia.	Spleen greatly enlarged.	Not enlarged.	Leucopenia; marked anæmia.

Acute Leukæmia or *Lymphocythæmia* is a subvariety of the lymphatic form.[1] There is (i.) rapidly progressive anæmia and multiple hæmorrhages; (ii.) the glands are usually, but not necessarily enlarged; (iii.) the blood shows a relative increase of small or large lymphocytes, although the total number of white cells may not be increased; in fact, there may be leucopenia. The red blood corpuscles may be decreased to between one and two millions, or even lower, and the colour index may be either above or below normal. Besides the enlargement of the liver and spleen, the kidneys may form large abdominal tumours. Sometimes necrosis or ulceration of the mouth or tonsils occurs.[2]

The *Prognosis* of leukæmia is very grave. The disease is progressive in character, though the course is irregular, with apparent restorations to health followed by relapses. Its average duration is two to three years, but its course may extend over five to ten years, when it is of the chronic type. The acute cases especially occur in children, and the disease then runs a rapidly fatal course in a few months or less. The complications are (i.) ascites and general dropsy due to heart failure; (ii.) intracranial hæmorrhages, or profuse hæmorrhages in other situations; and (iii.) severe diarrhœa and acute peritonitis.

Etiology.—The disease is twice as frequent in males as in females. It usually comes on between the ages of thirty and fifty, but may occur at any age. Syphilis, rickets, rheumatism, traumatism, and heredity have been supposed to play a part in the causation.

Treatment is of little avail. In addition to general hygienic measures arsenic is the remedy usually employed, though it does little except hold the disease in

[1] Forbes and Langmead, Proc. Roy. Med. Soc., May, 1908.

[2] Rose Bradford and Batty Shaw, Trans. Med. Chir. Soc., vol. lxxxi., p. 343.

check for a time. X rays have lately been employed with some measure of success; in many cases they have led to a diminution in the size of the spleen, and a decrease of the white blood-cells. Cod-liver oil, quinine, and other tonics have been employed without much success. Oxygen inhalations have been suggested. Splenectomy is not justifiable.

Chloroma or " green cancer " is an anatomical term applied to a semi-malignant growth met with in the periosteum of the head and face, and other places where lymphoid tissue is found. It is a rare disease, similar in its clinical symptoms to lymphatic leukæmia and lymphosarcoma, and is supposed to be pathologically a connecting link between them.

Dr. R. Hutchison[1] says: " It would seem we have in lymphosarcoma, in chloroma, and in lymphatic leukæmia three closely-allied processes exhibiting a descending scale of malignancy, but presenting many features in common, and in the case of chloroma and lymphatic leukæmia at all events possibly tending to pass into each other."

§ **408.** II. **Hodgkin's Disease** (Synonym: Lymphadenoma) is a disease characterised by anæmia, progressive hyperplasia of the lymphatic glands, and sometimes lymphoid growths in the liver, spleen, kidney, and other organs. There are two forms of the glandular enlargement, soft and hard. In the former the glands are soft in consistence and somewhat enlarged; there is a proliferation of the endothelial cells, dilatation of bloodvessels and lymph-sinuses, while masses of lymphocytes crowd the lymph sinuses, and large multinuclear cells also occur. In the hard variety, which is usually a more advanced form, the glands are much enlarged and hard in consistence, and there is a great increase in the fibrous tissue which takes place at the expense of the other cells.

Symptoms.—(1) Sometimes debility and anæmia are the first symptoms, but more frequently enlargement of the lymphatic glands of the neck, axillæ, or groins first attract attention. The enlargement of the cervical or other single group of glands may precede that of any other glands for a considerable time, even for a few years, but usaully the extension to other glands is more rapid. In chronic forms of Hodgkin's disease the glands are hard, separate, and movable under the skin; but in the acuter forms the glands feel soft, and great enlargement and suppuration may occur. For accurate diagnosis a gland should be excised under local anæsthesia, and examined microscopically.[2] In Hodgkin's disease the glands readily become the seat of secondary infection; hence probably the variation in the clinical symptoms and course. (2) Pressure effects occur when the deep glands—*e.g.*, in the thorax—become involved, and occasionally they are the first to enlarge. The pressure symptoms are described in § 54. Bronzing of the skin may arise as a consequence of pressure on the solar plexus. (3) Irregular paroxysms of intermittent pyrexia occur at intervals of a few days or a few weeks, and these attacks coincide with a paroxysmal enlargement of the lymphatic glands. (4) The spleen enlarges as the glands enlarge, but the enlargement is never very great; usually the edge can just be felt below the costal margin. The liver also enlarges in most cases. (5) The blood changes are not characteristic; they consist chiefly of diminution of the hæmoglobin and the number of red corpuscles with poikilocytosis in the later stages. Slight leucocytosis may occur towards the end. (6) The constitutional symptoms, anæmia, and languor, increase, and in the later stage may become extreme, with the concomitant symptoms of emaciation, and a marked tendency to hæmorrhages (as is usual in all profound anæmiæ).

Diagnosis.—Clinically, Hodgkin's disease and the *lymphatic variety of leukæmia* are alike, but an examination of the blood at once reveals the difference. The blood in Hodgkin's disease shows only a diminution of the hæmoglobin and the red cells, and in the later stages only a slight degree of polynuclear leucocytosis;

[1] The *Lancet*, May 21, 1904, p. 1408.

[2] Dr. Dorothy Reed has shown that the glands show characteristic changes: (i.) proliferation of the endothelial cells, and excess of lymphocytes filling the lymph sinuses; and, later, (ii.) excess of fibrous tissue. (" Johns Hopkins Hospital Reports," 1902, vol. x., p. 133.)

in leukæmia there is always a *marked and characteristic increase in the number of lymphocytes* throughout, and usually an increase in the total number of leucocytes. *Lymphosarcoma* has been considered on the Continent to be akin to Hodgkin's disease. In lymphosarcoma there is a degree of lymphocytosis present in the blood, but the growth, though primarily involving the lymphatic glands, invades the surrounding tissues, and thus reveals its malignancy. *Lymphoma* cannot be diagnosed from the chronic cases of Hodgkin's disease, when only one group of glands is enlarged, and the diagnosis from *tuberculous adenitis* is often difficult. Tubercle is more common at an earlier age ; the glands tend to be matted together, and to caseate or suppurate. The tuberculin tests, if positive, prove the presence of tubercle, but they cannot decide whether the morbid process in a given case was tuberculous at the onset. *Syphilitic* glandular enlargement is preceded by the appearance of a chancre ; the glands are very hard, and tend to disappear rather than to spread.

Prognosis.—Hodgkin's disease usually runs a slow chronic course, months, or even years elapsing before extension from one group of glands to another. On the other hand, the disease may run an acute course, all the glands enlarging within a few months. Cases are reported to have recovered or improved or remained stationary for a long period without special treatment, and chronic cases have entirely recovered under treatment. In severe cases the anæmia and emaciation are marked, and death occurs from exhaustion, or with delirium and coma. Complications such as pneumonia, pleural effusion, pressure on the bronchi or trachea may also cause death. Difficulty in swallowing and deafness may arise from overgrowth of the adenoid tissue in the pharynx or thorax, and lymphoid growths in the walls of the stomach lead to dyspepsia and vomiting.

Of the *Etiology* little is known. The disease usually arises in the first half of life. Among Sir William Gowers' 100 cases 30 were under twenty years old, 34 between twenty and forty, and 36 over forty. It is three times as frequent in men as in women. An infective origin has been suspected. In some cases the disease has been attributed to an attenuated tuberculous infection. In other cases a local cause of irritation, such as nasal catarrh or a bad tooth, has led to a local enlargement of a group of glands which was followed later by a generalised lymphadenomatous enlargement.

Treatment.—Local chronic groups of glands should be removed. Recoveries have been reported with the use of arsenic. It should be administered for months at a time in gradually increasing doses until tolerance is reached. Iodides are useless. Phosphorus should be tried if arsenic is ill borne. Tonics, cod-liver oil, and all other means to keep up the patient's strength are useful.

§ **409.** III. **Splenic Anæmia** is a rare disease, the characters of which have been thus summarised by Dr. H. D. Rolleston[1] : (1) Splenic enlargement which cannot be connected with any recognised cause ; (2) absence of any enlargement of the lymphatic glands ; (3) anæmia of a type midway between secondary anæmia and chlorosis ; (4) leucopenia, or at most no leucocytosis ; (5) an extremely prolonged course lasting years ; and (6) a tendency to hæmorrhages, especially gastro-intestinal, from time to time. The patient may come under observation during the *early* stage of the disease when anæmia with its concurrent symptoms is complained of. In the *second* stage there is enlargement of the spleen, accompanied by attacks of pain during periods of enlargement of the organ. The spleen in this disease attains an enormous size, almost as great as that which occurs in leukæmia. The enlargement is due to fibrosis of the organ, with atrophy of the Malpighian bodies. In some cases the splenic enlargement appears to precede the anæmia, and the patient may not seek advice until his spleen has reached the umbilicus. As the disease progresses there is loss of strength without emaciation, accompanied by gastric disturbance and a tendency to hæmorrhages. Sometimes there is moderate enlargement of the liver. Pyrexia is present during the active stages of the disease. The chief distinction between this disease and splenic leukæmia is in the blood changes, and particularly in the absence of leucocytosis in splenic anæmia. The

[1] The *Lancet*, August 8, 1903, p. 401.

blood shows a diminution of the number of the red cells, with a diminution of the hæmoglobin corresponding to the chlorotic type of blood. Poikilocytosis may be present. In the *third* stage of the disease all the symptoms are aggravated, and in a few cases of the disease the so-called " Banti's disease " supervenes, with fatal termination. The name " Banti's disease " has been given to a group of symptoms comprising cirrhosis of the liver, jaundice, and ascites, consequent on splenic anæmia, as described above.[1]

The *Diagnosis* from most forms of *secondary anæmia* is effected by the great enlargement of the spleen, and from *leukæmia* by the characteristic blood changes in that disease (§ 407). *Pernicious anæmia* is rarely associated with an enlarged spleen, but difficulty may arise when the blood changes in a severe case of splenic anæmia resemble those of pernicious anæmia. The chief practical difficulty lies in diagnosing the disease from *cirrhosis of the liver* with accompanying enlargement of the spleen. If the red corpuscles and the hæmoglobin rapidly increase under treatment by iron, it is improbable that the condition is due to splenic anæmia. Banti's disease may be almost impossible to diagnose from cirrhosis of the liver unless a history including the blood changes of previous years can be obtained. Many cases regarded during life as splenic anæmia are found after death to be due to visceral syphilis, cirrhosis of the liver, or thrombosis of the portal vein.

Prognosis.—The disease is a chronic progressive disorder. It used to be said that death occurred in six months to two years, but it is now known that cases may live ten, twelve or even twenty years after the commencement of the disease. Death takes place by asthenia, occasionally by syncope or hæmorrhage.

Etiology.—Men are more often affected by this disease than women, and it occurs mostly in adult life. The cause is unknown.

The *Treatment* is the same as that of other anæmias. Arsenic is the most efficacious drug. Splenectomy may prolong life.

The patient is very **pale** *and anæmic, and there are or have been* SORENESS *of the* GUMS, PURPURIC SPOTS, *or other hæmorrhagic lesions.* The disease is probably SCORBUTUS.

§ **410.** IV. **Scurvy** (Synonym : Scorbutus) is a constitutional disease due to deficiency of fresh food, animal and vegetable, attended by extreme debility and anæmia, sponginess of the gums, and hæmorrhages. The disease is only rarely met with now, but used to be the scourge of the British navy, until the introduction of lime-juice as a prophylactic, which now all ships are obliged to carry.

The *Symptoms* start insidiously, and consist of (i.) progressive debility and anæmia, with mental depression and headache, but no pyrexia. Palpitation, hæmic murmurs, syncopal attacks, and other symptoms of anæmia develop. Pains in the back and limbs are usually complained of early. The urine is scanty and highly acid, and may contain albumen. (ii.) The gums become spongy, swollen, and bleed readily. Sloughing may follow, and the teeth become loosened ; the breath is very offensive. Constipation is usual, but diarrhœa with blood may occur later on. (iii.) A characteristic eruption appears, consisting of purpuric spots and swellings of brawny consistence found about the flexures of the joints, especially the popliteal space. These swellings are due to hæmorrhages into or beneath the skin ; if the former, they are purple ; but if beneath the skin, the colour may be pale. Swellings may also occur under the periosteum of the bones of the legs. Epistaxis often occurs, but hæmorrhage from other mucous membranes is not common except in severe cases. (iv.) Death may ensue either from syncope, asthenia, or complications. Among the latter may be mentioned sanguineous effusion into the pleura or meninges, pneumonia, and sloughing of the skin.

Diagnosis.—The diagnosis of scurvy from other causes of *purpuric* eruption is afforded by the condition of the gums, and the hard brawny swellings, which are

[1] Murrell, The *Lancet*, April 26, 1902, p. 1177.

peculiar to scurvy, and also by the degree of prostration present. Slighter cases are, however, very difficult to diagnose, as similar symptoms may be seen with purpura. Rapid improvement with suitable diet favours a diagnosis of scurvy. *Syphilitic nodes* on the tibiæ accompanied by cachexia should be carefully differentiated from scurvy, because mercury is so injurious in the latter disease. In *pernicious anæmia* there is a longer history, and blood changes are present.

Prognosis.—As a rule this is good if the cause be discovered, and removed. Unfavourable symptoms are severe dyspnœa, syncope, scanty urine, and elevations of temperature. Convulsions, hemiplegia, and other cerebral symptoms follow intracranial hæmorrhage. The outlook is grave when dysentery complicates the disease. Necrosis of the jaw or other bones is rare.

Etiology.—The chief cause is an absence of fresh vegetable from the dietary, though this will not produce scurvy if fresh meat is available. It is the combination of salted or tainted meat and the absence of fresh vegetable which leads to scurvy. It has occurred in well-to-do women who live on tea and bread-and-butter. It appears that organic salts are more unstable than others, and thus potassium, for example, if combined with an organic salt, will more readily reach the tissues and there be converted into a carbonate, than if it be taken in the form of a chloride or a phosphate, which are more stable salts. In fruits and vegetables which have organic salts, therefore, the mineral constituents more easily reach the tissues than in animal foods, which have inorganic salts. Sir A. E. Wright has found a deficiency of alkalinity of the blood in scorbutic patients, and believes that the disease is due to a too acid dietary; but Professor Axel Hölst[1] has shown that rabbits fed on a scurvy-producing diet can only be kept free from scurvy if given *fresh* vegetable, not if the vegetable be previously dried, even if sodium bicarbonate be added to it.

Treatment consists in giving a liberal diet, with fresh vegetables, the juice of two lemons daily, fruits, light wines, claret, tea, and cider. Scraped raw meat and milk are good for severe cases. For the mouth give a gargle of potassium chlorate or Condy's fluid, or pencil the gums with a strong solution of silver nitrate. Bismuth and opium are needed for diarrhœa, and foods which would irritate such complications as dysentery should be avoided.

The patient is **pale** *and liable to* UNCONTROLLABLE BLEEDINGS, *from little or no cause.* The morbid condition is probably HÆMOPHILIA.

§ **411.** V. **Hæmophilia** is a hereditary disease characterised by a constitutional tendency to uncontrollable hæmorrhage without sufficient cause.

The *Symptoms* are divided into three sets: (1) Hæmorrhages from mucous membranes, or after some slight injury, from the skin. Nothing abnormal may be noted in a subject of hæmophilia until he has a tooth extracted or a trifling abrasion, when uncontrollable bleeding due to capillary oozing sets in, and lasts for hours or days. When the bleeding occurs from a mucous surface, large blood tumours may form as the blood coagulates. (2) Interstitial hæmorrhages occur spontaneously or after injury in the form of petechiæ or hæmatomata. (3) Affections of the joints, especially the knees and elbows, are met with, and three stages are described: (i.) Recurrent hæmarthroses or effusions of blood into the joints, of acute onset, sometimes attended by pyrexia; (ii.) reactionary synovitis; and (iii.) cicatrisation which may lead to permanent deformity.

Diagnosis.—A single severe hæmorrhage does not warrant a diagnosis of hæmophilia, but recurrent hæmorrhages with slight cause are characteristic. The family history of a tendency to bleeding is important. The joint affections are diagnosed by the presence or history of other signs of hæmophilia.

Prognosis.—The disease usually becomes evident during the first few years of life, and as a rule tends to be less troublesome as life advances, disappearing about thirty or forty. Great anæmia occurs from excessive bleeding, and life has been ost after trivial operations such as the extraction of a tooth or circumcision.

[1] *Brit. Med. Journ.*, October 31, 1908, p. 1365.

Etiology.—Hæmophilia occurs in families for generations. It is met with in males, in the proportion of 13 to 1 female, but the diathesis is transmitted through the female who herself may remain unaffected. The cause of the condition is unknown. The bloodvessels in some cases have been found to be thin, and Sir A. E. Wright has found the blood coagulability is retarded as long as twenty to forty-five minutes.

Treatment.—Males in a bleeder's family should be guarded from any injury or operation. The daughters should not marry, for though they themselves are not endangered by parturition, their sons will probably be bleeders, and their daughters will pass on the same tendency in turn to their offspring. When bleeding occurs, rest is essential, and styptics are applied. Internally adrenalin chloride, calcium chloride, iron perchloride, and ergot have done good.

VI. **Addison's Disease** and other maladies mentioned in **Groups II.** and **III.** (below) occasionally come under our notice for anæmia.

There is **pallor** *of the skin and the patient has* BEEN ABROAD. Inquiry should be made for MALARIA, CHRONIC DYSENTERY, PARASITES, or other TROPICAL DISEASES.

VII. Various **tropical diseases** and other pyrexial conditions rarely, if ever, seen in England, are attended by intense anæmia.

(*a*) *Malarial anæmia* is usually accompanied by pigment changes around the eyes and other parts of the body. The history here, of course, is our first clue to diagnosis. The earthy pallor and the enlargement of the spleen (ague cake) are very characteristic.

(*b*) *Dengue, beri-beri, dysentery,* and other fevers unknown in this country are accompanied and followed by anæmia. In the two first named there is much general weakness and some emaciation, and in the third diarrhœa.

(*c*) In Egypt and other countries cases which used to be considered as idiopathic anæmia are now known to be due to *intestinal* and *other parasites.* The *ankylostomum duodenale* and *bothriocephalus latus* may be recognised by the presence in the fæces of ova or segments, and when the patient recovers under large doses of thymol it is probable that he has had ankylostomum duodenale. A widespread epidemic of ankylostomiasis occurring among the men employed in the Dolcoath mines, Cornwall, has been reported of the same kind as that occurring during the construction of the St. Gothard Tunnel in 1880. *Bilharzia hæmatobia,* endemic in Egypt and elsewhere, causes anæmia and hæmaturia (§ 300). The *filaria sanguinis hominis* and *strongylus gigas* also give rise to anæmia.

Distoma Pulmonale (Synonym : Distoma Ringeri) is a parasite found in the lungs, liver, testes, peritoneum, and brain. It is met with in Formosa (being present, according to Manson, in 15 per cent. of the inhabitants), China, and other tropical countries, where it gives rise to endemic hæmoptysis. It measures 8 to 10 mm. in length by 4 to 6 mm. in breadth. It is reddish-brown in colour, oval in form. In the lung, its favourite habitat, it gives rise to chronic cough without physical signs, rusty sputum, and irregular attacks of hæmoptysis, accompanied by intense anæmia.

§ 412. Anæmia in Children.—All the forms of anæmia above described may occur in children under fourteen, and are produced by the same causes which affect adults, but they occur in a very different order of frequency and present certain marked differences. (*a*) The spleen tends to become enlarged in all forms of anæmia in children, but is markedly so in (1) splenic anæmia infantum, (2) lymphatic and myelogenous leukæmia, and (3) Hodgkin's disease. (*b*) The blood changes also differ considerably from the blood changes met with in the same diseases in adults. In infancy and childhood slight causes lead to blood alterations

of a marked type which, if occurring in an adult, would signify severe disease.[1] (*c*) In secondary anæmias in children, as in adults, a diminution of the hæmoglobin is the earliest change, but there are important differences. In childhood (1) the number of red corpuscles is reduced at a comparatively early stage and new ones enter the blood in a half-formed condition—poikilocytosis and nucleated red cells—and (2) leucocytosis occurs more readily, chiefly of the mononuclear cells (small and large lymphocytes) owing to the activity of the adenoid tissue in children. Adenoid tissue is very active and plentiful in children, and hence in childhood an increase of the lymphocytes (which are supposed to come from the adenoid tissue and lymphatic glands) is more common than an increase of the polynuclear neutrophil and other white cells (which are supposed to come from the bone-marrow). Thus in children among the leucocytic diseases lymphadenoma, lymphatic leukæmia, and lymphosarcoma are more common than spleno-myelogenous leukæmia.

I. Primary Anæmias.—*Chlorosis* is hard to diagnose with certainty in a child, but it may occur. *Pernicious anæmia* in childhood is so rare that some authorities deny its existence. But blood changes with poikilocytosis and nucleated red cells such as occur in the pernicious anæmia of adults may be met with in the secondary anæmias of children more readily than in adults.

II. The chief causes of Secondary Anæmia in children are *defective nutrition*, the *acute specific fevers*, *acute rheumatism*, *prolonged suppuration*, *syphilis*, *tuberculosis*, and *chronic diarrhœa*. Secondary anæmia frequently occurs in children who have had deficient proteid food. It is met with therefore in children who have been suckled too long, or have had only milk food at an age when they should have had proteid foods containing iron.

In addition to the foregoing, there are three anæmias special to children—III. Infantile Scurvy; IV. Splenic Anæmia Infantum; V. Congenital Anæmia.

§ **413.** III. **Infantile Scurvy** (Synonyms: Scurvy Rickets, Barlow's Disease) used to be considered a variety of rickets, but it is now known that it need not necessarily be associated with rickets.

Symptoms.—(1) The onset may be gradual. The child is noticed to become pale and in late stages profoundly anæmic. Muscular weakness becomes marked, but emaciation may be absent. The child cries when washed or dressed, screams if the legs are touched, and is very still when at rest. (2) Very soon an ill-defined swelling which visibly increases is seen along the tibia, just above the ankle. The swelling is not necessarily symmetrical, and is due to subperiosteal extravasation of blood. The legs appear as if paralysed, because the child keeps them everted and motionless, dreading the pain caused by movement.

[1] See Dr. Robert Hutchison, "Disorders of the Blood in Early Life," the *Lancet*, May 7, 1904.

Sometimes the femurs also are affected, and there may be œdema of the dorsum of the foot. The arms and scapulæ are next affected; the ribs, skull, and face rarely so. The joints are free. (3) Swellings may occur in the muscles, resembling abscesses, but there is no redness or fluctuation. (4) Sponginess of the gums develops, and petechiæ and internal hæmorrhages may occur as in adult scurvy. (5) Other symptoms are albuminuria and hæmaturia, and proptosis from hæmorrhage into the orbital periosteum. The *temperature* is normal except after large or recent hæmorrhages, when it may rise to 100° or 102° for a few days.

Diagnosis.—Infantile scurvy may be mistaken for *rheumatism*, but whereas in rheumatism the joints are affected, in scurvy they are free. *Infantile paralysis* is accompanied by no swelling or tenderness of the limbs. With *abscesses*, *nephritis*, and *stomatitis* there are no signs of scurvy. In *syphilitic* pseudo-paralysis crepitation and pain on moving the limb occurs, due to separation of the cartilage from the diaphysis.

Prognosis.—On the whole this is favourable. Under treatment recovery is rapid, and the child may be well in three weeks. If the patient is seen at a late stage, or if from failure to diagnose the disease the diet is not altered, death occurs from syncope or complications such as diarrhœa, bronchitis, and pneumonia, or any of the acute specific fevers.

Etiology.—The disease affects children of six to eighteen months usually, and is due entirely to defective diet—*i.e.*, absence of fresh food. It occurs particularly in infants fed only with proprietary foods, boiled, sterilised, or condensed milk.

Treatment.—The prophylactic treatment consists in the observance of a few simple dietetic rules. Fresh unboiled milk must be used, or the milk must only be scalded (brought to the boiling-point for a second). Peptonised milk and artificial foods should never be used for longer than a few weeks at a time. Remedial treatment consists in the administration of fresh milk. Steamed potato, rubbed through a sieve and beaten up with milk to the consistence of thick cream, should be given in doses of 1 drachm to 1 ounce with each bottle. A few ounces of beef-tea, in which carrots have been boiled, and then strained out, may be taken twice daily. Raw-meat juice is excellent. For children of a year old, grape, orange, lemon, and baked apple juice, with potato pulp and raw-meat juice should be given. *Local* treatment consists in wrapping the limb in cotton wool and preventing movement.

§ 414. IV. Splenic Anæmia of Children (Synonyms: Anæmia Infantum Pseudo-leukæmica, von Jaksch's Disease, Anæmia Splenica Infettiva dei Bambini) occurs in children from six months to two years of age, and is characterised by anæmia and leucocytosis and enlargement of the spleen. The splenic anæmia of adults is not the same disease.

Symptoms.—(i.) Pallor due to anæmia of insidious onset, sometimes preceded, sometimes followed, by (ii.) enlargement of the spleen, which may attain a great size. Attacks of pain may occur, due to perisplenitis. (iii.) The liver is moderately enlarged, and in some cases the glands also. (iv.) There is irregular pyrexia and

gastro-intestinal disturbance. (v.) The patient may remain plump throughout, but in severe cases usually becomes greatly emaciated. (vi.) In serious cases hæmorrhages occur from the mucous membranes and into the skin. (vii.) The blood changes are characteristic—the hæmoglobin is diminished, the number of the red corpuscles is reduced usually to two or three million, and a slight degree of poikilocytosis is present, together with nucleated red corpuscles. Leucocytosis may be absent in the earlier stages, but always marked in the later stages. The polymorphous character of the leucocytosis is a diagnostic feature. Myelocytes, lymphocytes, large mononuclear cells, and many transitional forms are seen, the transitional forms rendering a differential count impossible.

The *Diagnosis* is difficult only in the early stages. In both *syphilis* and *rickets* we often meet with anæmia, enlargement of the spleen and of the liver ; but the spleen never attains the same size, and the blood changes are never so marked, as in splenic anæmia of infants. In children severe *secondary anæmia* may present leucocytosis, but the polymorphism of the leucocytes is not found. *Splenic leukæmia* rarely, if ever, occurs in children, and the leucocytosis has different features. The diagnosis of splenic anæmia of children depends on different features at different stages ; and it would appear, from the numerous synonyms above mentioned, that this disease has been described by various observers, under different names, according to the stage under observation. In the early stage the changes in the red corpuscles are prominent, resembling those in severe anæmia. In the later stages the leucocytosis becomes more noticeable ; hence the name " pseudo-leukæmia."

The *Prognosis* is good. The course is short, and recovery usually complete, but cases relapse under bad hygienic conditions. Hæmorrhages and petechial eruptions are serious symptoms. The lower the number of red, and the higher the number of white corpuscles the graver is the prognosis. Death occurs from exhaustion or intercurrent diseases.

The *Etiology* is obscure ; the disease is supposed to be due to some specific gastro-intestinal toxin. It is often associated with rickets. No connection has been traced between this disease and syphilis.

The *Treatment* consists in remedying the hygienic conditions. Fresh air is essential, and good food, such as yolk of egg, raw-meat juice, potatoes, and bone-marrow should be given. Of drugs, iron and arsenic are the best ; cod-liver oil and malt are useful adjuncts.

V. **Congenital Anæmia** occurs occasionally. The causes are obscure. No iron is obtained during the period of suckling, but a child is born with a store of iron in the liver (Bunge), and it may be assumed that this store of iron has for some reason been deficient. Sometimes there is a history of icterus after birth, as if there had been an abnormal amount of blood destruction at that time. In other cases the bone-marrow has been found to be defective. The condition is very apt to be confused with congenital syphilis.

GROUP II. EMACIATION.

Wasting is a common sequence of nearly all acute and many chronic diseases, but when it is the leading or only symptom the following morbid conditions should be borne in mind. The fallacies have been referred to in § 393.

I. Malignant disease.

II. Defective feeding and digestive disorders, colitis, intestinal adhesions or stenosis, and other obscure intestinal conditions.

III. Tuberculosis, diabetes mellitus, diabetes insipidus, chronic Bright's disease, syphilis, and other diseases of Groups I. and III.

IV. Diseases of the pancreas.

V. Various nerve conditions.

Marasmus in children may be caused by defective feeding, diarrhœa, constipation, persistent vomiting, hereditary syphilis, rickets, tabes mesenterica, and pulmonary tuberculosis.

Emaciation in the last third of life is suspicious of carcinoma ; in the middle third of life, diseases in II. and III. above ; and in the first third, tuberculosis.

§ **415.** I. **Malignant Disease** (Carcinoma and Sarcoma) is a cause of emaciation which should be ever present in the mind when the patient is at or past middle age. There are two anatomical varieties—carcinoma (cancer proper) and sarcoma. The essential feature common to them both is that they tend to recur after removal, to invade the parts around, and to reproduce themselves in distant parts. This is the clinical meaning of the word malignant. Certain localities are much more prone to primary deposits than others, and in cases of latent malignant disease it is important to remember the places in which primary carcinoma and sarcoma may occur. The commonest seats for *primary carcinoma* are the skin around the mucous orifices, the tongue, œsophagus, stomach, colon, rectum, mamma, uterus, penis, and testis. In these regions secondary carcinoma is almost unknown, unless by direct infiltrating invasion. *Sarcomata* are rarely found primarily in these situations, but are prone to start in the peritoneum, the fascia, the bones, the corium, ovary, kidney in children, brain, spinal cord, retina, and in the fibrous structures of the muscles, breast, and testicle. Different varieties vary considerably in their malignancy and rate of growth. The most rapidly growing of all is the round-celled sarcoma, the next in order being the spindle-celled sarcoma, the myeloid sarcoma (*e.g.*, epulis) being of slow growth, and rarely producing secondary tumours. Melanotic sarcoma has a great tendency to reproduction in distant parts. Among the carcinomata the softer and encephaloid varieties are more rapid and malignant than the harder and scirrhous varieties. Any tissue or organ of the body may be involved either by continuity or along the lymphatics (in the case of carcinoma) or by the blood-stream (in sarcoma).

Symptoms.—The symptoms of malignant disease necessarily vary with the situation, and under the heading of tumours or growths the diagnosis has been previously dealt with (*e.g.*, the abdomen, § 188, the chest, §§ 54 and 99). We are here concerned with a general review of the symptoms. (1) There is loss of weight quite early in the disease, sometimes long before any local signs can be detected. This is accompanied by a typical cachexia—*i.e.*, an appearance of illness, in which the skin assumes an ashy or sallow hue. The sallowness of the skin may be so marked as to be with difficulty distinguishable from jaundice, or even Addison's disease. (2) The age of the patient is advanced in carcinoma, young in sarcoma. The four classical signs of cancer are pain, swelling, offensive discharge, and hæmorrhage. (3) Pain at the seat of growth is often complained of, especially in rapidly-growing varieties, or when they occur in tense parts. (4) In accessible situations

a thickening, swelling, or tumour may be detected, which is usually hard, nodular, and apt to fix and infiltrate the surrounding parts. Some sarcomata are soft and pulsating. (5) Whenever the growth involves a mucous or epidermal surface there is an offensive pink or sero-sanguineous discharge—*e.g.*, from the vagina. (6) In like manner hæmorrhage may occur, and take the form either of metrorrhagia, coffee-ground vomiting, or melæna ; and when the disease involves the pleura or peritoneum the *effused fluid will be blood-stained.* (7) In carcinoma the neighbouring lymphatic glands become enlarged and palpable. (8) The rate of growth is rapid, though it varies widely in different forms and localities. Scirrhous infiltration of orifices may only reach the thickness of half an inch in six to twelve months, and the patient may live two years ; but a round-celled sarcoma will reach the size of a hen's egg in a month or two and kill in six.

Diagnosis.—Malignant disease may have to be diagnosed from all the other conditions which give rise to emaciation. A malignant nodule may have to be diagnosed from syphilitic gumma (compare, for instance, syphilis of the tongue, skin, etc.), but the latter is usually attended by less pain and constitutional disturbance, and is amenable to anti-syphilitic treatment. Cases of sarcoma are in rare instances attended by pyrexia, four instances being recorded by Hilton Fagge and Pye Smith in their classical work on medicine.[1]

The *Prognosis*, if the case is untreated, is always of the gravest kind, the course rarely lasting more than one, or, at the outside, two years. A few cases of undoubted malignant disease have undergone spontaneous involution.[2] The prognosis largely depends upon the stage at which the true nature of the case is detected. On this depends very largely both the prospect of arrest or removal. In general terms the prognosis also depends on (1) the position and accessibility of the growth, how far vital structures are involved, and whether it is on or near the surface ; (2) the structure of the tumour (*vide supra*) ; and (3) the age of the patient, to some extent, for growth is more rapid in the young.

Etiology.—(1) In carcinoma the age of the patient is nearly always over forty, though I have seen cases of scirrhus of the pylorus in persons aged twenty-eight and thirty-three. Sarcoma, on the other hand, may affect children or adults of any age. Sarcoma is the malignant growth of the kidney that is specially met with under the age of nine more often than at any other time of life. (2) Sex has not much influence, but the great frequency with which cancer arises in the mammæ and uterus gives to statistics a marked bias towards the female sex. (3) Heredity has always been regarded by most physicians as a predisposing cause of malignant disease, though it has been disputed by others.

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[1] Fagge and Pye Smith's "Principles and Practice of Medicine," fourth edition, pp. 88, 89.

[2] See Clin. Soc. Trans., 1898-1899, and elsewhere.

It is probable that malignant disease is hereditary in the same sense as tuberculosis, by offering a predisposition.

(4) *Infectivity.*—The question is still under discussion whether cancer is due to infection from outside sources, to peculiarity of cell growth, or to the invasion of a low form of animal parasite or protozoon. The reproductive cells divide in a manner different to the somatic or body cells, and it has lately been shown that malignant cells divide after the manner of reproductive cells.[1] The infectivity of malignant disease rests mainly upon four grounds : (*a*) Certain facts which appear to point to the appearance of cancer in individuals who occupy certain houses where cancer has previously occurred ; (*b*) the clinical resemblance of a rare form of disease now known to be due to the ray-fungus (actinomycosis), to a malignant new growth, and a similar resemblance in structure between certain other fungoid diseases and malignant disease ; and (*c*) the undoubted infectivity of artificially produced sarcoma in animals ;[2] (*d*) a plasmodium has been found to cause tumours in certain cruciferous plants.

The only reliable *Treatment* of cancer is (1) removal as early as possible ; the earlier and more freely this is done the more hopeful the result. (2) In certain cases of malignant disease of the mamma removal of the ovaries has been attended by involution of the growth.[3] (3) The treatment of malignant growths (more especially of sarcomata) by the injection of mixed toxins (the virus of erysipelas and other pyogenic organisms mixed) was introduced a few years ago by Coley. A number of cases of sarcoma that had been given up as hopeless after repeated operations were reported by Mr. C. Mansell Moullin[4] as having completely recovered under this treatment. (4) X-ray treatment has proved efficacious in mammary cancer.[5] It has also proved even still more efficacious when cancer has been met with on the surface of the body. But so far it has not produced any influence on the progress of the internal cancerous deposits. (5) Trypsin has been administered in 20 to 30 minim doses daily by the mouth, combined with or followed in some cases by amylopsin. Cases of involution of growth or abatement of the symptoms have been recorded. The results must at present be accepted with caution. (6) Vaccination with the dead virus of b. neoformans, an organism discovered by Doyen, has been employed, and in certain cases the symptoms have been ameliorated and the rapidity of the growth has been checked. Little, however, can be expected from this line of treatment, for the b. neoformans is discredited by most pathologists as being the causal factor. (7) Quite recently cases of undoubted carcinoma are reported to have been cured by radium.

II. **Defective Feeding and Digestive Disorders.**—Although malignant disease or tubercle should always be remembered in obscure cases of

[1] A summary of the two latest theories on the origin of cancer is given in the *Lancet*, 1904, vol. ii., p. 467.

[2] E. Billingham Smith and J. W. Washbourne, " Infective Sarcoma in Dogs," *Brit. Med. Journ.*, 1899, vol. ii., p. 1346.

[3] Dr. G. E. Herman, *Lancet*, 1899, vol. i., p. 1031.

[4] *Clinical Journal*, April 6, 1898, p. 435.

[5] Discussion at the British Medical Association Meeting (Surgical), 1902, and Clinical Society of London, May, 1904.

emaciation in the old and young respectively, perhaps the commonest causes of slight loss of flesh met with in practice are defective feeding and digestive disorders. Digestive disorders may of course exist without any wasting, and if the latter be marked and the patient advanced in life it is always suggestive of cancer of the stomach, especially if there be loss of appetite. Defective feeding without digestive troubles, and particularly deficiency in the fats and carbohydrates, may without any digestive disorder be attended by emaciation. Defective teeth are a potent source both of digestive troubles and loss of flesh. Various INTESTINAL CONDITIONS are often attended by undue spareness of body. Among the latent causes of this may be mentioned obscure intestinal stenosis and catarrhal colitis, both of which may be overlooked for a considerable time. Severe diarrhœa is often followed by rapid wasting, especially in children. Incipent cirrhosis of the liver may also be remembered as a cause of emaciation.

III. **Tuberculosis** often first makes itself known to us by a causeless loss of weight. DIABETES MELLITUS and DIABETES INSIPIDUS, CHRONIC BRIGHT'S DISEASE, and other diseases mentioned in Groups I. (*ante*) and III. (*post*) may first seek medical aid by reason of wasting. This is particularly so in diabetes, where the inconsistency of his ravenous appetite and constant thirst with loss of weight may even impress the patient. On the other hand, some cases of diabetes, especially those which occur later in life, are associated with a well-nourished if not a full habit of body. Syphilis, so frequently a cause of wasting in infancy, rarely causes much emaciation in the adult, though children affected with the hereditary disease grow up stunted, slight, and delicate. In latent tuberculosis the trunk and limbs may be wasted although the face be plump and rosy.

IV. Among the rarer causes of loss of flesh which should be remembered in obscure cases are obscure visceral disease, and especially **disease of the pancreas.** Emaciation frequently accompanies kala-azar and beri-beri.

V. Diseases of the **nervous system** may sometimes start with or present generalised wasting, such as bulbar paralysis, and the idiopathic myopathies (mostly met with in childhood), but they usually present their proper symptoms.

§ **416. Marasmus in Children.**—Infants and children emaciate with almost any disorder and with surprising rapidity. A sudden attack of diarrhœa may give rise to loss of flesh in twenty-four hours.

The principal causes are eight in number :

(*a*) Those which occur chiefly under two years of age : (1) Defective food or feeding ; (2) diarrhœa, or constipation ; (3) persistent vomiting ; (4) hereditary syphilis ; and (5) rickets.

(*b*) Those which are met with chiefly after two years of age : (6) Tabes mesenterica (enlargement of the mesenteric glands) ; (7) pulmonary tuberculosis.

(1) DEFECTIVE FEEDING constitutes the commonest cause of emaciation amongst the children of the lower classes. Such children are always

fretful, the bowels are irregular and often constipated, the stools, instead of being the normal orange colour of infancy, become either green, grey, or white and "chippy" with particles of undigested food. The error may consist either of over-feeding, under-feeding, or a defect in the quality of the food or the time that it is administered. Over-feeding is perhaps more common than the reverse. Undiluted cow's milk is very indigestible, and mothers of all classes have a tendency to make their child's feeds too strong and to give them too frequently under the notion that it will make a child "grow strong." The mother's milk, when she is out of health, or when lactation has been too prolonged, may be of too poor a quality to afford adequate nutrition. Preserved milks and foods taken for too long a time without any fresh milk lead to scurvy (§ 413). A suitable dietary, which is within the reach of all classes, has been given in § 212*a*, *ante*. A good way of giving cod-liver oil to older children is to pour away the oil from a box of sardines and replace it by cod-liver oil. After standing for twenty-four hours the oil becomes flavoured with the sardines, and both can be given together to the child.

(2) Diarrhœa or Constipation, either alone or alternating, are potent causes of wasting in infancy and childhood, and these are frequently due to dietetic errors or want of care and cleanliness in the nursery. The subject of infantile diarrhœa is fully discussed in § 218. Many cases of chronic diarrhœa in childhood will yield to mistura ricini calcis (F. 64). Chronic constipation will undoubtedly result in marasmus. In a family with which I was well acquainted the first two children died of marasmus associated with the most obstinate constipation, probably due to a chronic colitis; the case of the third child, which the mother stated exactly resembled the others in all particulars, was following the same fatal course until systematic treatment by mist. ricini calcis resulted ultimately in restoration to health.

(3) Persistent Vomiting is another cause of wasting in childhood. Like the preceding, it may be due to errors of diet, especially too frequent or over-feeding, or to gastro-intestinal catarrh. Careful dieting, lime-water, and proper intervals between the feeds will cure most cases. The reflex and other causes of vomiting (§ 191) must be considered when simple treatment is unavailing. In intractable cases feeding by the nose has been resorted to. Hypertrophied pylorus is a rare local cause of vomiting in infants.

(4) Hereditary Syphilis is a recurrent cause of wasting in some families. It is generally accompanied by snuffles or skin lesions of some kind (§ 404). The manifestations of hereditary syphilis always appear during the first year of life, generally during the first six months.

(5) Rickets (§ 447) may be accompanied by wasting, but, as Dr. Judson Bury aptly remarks,[1] "fat rickets are commoner than lean

[1] "Clinical Medicine," p. 28, Griffin and Co., London, 1894.

rickets." This disorder is recognised by the characteristic bone changes, generalised sweating, and generalised tenderness. Rickets may appear at any time between the fourth to the eighteenth month of life ; very rarely after two years of age.

§ 417. (6) **Tabes Mesenterica** is a wasting disorder occurring for the most part in children of two years and upwards, due to tuberculosis of the mesenteric glands. It is becoming less and less frequent as greater care is exercised in the cleanliness and asepsis of the milk.

Symptoms.—The onset is very insidious, and may extend over many months. Gradually the limbs and face become shrunken, and there are anæmia, listlessness, vague attacks of pyrexia, and sometimes abdominal cramps. The leading physical sign is the enlarged abdomen, which is generally tympanitic on percussion. Sometimes the enlarged glands can be felt, but more frequently there are localised thickenings and masses, which give a doughy feeling, due to chronic tuberculosis of the peritoneum. Attacks of diarrhœa with offensive stools occur from time to time.

The *Varieties* depend upon the extent to which the tuberculosis affects the peritoneum as well as the mesenteric glands. If the peritoneum is extensively affected, ascites or matting of intestines is also present (§ 176). There is no doubt that many cases of primary tuberculous peritonitis are still called tabes mesenterica. Sometimes the disease runs a more acute course with pyrexia, and resembles enteric fever, from which it can only be differentiated with difficulty when a chart is procurable.

Diagnosis.—In addition to the diseases just mentioned tabes mesenterica may have to be distinguished from the distension of the bowels due to improper feeding, in which there is generally no pyrexia, no resistant masses, and disappearance on regulating the diet. *Rickets* may be attended by a flabby or distended abdomen, but is not usually attended with so marked emaciation, and the characteristic rachitic changes in the skeleton differentiate it. *Morbus cœliacus*, a condition occurring in children, in which wasting is accompanied by atonic dilatation of the intestine, and frothy, porridge-like, offensive motions, may be difficult to distinguish, but there are no glandular masses in this disease.

Prognosis.—The course of the malady is apt to be irregular, with intervals of apparent recovery, followed by relapses. Sometimes the glands undergo a fibroid change, and what appear to be the most unlikely cases recover. Among the untoward symptoms are acute local pain and tenderness, indicative of peritonitis ; constant diarrhœa, indicating ulceration of the bowels ; and the evidences of tubercle elsewhere. The complications are numerous—ulceration of the bowels, attended by pyrexia and intractable diarrhœa ; general tuberculosis ; abscesses forming and bursting in various situations, such as into the peritoneal cavity or from the umbilicus, the latter forming a chronic fistula. Intestinal obstruction may result at any time from the formation of bands of adhesion.

Etiology.—Tuberculosis of the mesenteric glands (tabes mesenterica) may occur at almost any age. I have met with it at twelve months and also at the age of forty-five, but it is relatively rare under two years of age. Male children appear to be more prone than females. The introduction of the tubercle bacillus is the proximate cause, and it has been generally supposed that this is introduced by the ingestion of milk from tuberculous cows, or milk which has otherwise become contaminated. If the mucous membrane of the alimentary canal is healthy there seems to be less risk of contamination. There is often a history of recurrent attacks of intestinal catarrh which have led to a weakening of the mucous membrane and the consequent entrance of the bacilli, but since more attention has been paid to the supervision of dairies and the sterilisation of milk, the disease has undoubtedly become less frequent, and it seems probable that it will ultimately be stamped out.

Treatment.—Prophylactic measures consist in sterilising or Pasteurising the milk, and regulating the supply whence it is obtained. In the remedial treatment the diet needs careful attention, both in its quantity and the quality. All food

should be raised to the boiling-point before administration. Small doses of creosote by the mouth or by enema, together with tonics, cod-liver oil, iodide of iron, and other remedies mentioned under Phthisis (§ 94), are employed. Among local measures mercurial or iodoform ointment rubbed into the abdomen has proved valuable in many cases.

GROUP III. DEBILITY ONLY (ASTHENIA).

The causes of debility not necessarily accompanied either by pallor or emaciation are as numerous as those of the two preceding groups, and it must be remembered that all the disorders in both of those groups may commence with weakness only; in short, the majority of chronic disorders begin with debility. The fallacies (§ 393) and methods of examination have already been given.

Commoner Causes.

I. Senile decay and arterial disease.
II. Chronic interstitial nephritis.
III. Neurasthenia, or other incipient or obscure diseases of the nervous system.
IV. Chronic dyspepsia and obscure diseases within the abdomen.
V. Cardiac, tuberculous, and other obscure diseases within the chest.
VI. Diabetes mellitus and diabetes insipidus.
VII. Conditions referred to in Groups I. and II., in which anæmia or emaciation are ill-marked.

Rarer Causes.

I. Myxœdema.
II. Addison's disease.
III. Bronzed diabetes.
IV. Disease of the pancreas, acromegaly, beri-beri, pellagra, and many other conditions mentioned in Groups I. and II.

When a patient is suffering from debility or loss of vigour of mind and body, without any very marked pallor or obvious loss of flesh, and without any marked physical signs or other evidences of disease, in the *first half* of life one would suspect neurasthenia, chronic dyspepsia or gastro-intestinal disorders, incipient or latent tuberculosis, diabetes.

In the *second half* of life one would suspect senile decay, chronic interstitial nephritis, obscure cardiac valvular or aortic disease, diabetes, myxœdema, Addison's disease.

And failing these, some of the conditions previously mentioned among the anæmic or wasting disorders (Groups I. and II.).

§ **418.** I. **Senile Decay and Arterial Disease.**—As we advance in years the power both of body and mind notably declines. This should not be very obvious under sixty, but the age at which it appears differs considerably in different persons, and still more in different families, for the onset of decay in persons, as in plants and animals, is largely a question of heredity plus the previous habits of the individual. Structurally there is a universal tendency to atrophy or degeneration of the parenchyma or functionally active tissues, and slight increase in the lower forms of tissue (such as fibrous and supporting tissues) in all the organs and structures of the body. This is particularly seen in the

cardio-vascular system where it is the muscular coat—*i.e.*, the functionally active tissue of the arteries which first show signs of senile degeneration.[1]

Symptoms.—Consequent on the changes just mentioned there is a universal lowering of vitality and nutrition, and the general enfeeblement of thought, word, and act which results in the mumbling, fumbling, and stumbling of old age. Physical weakness comes on so slowly that even the patient himself is hardly aware of it, and it is not sufficiently recognised that widespread disease of the arteries alone may give rise to progressive mental and bodily enfeeblement at whatever age it comes on.

The following case may be quoted by way of illustration: Jessie T—— was admitted into the Paddington Infirmary in 1889 at the age of forty-nine. At the age of forty-five she began to complain of muscular and mental weakness. This gradually increased, so that at the time of admission she could only walk by pushing a chair before her, and the case was thought, therefore, to be some kind of paraplegia. There were absolutely no physical signs in any organ and no evidences of disease in the nervous system at any time, and the urine was always normal. She became progressively more and more enfeebled in body and mind, gradually took to bed, and died ten years after admission of progressive asthenia. I was present at the autopsy in 1899, and all the organs were normal, both macro- and microscopically, with the exception of atrophy; but there was extreme and widespread disease of all the arteries of the body and of the brain, the main change being granular degeneration of the muscular coat of the heart and arteries, with consequent yielding and great dilatation of the arteries. In infirmary work I have met with arterial disease in persons as young as thirty-eight and forty—cases which were examined post-mortem—and weakness was the only symptom.

The condition of the heart and aorta should be carefully noted, especially any rigidity of the latter, as shown by accentuation of the second sound at the base and the character of a pulse-tracing (Fig. 31, § 66). The arterial tension should be noted from day to day, and the walls of the superficial arteries carefully investigated (§ 65 *et seq.*). Among the later symptoms associated with senile decay of the cardio-vascular and other tissues, perhaps vertigo is the commonest. A large number of other vague cerebral sensations may be experienced, and even convulsions (senile epilepsy) may occur. The urine should always be carefully and repeatedly examined so that senile decay may not be confused with other causes of debility (*infra*), particularly chronic interstitial nephritis.

The *Prognosis* depends a good deal upon the amenability of the cardio-vascular system to treatment. The diseases to which old age is most liable are of a chronic and degenerative nature, the arterial—*i.e.*, the nutritive system being responsible for this, and itself showing the most definite and widespread signs of degeneration. The immediate cause of death in old age is usually some pulmonary complication. An analysis of 409 fatal cases in persons of sixty years of age and upwards, who died consecutively in Paddington Infirmary—February 1, 1886, to

[1] "On Senile Decay," Trans. Med. Soc. of Lond., 1897; and "On Arterial Hypermyotrophy and Medial Sclerosis," Trans. Path. Soc. of Lond., 1904.

December 31, 1892, showed that 121, or 30 per cent., died of some pulmonary condition other than tubercle (pneumonia, bronchitis, hypostatic congestion, and pulmonary apoplexy). The next most fatal disease was cancer, 62 cases (15·5 per cent.), then simple senile decay, 35 cases (9 per cent.), then contracted granular kidney, 24 cases (6 per cent.), then pulmonary tuberculosis, 22 cases (5·5 per cent.).

The *Treatment* should be mainly directed to the cardio-vascular system, and especially to the raising of low tension (§ 62) and the lowering of high arterial tension (§ 61).

Stimulants are nearly always called for in the treatment of disease in the aged. The food should be light, nutritious, and easily assimilable, and small in quantity ; it is wonderful how small a quantity of food the aged require, and it has been reckoned that 12 ounces of solid food per diem are sufficient. It is not only useless but harmful to over-feed the aged ; keep them warm and prevent chill, but do not over-feed them. Strychnine is *par excellence* the tonic of the aged.

II. **Chronic Interstitial Nephritis** (§ 297) (Synonyms : Chronic Bright's Disease, Granular or Gouty Kidney), should always be remembered as a cause of progressive enfeeblement coming on at or past middle life. It is indeed very apt to be mistaken for senility, and failing vigour is the leading symptom for which the patient seeks advice in a large proportion of both these conditions. Sometimes this weakness is accompanied by generalised muscular wasting, but quite as often there is none. The complexion is generally sallow, but there is no definite pallor till late in the disease. Headache is common, chronic interstitial nephritis being one of the commonest causes of headache coming on after middle life.

III. **Neurasthenia** (§ 523) and various other **functional** and **degenerative conditions of the nervous system** may be evidenced by general weakness. This is particularly the case in the functional disorders, such as neurasthenia and hysteria, where the weakness may amount to complete prostration. Such cases are usually met with in the first half of life or middle age. Among the gross lesions which are apt to come on insidiously with weakness are *paralysis agitans*, *bulbar paralysis*, and frontal *cerebral tumour*—diseases more often met with in the second half of life. *Myasthenia gravis* is a rare condition, coming on usually with generalised weakness (see also Generalised Paralyses, § 573).

IV. **Chronic Dyspepsia,** gastric dilatation, and other obscure **diseases within the abdomen** may be attended by debility only for a long time. Gastro-intestinal troubles produce it by chronic toxæmia and malassimilation of food. *Mucous colitis* may be specially mentioned in this connection, also *appendicitis*, *abdominal cancer*, and many of the other conditions mentioned in Chapter IX.

V. **Obscure Diseases within the Chest** may be manifested by general weakness. *Incipient tuberculosis* of the lungs should always be remem-

bered in cases of unexplained general debility, especially in younger subjects. In the second half of life, and especially in those with an alcoholic history *cardiac enfeeblement* and fatty heart (§ 52) may cause no other symptom than debility, and the same may be said of *aneurysm* of the third part of the aorta.

VI. **Diabetes Mellitus** and **Diabetes Insipidus** are often first revealed by progressive weakness, though our attention may also be drawn to these conditions by the thirst, polyuria, emaciation, or glycosuria (§§ 309 and 310). These diseases may occur in either the first or the second half of life.

The **rarer causes of debility** not necessarily accompanied by pallor or emaciation are Myxœdema, Addison's disease, Diseases of the Pancreas, Acromegaly, and some of the conditions mentioned in Groups I. (anæmia), and II. (emaciation).

§ **419.** I. **Myxœdema** (μυξα, mucous; οιδημα, swelling) is an insidious disease evidenced by weakness, lethargy, and other manifestations of deficiency in the metabolic processes of the body, due to diminished thyroid function. It was so named by my respected teacher, the late Dr. W. M. Ord, on account of the mucoid degeneration which takes place in the subcutaneous tissue throughout the body, and is the most obvious anatomical change, producing a kind of solid œdema. It was at first believed by him to constitute a new and hitherto undescribed form of generalised œdema, but it is now known—and this was admitted by him—to be in reality a sporadic cretinism (§ 138) occurring in adult life. It is due to a diminished function of the thyroid gland, generally accompanied by a diminution in volume of the organ (see § 135). It is not a very uncommon disease, but is frequently overlooked.

Symptoms.—The weakness here takes the form of a very characteristic slowness of action, of thought, and of speech. It comes on very gradually, and the patient frequently seeks advice for some other reason. (2) The aspect (Fig. 1, § 9), however, is so characteristic that when the doctor has once seen a case he recognises it again directly. The face is slightly puffy, and, the lines of expression being obliterated, it appears immobile and vacant; the puffiness of the eyelids may be mistaken for dropsy; the malar capillaries are injected, and cause a characteristic flush on each cheek. The hair of the scalp and eyebrows is coarse, scanty, and brittle. The speech is slow and drawling, the hands are flat, puffy, and spade-like, and the nails brittle. All the movements are slow, and the mental processes lethargic. (3) As the disease advances the skin of the whole body is thickened, and at first sight gives the appearance of generalised dropsy; but myxœdema may be distinguished from the latter by the absence of pitting; puffy swellings may be noticed above the clavicles. (4) The urine is copious, of low specific gravity, and deficient in urea, but there is no albumen or other change. (5) The thyroid is usually atrophied, though it is difficult to make this out, partly on account of the myxœdematous condition of the neck.

Diagnosis.—The disorder may be mistaken in its earlier stages for *anæmia* and the other disorders mentioned in Group I., also from the other causes of *debility* now being considered; but the facial appearance is very characteristic. It may be diagnosed from chronic interstitial nephritis and other forms of chronic renal disease by the absence of pitting on pressure and the absence of the urinary changes of renal disease.

Prognosis.—Before the introduction of the thyroid treatment patients rarely lived more than a few years, dying usually of some intercurrent malady or complication. Mental changes, such as hallucinations, and even dementia, may supervene. Menorrhagia and other hæmorrhages are sometimes associated with the disease.

Etiology.—The disease is much more frequent in women, in whom it supervenes usually about middle life. It is undoubtedly due to a deficiency of thyroidal function (Athyroidism, § 135), but what constitutes the proximate cause of thyroidal atrophy is not known. It is certainly not confined to any particular district as is endemic cretinism, and it differs from the latter in the age and sex which it chiefly affects.

Treatment.—The treatment by the interanl administration of the thyroid gland is so certain and efficacious that this may be used as a means of diagnosis. Tabloids of the extract, 4 or 5 grains, or one-third of a fresh sheep's gland, may be administered twice or thrice daily after meals, gradually increasing until intolerance is produced. This intolerance is known by the supervention of headache, giddiness, palpitation, or a general feeling of discomfort. The addition of arsenic to the treatment will sometimes relieve these symptoms, and enable us to push the thyroid for a long time. Complete recovery may ensue after a few weeks' or few months' treatment. Relapses are very apt to occur upon ceasing the thyroid administration and the patient may be obliged to continue treatment indefinitely.

§ **420.** II. **Addison's Disease** is a rare malady, described by Dr. Addison[1] in 1854, characterised by progressive loss of strength and general pigmentation of the skin, due to disease of the suprarenal capsules. The most common morbid change in the latter undoubtedly is tuberculosis, which may go on to caseation, fibroid, or other changes. Occasionally the suprarenals are affected in this disorder by malignant and other disease.

The *Symptoms* come under five categories: (1) Progressive *general weakness* is its most marked feature and may appear long before any other symptom. It is unaccompanied, as a rule, either by anæmia or marked emaciation until perhaps towards the end. Uncomplicated cases present a sub-normal temperature throughout. (2) *Pigmentation* of the skin of a more or less general distribution ensues sooner or later. The colour begins with a yellowish tint, which gradually deepens into a bronze mahogany colour. The localities most affected are the exposed parts—the face, neck, and hands—and those where pigmentation is normally present, such as the axillæ and nipples and sites of pressure. The edge of a patch of colour shades gradually into the healthy skin around, which makes it difficult to discover such a patch in its early stage. The mucous membranes of the tongue, mouth, and throat frequently present the same kind of patches. (3) *Gastric* symptoms generally occur at some time, such as vomiting, hiccough, and cramp-like pains in the abdomen and loins. Pains in the limbs may also be complained of. The bowels are often constipated, but sometimes there is intractable diarrhœa, which may be fatal. (4) *Cardio-vascular symptoms* may be present—palpitation, dyspnœa, sighing, yawning, and later on a tendency to collapse. The small rapid pulse, with its extreme low tension and toneless artery, is almost of itself sufficient to warrant a diagnosis. (5) *Nervous symptoms* are less common, but may consist of headache, vertigo, and nervousness. The mind is clear, except towards the end, when delirium, convulsions, or coma may set in. These five groups of symptoms vary in their predominance, but asthenia is always present, and pigmentation nearly always. There are two varieties—acute and chronic.

The *Diagnosis* is often very difficult on account of the vagueness of the symptoms, the absence of physical signs, and the resemblance of the pigmentation to various other cachectic states, especially cancer. *Cancer of the pylorus* is accompanied by sallowness, which is often mistaken for the pigmentation of Addison's disease. Both, moreover, are accompanied by enfeeblement, gastric pain, and vomiting. The diagnosis from *other pigmentary conditions* is given among the causes of pigmentation (§ 495). Slight jaundice, the pigmentation of malaria, chloasma, and arsenical pigmentation must be borne in mind. *Chronic Bright's disease*, neurasthenia, and other conditions attended by asthenia mentioned in this group are apt to be mistaken for the disease.

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[1] "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules," London, 1855.

Prognosis.—The course of the disease is progressive, and usually prolonged; it may last one to ten years. There are frequent relapses, with intermissions of comparative health, but it always terminates in death. It may end suddenly with syncope, severe vomiting, and diarrhœa, convulsions, or coma, or it may terminate gradually by asthenia. The commonest complication is tuberculosis of the lungs, or elsewhere; and pulmonary tuberculosis is the commonest cause of death.

Etiology.—Patients are usually about middle life, and by far the larger number are males. The essential cause is disease of the suprarenal capsules.

Treatment.—Suprarenal extract may be tried in tabloids containing 1 grain (equivalent to 15 of the gland) twice daily, and in gradually increasing doses. The pathology of the disease rests upon the loss of the internal secretion of the suprarenal bodies, and if the secretion can be made good the fatal issue should be averted; suprarenal extract and fresh gland, however, have not been successful in arresting the disease hitherto. The symptomatic treatment consists in rest, and supporting the strength by cod-liver oil, tonics, good nourishment, and hygienic living, on the same principles as other tuberculous affections. Cold and over-exertion should be avoided.

§ 421. III. **Bronzed Diabetes,** or what should more properly be called "pigmentary cirrhosis of the liver with glycosuria," is a rare condition which might be mistaken for Addison's disease. In certain cases of hypertrophic cirrhosis of the liver there is a considerable amount of *hæmo-chromatosis*, which may give rise not only to the symptoms characteristic of cirrhosis, but to definite pigmentation of the skin. Certain of these cases may also be associated with a more or less permanent glycosuria, possibly owing to concurrent cirrhosis of the pancreas. The glycosuria usually occurs as a late event in such cases.[1]

IV. **Disease of the Pancreas, acromegaly, beri-beri, pellagra,** and **other conditions mentioned in Groups I.** and **II.** (*q.v.*), may come on with debility only, or the patient may seek relief for debility.

[1] See also Dr. Alex. Bruce, the *Lancet*, 1898, vol. i., p. 1692; Prof. Wm. Osler, *Brit. Med. Journ.*, 1899, vol. ii., p. 1595; and Auschütz, *Deut. Arch. für Klin. Med.*, vol. lxii., p. 411.

CHAPTER XVII

THE EXTREMITIES

In the preceding pages we have seen on several occasions that so-called local diseases, such as pneumonia and endocarditis, have by scientific research been shown to be only local manifestations of a general microbic infection. This principle will here again be illustrated, for a gouty joint is only the local evidence of disordered metabolism, and acute rheumatism has now been shown to be microbic in origin. My own belief is that all joint diseases (other than traumatic) are but local manifestations of some toxic, septic, or infective blood condition. In conformity, however, with the scheme of this work, whereby all diseases are approached from a symptomatic standpoint, certain diseases, the symptoms and physical signs of which are referable mainly or entirely to the upper or lower extremities, will now be considered.

PART A. SYMPTOMATOLOGY.

The CARDINAL SYMPTOM referable to the extremities is **pain** (or painful sensations of some kind), which may or may not be accompanied by some **physical change.**

§ **422. Pain in the Limbs** should be investigated, like pain in other situations, as to its *position*, *character*, *degree*, *constancy*, and *duration*. Its position may be localised to the skin, or to a joint or any other structure, or be generalised, as in sheer exhaustion ; its character may be sharp and shooting (as in tabes) or dull and heavy (as in vascular lesions), or like pins and needles (as in nerve and neuro-vascular lesions). Pain in the limbs may come on *acutely* or *insidiously*.

(*a*) *Acute pain in the limbs* coming on more or less SUDDENLY may herald influenza, enteric fever, malignant endocarditis, variola, scarlatina, or some other specific fever. In many cases of influenza this pain and slight pyrexia are the only symptoms. *Acute rheumatism* also comes on rapidly with pains referable to the muscles, bones or joints, and so does *dengue* (break-bone fever). *Trichinosis* is attended by excruciating muscular pain in the second stage of the disease, when the parasite begins to migrate. A sudden sharp pain in one spot in the limb

is felt when *embolism* of an artery occurs ; so also in thrombosis of a vein. In both cases pyrexia may be absent.

(*b*) Pains in the limbs coming on more or less *insidiously* may be due to (1) *peripheral neuritis*, and therefore all its causes—*e.g.*, alcohol, diphtheria, or syphilis. Long before such a disease as neuritis was recognised by the profession " pains in the limbs " were known to arise from excessive indulgence in alcohol, and in dealing with alcoholic subjects this should be remembered. The same pains may occur in neurasthenia, and it is quite possible that the pathological condition in this disease, as in alcoholism, neuritis, and the acute specific fevers, may be a toxic condition of the blood. (2) Pains in the joints or muscles are characteristic of *chronic rheumatism*, *rheumatoid arthritis*, *osteoarthritis*, and *gout ;* when the pain seems to be in the bones *syphilis* should be suspected. (3) Pain affecting the nerves has a shooting or darting characteristic of its own, as in neuralgia, sciatica, or tabes dorsalis. (4) Growing pains (so-called) in children are often of a somewhat serious import, as being the only tangible evidence in them of subacute rheumatism, which may nevertheless be sufficient to produce endocarditis with permanent damage unless the condition is recognised and rest in bed with salicylates prescribed. (5) The " numbness " or tingling of the hands and feet known as acroparæsthesia (a word which indicates perverted sensations in the extremities) is very characteristic ; it may be indicative of some vaso-motor disorder such as erythromelalgia (§ 429), or the incipient stage of tabes, general paralysis, or other organic disease. (6) Severe pain in the foot should lead us to suspect flat foot or metatarsalgia. Metatarsalgia is a neuralgia of the foot due to lateral displacement of the heads of the metatarsal bones which press upon the nerves, and may also produce a corn (for which, indeed, the patient may seek advice).[1] (7) Various diseases of the bones (§ 446) may come on insidiously, with nothing more definite than vague pain in the limb or limbs. This must be specially remembered in children in whom pains of gouty or alcoholic origin are rare. Various forms of inflammation, acute or chronic, may arise, and unless the bone be superficial there may be no surface indications at all. Some of them, such as osteomyelitis, are very serious, and require prompt recognition. (8) A muscular strain or rupture of some muscular fibres may leave a chronic pain and partial loss of function (unattended by any physical sign) which is often hard to cure. In one of my cases the pain lasted over five years. (9) *Local injury* or pressure may cause pain, such as injury from a crutch, or sleeping in a cramped position, or lymphatic glands or other tumours in the axilla, neck, or pelvis. Shooting pains down the arms, especially the left, occur in aneurysm of the aorta and angina (see also Causes of Single Nerve Paralysis, § 569).

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[1] J. Jackson Clarke, the *Medical Press and Circular*, June 14, 1899.

PART B. PHYSICAL EXAMINATION.

The physical signs referable to the extremities mainly consist of some visible or tangible alteration in the skin and general contour of the limb, the joint, the muscles, the bones, or the vessels and nerves.

§ **423. Inspection of the Limb** may reveal generalised redness or alteration of colour, œdema, varicose veins, or some other diffuse or localised swelling. Eruptions prone to affect the skin of the extremities are specially dealt with in Chapter XVIII.

Even without the skill of a palmist or the acumen of a Sherlock Holmes a great deal concerning the temperament, habits, and diseases of a patient may be learned by a careful **inspection of the hands.** For instance, the long, thin, dextrous fingers, perpetually on the move, will almost surely indicate a nervous temperament and imaginative disposition, just as the short, thick, almost clumsy fingers and hands of another will bespeak slowness, deliberation, and doggedness. The occupation of a patient may often be learned from a glance at the palms. Some people habitually have cold, damp, clammy hands, and these are generally the subjects either of the alcoholic habits or the rheumatic diathesis, occasionally some other condition causing a defective vaso-motor tone. The *nails* can also afford us some information. They are dusky in all conditions of impaired circulation, and pale in anæmia ; compression on the tip of the nail should not completely empty the capillaries, as it does in anæmia. In aortic regurgitation compression of the nail tip reveals a capillary pulsation. A transverse ridge or groove in the nails indicates an arrest of growth, and may mark the date of an illness or any disturbance of nutrition, of even so slight a nature as seasickness.[1] It is useful to remember that the nail takes about five or six months to grow from root to tip. Various distortions of the nail occur in neuritis and injury. Clubbed fingers—*i.e.*, fingers with a bulbous end and great convexity of the nails (filbert-shaped nails), are characteristic of congenital cardiac disease or valvular disease in early life. Emphysema and chronic phthisis and any disease attended with profuse expectoration may be attended by the same deformity. *Glossy fingers* (fingers with smooth, thin skin) are the result of a neurotic dystrophy, and are associated with destructive and paralytic lesions of the nerve trunks ; they also occur in sclerodermia. Dactylitis is a thickening of one phalanx due to disease of the bone, with infiltration of the tissues of the fingers, resulting in a deformity known as the " champagne bottle finger." It is met with chiefly in tuberculous, and sometimes syphilitic, children. " Heberden's nodes," lipping and distortion of the phalangeal joints, are in reality osteo-arthritis of the fingers. Gouty nodules of urate of soda form white masses near the joints, just beneath the skin, and have an external resemblance to Heberden's nodes. The bone ends of the wrists are enlarged in rickets, syphilis, and pulmonary osteo-arthropathy. " Spade-shaped " hands (with thickened tissues) are suggestive of myxœdema, and large, flat, ungainly hands with osseous enlargement, of acromegaly and pulmonary osteo-arthropathy. The " claw hand " (*main en griffe*) occurs as the result of injury or neuritis of the ulnar and median nerves ; it is also seen in progressive muscular atrophy, syringomyelia, and cervical pachymeningitis. Wrist-drop is very characteristic of lead palsy.

Cyanosis (Blueness) of the Extremities.—Many people are born with a tendency to congestion—*i.e.*, blueness or redness—of the hands and feet. They appear to be the evidences of a vaso-motor instability or want of vascular tone. Such people are specially liable to chilblains and other kinds of erythema ; in short, they might be called angio-neurotic subjects. The slighter degrees of cyanosis can be revealed by examining the nails and depressing the nail tip. The *causes of cyanosis* were fully discussed in § 26. Erythromelalgia, " dead hands," gangrene, and Raynaud's disease are referred to below.

§ **424. Varicose Veins** consist of dilatation and tortuosity of the superficial veins, and are practically only met with in the legs, where their tortuous elevations produce obvious and characteristic alterations in the contour of the limbs. They

[1] *Vide Illustrated Medical News*, about 1890.

occur chiefly in those who stand a great deal, and are more frequent in the female sex, and especially in those who have borne children. Varicosity of the veins predisposes to eczema and ulceration, and severe hæmorrhage may ensue from their rupture. The *Treatment* of varicose veins belongs mainly to the surgeon, but it may be useful to mention here four hints which Mr. C. B. Keetley offers—viz., (i.) the more exercise the patient takes the better, especially in the form of bicycling; (ii.) the legs should be kept as cool as possible; (iii.) if the varicose veins occur on the thigh or the knee, operation is advisable in preference to the wearing of stockings or rubber bandages; (iv.) elastic stockings (the usual mode of treatment), should be made to lace. The best bandage, however, in my experience is the crepe de voile; it is cool, light, and (if not ironed after washing) of very equable pressure.

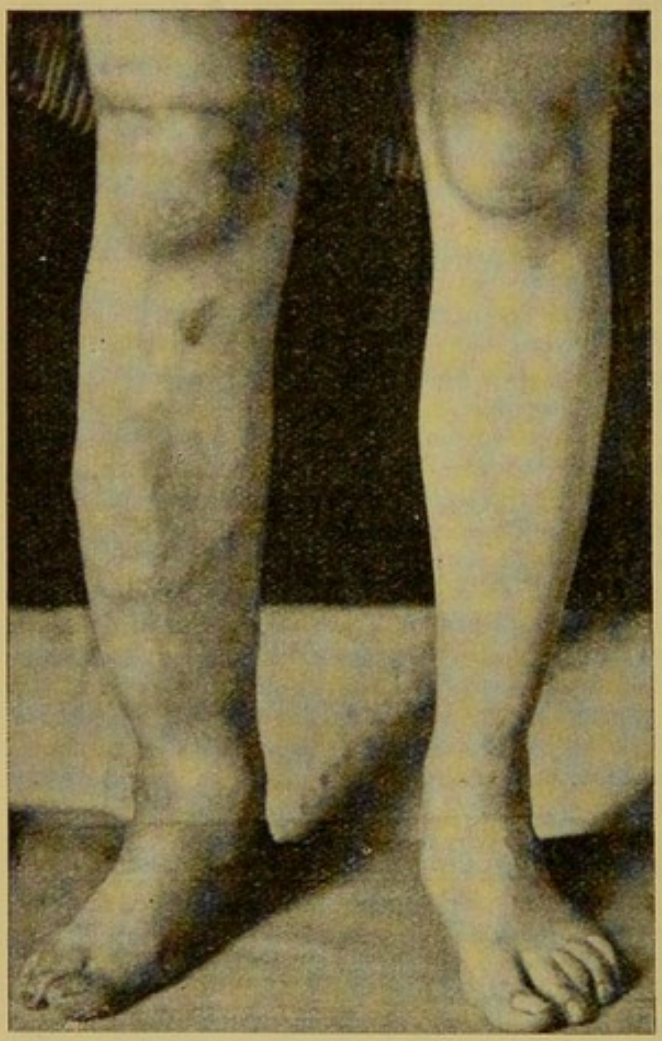

Fig. 120.—ELEPHANTIASIS TELANGIECTODES, in a woman about forty-eight years of age.

Elephantiasis Telangiectodes (Fig. 120) is a rare condition which is apt to be mistaken for varicose veins. It consists of a hyperplasia of the subcutaneous tissues together with a varicosity of the superficial veins which form loose masses like bunches of grapes beneath the skin. They are usually associated with a certain amount of superficial telangiectasis (dilatation of venules) in the skin over and around the masses.

§ 425. Œdema of one Limb (localised dropsy) produces generalised swelling which pits on pressure. The swelling due to the rare condition elephantiasis lymphangiectodes (see below) is of a much more solid character. Apart from *inflammatory œdema* and an extensive *angio-neurotic œdema* (§ 454), dropsy of one arm or one leg always points to some obstruction of the main vein of the limb by thrombosis within or pressure upon the vein. (1) *Thrombosis* (coagulation within the living vessel) with or without phlebitis (inflammation of the vein) is not uncommon in the femoral or iliac vein in the leg, and the brachial in the arm. In addition to œdema there are pain and tenderness at the seat of the obstruction, and a history or evidence at the time of some cause of thrombosis or phlebitis, such as phthisis and other wasting disorders, any of the acute specific fevers, injury or local extension (as from an ulcer). The commonest example of thrombosis is *phlegmasia dolens* (or white leg), which is so apt to come on after confinement, partly as a result of the hyperinotic condition of the blood (*i.e.*, the excess of fibrin-forming constituents) which is associated with the puerperal state, and partly owing to previous pressure on the veins within the pelvis. Little can be done in the

way of treatment beyond complete rest in the horizontal position, warmth applied to the limb, and the administration of iron. Nothing will remove the obstruction, but in course of time the condition is relieved considerably, if not altogether, by the establishment of collateral circulation. (ii.) Œdema may also be due to *pressure upon a vein* by a tumour, such as enlarged glands in the axilla or elsewhere, aneurysm, or other intra-thoracic growth pressing upon the veins coming from the arm ; pelvic cellulitis, carcinoma of the uterus or bladder, bands of adhesion, hydatid, or other intra-pelvic growth pressing on the veins of the leg.

Elephantiasis Lymphangiectodes (Fig. 121) is a solid œdema, not pitting on pressure in any notable degree, affecting one leg, occasionally one arm, or the scrotum, due to a blocking of the lymphatics of the limb. It is met with chiefly in tropical countries in persons whose blood at night contains the embryo of the filaria Bancrofti. The adult worm is believed to block the lymphatics, and so produce the disease. It is, however, occasionally met with in temperate climates in persons whose blood does not reveal the parasite, and the cause of the blocking in these cases is obscure.

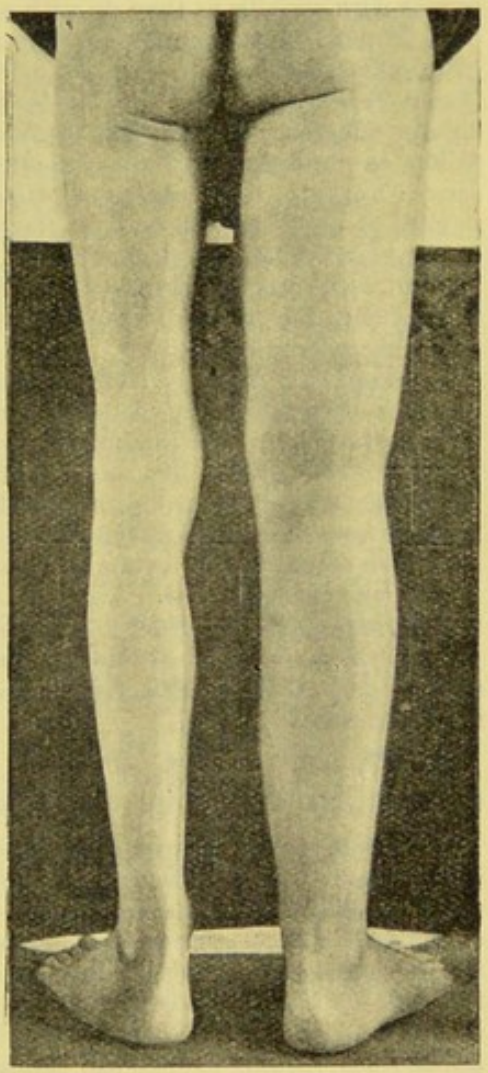

Fig. 121.—ELEPHANTIASIS LYMPHANGIECTODES in a man about forty years of age who had never been abroad.

§ 426. Swelling of the Lymphatic Glands in the neck, axillæ, groins, or elsewhere on the surface of the body or limbs may be due to : 1, injury and septic or infective processes ; 2, tuberculous disease ; 3, malignant disease ; 4, syphilis ; 5, acute specific fevers ; 6, lymphatic leukæmia ; 7, Hodgkin's disease ; 8, lymphoma (?) ; 9, trypanosomiasis. The first three arise in glands adjacent to some focus of mischief, and the glandular swelling usually remains localised ; in the remainder all the lymphatic glands tend to become affected.

1. *Local* injuries, septic sores, and abscesses give rise to enlargement of the neighbouring lymphatic glands. When a patient complains of pain and enlargement of the glands in the groin, for instance, these may be due to direct injury to or pressure on those glands ; but one should always carefully inspect the foot for abraded skin around the toe-nails, through which dirt or stocking dye may have been absorbed. *Post-mortem scratches* or inoculation from septicæmic cases are of a much more virulent nature. Red streaks along the course of the lymphatics indicate lymphangitis. The glands at the elbow and axilla become acutely painful and tender, and they may rapidly suppurate. This is a conservative process, for in this way the septic virus is prevented (usually) from extending to the general circulation. If, however, the virus is too intense or the dose be too large, general septicæmia and death in a day or two is the result before suppuration can occur.

2. *Tuberculous* disease of the lymphatic glands, especially of the neck, is very frequent in children. The disease is usually secondary to some other focus latent or active, but is generally localised to one group of glands, and the process is slow and chronic. It is also recognised by the fact that the glands very soon become

matted together into one solid mass, which in due time, if the case be untreated, undergoes caseation, breaks down, and leaves a characteristic ulcer.

3. *Cancer* gives rise first to inflammatory enlargement of the adjacent glands; later, the adjacent and distant glands become the seat of secondary cancer (§ 415). Lymphosarcoma is a sarcomatous growth *starting in* the lymphatic glands. It rapidly invades the surrounding structures, and the neighbouring glands.

4. *Syphilis* first affects the lymphatic glands in the neighbourhood of the chancre. They are small, hard (shotty), painless, and only perceptible on palpation, but for many years afterwards all the glands of the body, especially those in the groin, may be discovered on careful palpation to be thus indurated. They never suppurate with syphilis as they do with a soft chancre.

5. In most of the *acute specific fevers* there is, as in syphilis, a slight generalised glandular enlargement. In those fevers which have a local manifestation, the throat in scarlatina and diphtheria, for instance, the adjacent glands are first and chiefly affected. In bubonic plague the enlargement is very great. In certain milder cases of *plague* nothing but slight glandular swelling and a little fever occurs (pestis minor). Such cases are often overlooked, but they may give rise to epidemics of pestis major, or true plague. *Rheumatoid arthritis* is accompanied by enlargement of the glands and spleen, especially in children.

6. In *Lymphatic Leukæmia* there is a generalised glandular enlargement, but it is a subordinate feature to the enlargement of the spleen. The blood changes are very characteristic (§ 407).

7. *Hodgkin's Disease* (lymphadenoma, anæmia lymphatica) starts with a swelling of one group of glands, which enlarges paroxysmally, generally attended with corresponding paroxysms of fever. The individual glands remain separate, painless, and may feel like a bunch of grapes. Each gland may attain a large size. Sooner or later other groups of glands become similarly involved; but the spleen is never much increased (§ 408).

8. *Lymphoma* is a term applied to the enlargement of a single group of glands where no extension or constitutional symptoms occur. It is possible that lymphoma is a localised form of Hodgkin's disease.

9. *Trypanosomiasis.*—Glandular swellings, particularly of the neck glands, in negroes and others in East Africa and other infected areas, occur as evidences of trypanosomiasis.

§ 427. The JOINTS, MUSCLES, BONES, VESSELS, NERVES, and CONSTITUTIONAL SYMPTOMS should be next investigated.

The **joints** may need investigation for tenderness, pain, heat, swelling, or redness, and for loss of function or range of movement. The affected and the unaffected sides should be carefully compared. Slight degrees of fluid in a joint are often difficult to detect. The active movements (those which the patient can make) and the passive movements (those made by the doctor) should, with due consideration and caution, be tested. Among the *fallacies*, paralysis, or muscular weakness is often simulated by chronic joint diseases, and *vice versa*, and pain in the limbs from various causes will often simulate a stiffness of the joint. In acute joint disease the fallacies of epiphysitis and acute osteomyelitis must be avoided.

The **muscles** may be investigated for tenderness, stiffness, or swelling. The investigation of paralysis, tonic or clonic spasm, or wasting, is given under diseases of the nervous system (Chapter XIX.). We are here concerned only with pain, tenderness, or swelling localised in the muscles; it is the presence of these localised symptoms which helps us to differentiate muscular diseases from paralysis and other diseases of the nervous system. To decide that the lesion is not in the bones or ligaments may be difficult; if it be in the muscle the pain is greater during active than passive movement of the affected muscle; if in the ligaments or joints the pain is about equal.

The examination of the **bones** belongs specially to the surgeon, but disease situated in the bones may be evidenced by pain, tenderness, swelling, or deformity.

They often first come under the notice of the physician when pain is their only symptom, and diagnosis presents considerable difficulty.

In the diagnosis of SWELLINGS CONNECTED WITH BONES it is well to remember the following data.[1] Symptoms come on *acutely* with trauma, periostitis, osteomyelitis, and deep abscess; slowly and *chronically* with caries, necrosis, chronic periostitis and osteitis, rickets, syphilis and tumour. In regard to physical signs the *diaphysis* is mainly affected in acute and chronic inflammation, in sarcomatous and other tumours; the *epiphysis* in rickets, syphilis, and central sarcoma. The consistency of the swelling is *soft* in abscess and vascular sarcomata, *hard* in chronic inflammation. As regards the mode and rate of growth, the swelling *progressively enlarges* in inflammatory and malignant tumours, and is *stationary* in chronic inflammation and benign tumours; *receding* swellings are always inflammatory.

The **vessels** and **nerves** need examination when any of the symptoms indicate their implication, as in erythromelalgia and some other conditions in Group I. below. Pressure along their course may elicit tenderness, indicative of inflammation. The symptoms and effects of peripheral neuritis are given in Chapter XIX., and embolism of an artery or thrombosis of a vein in § 430.

The **viscera** should be examined, particularly in acute joint diseases, which are almost always the product of some blood disorder—*e.g.*, the heart must always be examined in rheumatic conditions, the kidney in gouty disorders.

Pyrexia and **Constitutional Symptoms** are present in a considerable number of diseases of the extremities, particularly in the acute joint and bone disorders, and they may be investigated on the lines laid down in Chapter XV. Rigors and sweating indicate a pyogenic process. Characteristic blood changes are found in several diseases, notably glandular and septic processes.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASES CAUSING SYMPTOMS REFERABLE TO THE EXTREMITIES.

§ **428. Routine Examination** and **Classification.**—As a matter of routine, as in other cases, investigate—

First, the LEADING SYMPTOM, which in this instance is very often as visible or palpable to the patient as to the physician.

Secondly, the HISTORY of the case, its mode of onset (acute or chronic), and evolution in chronological order.

Thirdly, examine the AFFECTED LIMB or limbs, their colour and contour, the joints, muscles, bones, vessels, or nerves, as may be indicated; and, finally, examine the VISCERA and the TEMPERATURE.

If there is any visible abnormality in the COLOUR of the hands or limbs, turn to Group I., below, and p. 617.

If the symptoms point to JOINT disease, acute or chronic, turn to Group II., p. 425 (Acute), or p. 436 (Chronic).

If the symptoms point to disease of the MUSCLES, turn to p. 646.

If the symptoms point to disease of the BONES, turn to Group IV., p. 650.

GROUP I. ALTERATIONS IN COLOUR OF THE EXTREMITIES.

This group comprises only the following morbid conditions which may be considered medical. Other alterations in colour or contour,

[1] See also Mr. A. Pearce Gould's "Elements of Surgical Diagnosis."

such as œdema of one limb and varicose veins, have already been referred to on pp. 617, 618. There remain—

1. Erythromelalgia and Acroteric Scleroderma.
2. Gangrene.
3. Raynaud's disease.
4. Dead hands.
5. Intermittent claudication.
6. Cyanosis, clubbed fingers, etc. (§ 423).

§ **429. Erythromelalgia** (a term first used by Weir Mitchell[1]) is a painful redness and swelling occurring in paroxysms, and symmetrically affecting both hands, sometimes the feet, and sometimes spreading to the arms and legs. One side may be more affected than the other, but I have not seen any cases in which both sides have not been involved to some extent.[2] The disorder starts intermittently with tingling and numbness in the extremities (acroparæsthesia), and later on a painful

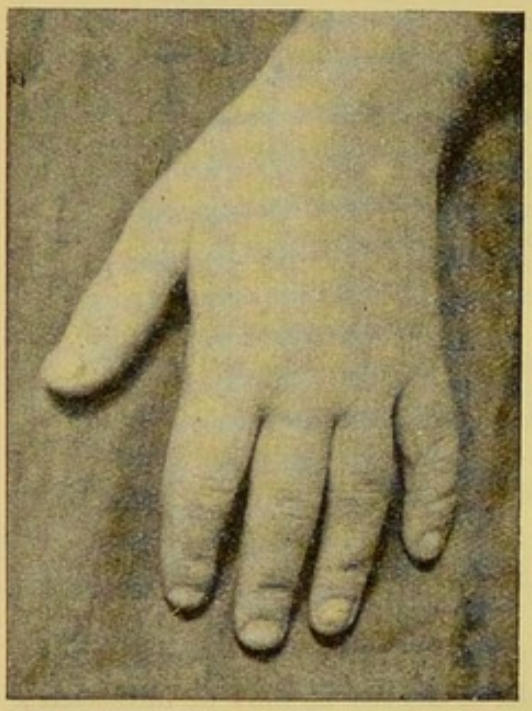

Fig. 122.—Erythromelalgia in a woman aged about thirty.

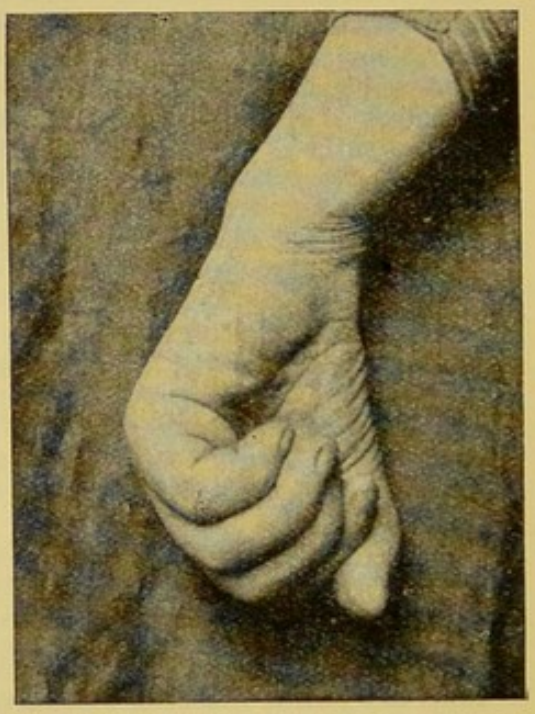

Fig 123.—Erythromelalgia, showing maximum closure of the hand.

redness supervenes. The paroxysms are often determined, and always aggravated by hanging the limbs down, and also by placing them in very hot or very cold water. They are often worse when the patient lies down and goes to sleep, and thus the night may be badly disturbed. The pain and swelling are lessened by holding the hands over the head, or raising the feet. There is no paralysis, but owing to the numbness and swelling the fingers cannot easily be bent. The swelling and redness affect the whole hand (Figs. 122 and 123)—not patches, as in chilblains, lupus, or erythema. On this account the *Diagnosis* is not difficult. In the cyanotic form of *Raynaud's disease* the symptoms start and prevail in one or two finger tips; in erythromelalgia all the fingers and the whole hand are about equally involved. Thickening of the subcutaneous tissues may ensue, and the paroxysms are apt to return even after long intervals. It is a prolonged and very

[1] *Philadelphia Medical Times*, November 23, 1872.

[2] See a clinical lecture on this subject in the *Lancet*, June 1, 1902; and "Lectures on Hysteria," Glaisher, London, 1909.

painful disorder, but it is not fatal, and is in my experience to some extent amenable to treatment.

Etiology.—The female sex is far more prone to the disease; out of thirty-seven consecutive cases which I observed between 1900 and 1902 only two were males. It appears to arise more frequently between the ages of eighteen and twenty-five, and at the climacteric. The rheumatic, gouty, and hysterical diatheses predispose to the complaint, and the determining cause is, in my belief, some altered condition of the blood. Several cases which I have observed have exhibited, concurrently with a severe paroxysm of the erythromelalgic symptoms, erythematous blotches on other parts of the body, and severe "rheumatic" pains in the limbs. The disease must, I think, be regarded as a vaso-motor paralysis due to a toxæmia; associated probably with an inherent instability of the vaso-motor centres controlling the ends of the extremities.

Treatment.—Bromides invariably relieve the condition for a time; arsenic, strychnine, quinine, and other tonics are useful. Ergot sometimes does good. The general health should be attended to, and particularly the digestion. I have tried salicylates once with success. A weak descending galvanic current is the most efficient curative agent in my experience.

Acroteric Scleroderma (Hutchinson) or **Sclerodactylia** is a scleroderma affecting the hands and feet, and sometimes the nose (see a case reported by the author, Proceedings of the Clinical Section, Roy. Soc. Med., March, 1909), in which the skin is bluish and thickened at first, white and atrophic afterwards.

§ **430.** Of **Gangrene,** necrosis, or death of part of an extremity, there are two kinds:

(*a*) In Dry Gangrene the extremity becomes white and cold, then of an ashy and black colour; the part shrivels up, becomes dry and mummified. It is chiefly met with among old people—senile gangrene—and is due to the gradual obliteration of the lumen of the artery supplying the part, combined with more or less cardiac enfeeblement. It is also met with in younger patients in Raynaud's disease, and in cases of embolic blocking of an artery. The artery is tender at the seat of the embolism, and ceases to pulsate below.

(*b*) In Moist Gangrene the part becomes cold, purple, or mottled, and engorged with blood. Blebs then form on the surface, and a bright red line separates the dead from the living tissues. The dead part ultimately sloughs off, and leaves an ulcer. This gangrene is due to venous obstruction, the result of thrombosis, pressure, injury, or inflammation. The gangrene occurring in diabetes is of the moist variety. The treatment of both of these conditions belongs to surgery, but warmth, the posture of the limb, and the administration of diffusible stimulants are points to be attended to.

§ **431. Raynaud's Disease** (Synonyms: Symmetrical Gangrene, Local Asphyxia of the Extremities).—This disease, which was first described in 1862 by Dr. Raynaud,[1] is characterised by local vascular changes in one or more of the fingers, for the most part symmetrically on the two sides of the body, resulting very often in gangrene. Three types or stages of the disease have been described—a syncopal type, due to vascular spasm, an asphyxial type, due to vascular dilatation, and a gangrenous type.

Symptoms.—Usually the first thing noticed is a pallor (*local syncope*) and numbness of one or more of the fingers or toes, usually the corresponding finger or toe on both sides, coming on in attacks, lasting an hour or more. This pale or syncopal stage is generally followed by a reactionary stage of congestion and heat with swelling and lividity (local asphyxia), in which the tip of one or more of the fingers or toes, or the ears, may be of a dark purple hue. There is usually a good deal of pain. Sometimes the pale stage is very definite, sometimes it is wanting, or it may be so transient as to be unobserved. Occasionally the entire hands are involved. After a certain number of these attacks *gangrene* occurs at the tip of one or more fingers or toes, or of the ears, the dead becomes separated from the living part in the usual way, and the ulcer that is left heals normally, but slowly.

[1] "Thèse de Paris," 1862, a thesis written for the M.D. at the Paris University in that year.

Cases have been recorded[1] of extensive multiple gangrene in which the patient has lost entire limbs in this way. The attacks just described may be the only symptom, but in the majority of cases other symptoms of considerable pathological interest may be observed. In a certain number of cases there is a generalised scleroderma, the skin having the appearance of being stretched, and smooth, or sometimes cracked; and in such cases all the fingers are pale and dead-looking, and their entire substance becomes wasted. In a certain number of cases erythematous blotches occur from time to time in different parts of the body, which leave bruise-like stains. The patients are usually highly nervous, and prone to emotional attacks. Transient attacks of hemiplegia and aphasia have been observed, and attacks of paroxysmal hæmoglobinuria, all pointing to vaso-motor irregularities in other parts of the body. Effusion into the phalangeal and other joints may supervene, and may result in ankylosis.

The *Diagnosis* is not usually difficult. In the earlier stages it is closely allied to erythromelalgia, sclerodactylia, and to "dead hands," but these affections are not so localised to the finger's *ends*, are less severe, and never go on to gangrene.

Prognosis.—The disease runs a prolonged course of many years with a series of attacks which become gradually more prolonged and frequent, and the patient gradually becomes more and more helpless. There are many degrees of severity of this disease, ranging from what amounts to no more than a small localised syncope or asphyxia to gangrene of the entire segment of a limb. It is a curious circumstance which I believe to be uniform, that once a finger has become gangrenous the stump does not become similarly affected later on. The subjects of this malady in a marked form rarely reach old age, but usually die of some intercurrent malady.

Etiology.—The disease is more common in women, and especially those of a nervous diathesis. It appears for the first time usually between the ages of fifteen and thirty. Attacks may be brought on by chill or mental disturbances. The pathology of this strange disorder is unknown, but it is undoubtedly a vaso-motor affection, and in view of its symmetry is very probably a derangement of the local vaso-motor centres in the cord. Endarteritis in the peripheral vessels has been described in a few cases.

Treatment.—The affected limbs must be kept warm with cotton wool, and the patient protected from exposure to cold. The most efficacious remedy for the latter is undoubtedly electricity, as originally suggested by Sir Thomas Barlow. The method I have described under erythromelalgia may be employed, and as strong a current as possible used. Sir Thomas Barlow recommends the constant current, but one case under my care derived equal benefit from the interrupted current. Thyroid gland and nitroglycerine have been used, and would certainly be indicated in the syncopal type. The pain is intense, and may require morphia, which acts in a double way in asphyxial cases by giving tone to the vessels.

§ **432. Dead Hands** (Pallor of the Hands).—Many patients—but particularly those who present other evidences of an inherent vaso-motor instability, and are subjects of the gouty or rheumatic diathesis—complain that the hands or finger-tips "go dead," or white, like those of a corpse, and feel numb and cold. These attacks, which rarely last very long, may happen in warm summer weather, without any obvious cause, or consequent on anything which produces a nervous or emotional condition. This vascular disorder appears to be the converse of erythromelalgia, the fingers or hands being pale or shrunken instead of red and swollen. It is akin to acroparæsthesia and erythromelalgia, but it most resembles the slight or early phase of Raynaud's disease. The *Treatment* should be directed to the diathesis upon which they depend, combined if necessary with muscular exercise and electricity. These attacks are not as a rule serious. They often depend upon oral or gastro-intestinal sepsis.

§ **432a. Intermittent Claudication** (Limping) is a rare condition pathologically allied to angina pectoris, and if occurring in the leg is sometimes called angina

[1] Trans. Roy. Med.-Chir. Soc., Lond., vol. xii.

cruris. It is due to sclerosis of the arteries supplying the affected limb, usually of the smaller branches, but sometimes of the main trunks. The symptoms come on when an extra local supply of blood is required, such as during unusual exertion, or when the circulation is at a disadvantage, as when the limb is kept dependent or is cold. The symptoms are cramp-like pains in the extremity associated with pallor or blueness, and the limb goes cold, numb, and powerless. During an attack the pulse of a distal vessel (*e.g.*, the dorsalis pedis) may cease to beat; sometimes gangrene supervenes.

Treatment.—There are four indications for treatment: (i.) To treat the arterio-sclerosis. This is only effectual in the case of syphilis; (ii.) to avoid undue exercise or fixation of the affected limb; (iii.) to avoid cold; (iv.) to increase the peripheral blood-supply by vaso-dilators such as nitroglycerine, by warmth, and by massage or galvanism.

GROUP II. JOINT DISEASES.

The methods of examination and exclusion of fallacies have already been described. Arthritic disorders may conveniently be grouped into acute and chronic:

Acute.	*Chronic.*
I. Acute gout.	I. Chronic gout.
II. Acute rheumatism.	II. Chronic rheumatism.
III. Acute gonorrhœal arthritis.	III. Rheumatoid arthritis.
IV. Acute rheumatoid arthritis.	IV. Osteo-arthritis.
V. Pyæmia.	V. Spondylitis deformans.
VI. Acute specific fevers.	VI. Gonorrhœal arthritis.
VII. Purpura, scurvy, hæmophilia.	VII. Chronic septic processes.
VIII. Traumatism.	VIII. Tuberculous synovitis.
IX. Extension from adjacent bone.	IX. Syphilitic arthritis.
	X. Hysterical joint disorder.
	XI. Neuropathic joint disease.

(*a*) *Acute Joint Diseases.*

Acute joint diseases, a list of which has just been given, come on more or less abruptly, and are as a rule attended by the local and general signs of inflammation. Acute rheumatism is essentially an erratic polyarthritis from the commencement; acute gout usually affects a single joint; most of the other causes start in one joint, but (excepting VIII. and IX.) tend to a progressive involvement of others. It is worth noting that all the acute joint disorders (traumatism being excluded) are due either to some microbic process or to some other blood disorder. These facts emphasise the necessity of investigating the constitutional symptoms, the viscera, and the blood.

§ **433.** I. **Acute Gout.**—Gout is a diseased or disordered metabolism associated with excess of uric acid in the blood, and characterised by recurrent attacks of acute inflammation of the joints with deposition of sodium urate. It is one of the oldest known diseases. Gout occurs in acute, chronic, and irregular forms.

The *Symptoms* of an attack, or paroxysm, of acute gout are usually preceded by gouty dyspepsia, heartburn, flatulence, and weariness after food, fulness and tenderness in the epigastrium and liver, indentation of the tongue, a bad taste in the mouth, and excessive secretion

from the fauces in the morning, scanty, high-coloured urine constantly depositing urates, cardiac irregularities and intermissions in the pulse, restlessness at night, and a tendency to catarrh of the mucous membranes on the slightest exposure. The onset of an attack is usually *very sudden*, often in the middle of the night. It affects preferably one of the smaller joints, and especially the metatarso-phalangeal joint of the big toe. The swelling in a marked case is tense, shining, red, pits on pressure, and is acutely tender, but suppuration never occurs. Other joints may become affected, but the inflammation does not shift from one joint to another as in acute rheumatism. Mild constitutional symptoms are present with pyrexia (102°); there may be muttering delirium at night. The urine contains less uric and phosphoric acid before the attack, and more during it, and may contain a trace of albumen. An attack lasts from two to three days or two to three weeks. After an attack the health is frequently better than it was previously; but the intervals between the attacks gradually become shorter as time goes on, at first two or three years, then one year, then six months; finally the disease becomes chronic, and permanent changes take place in the joint. *Chronic gout* and its associated symptoms are described under chronic joint diseases, § 436.

Varieties.—(1) The symptoms of irregular or *atonic gout* consist chiefly of dyspepsia and a variety of symptoms referable to various organs of the body, supposed to be due to a deposit of gouty materials therein (see complications below). (2) *Retrocedent* or suppressed gout is a term applied to cases where the joint mischief suddenly improves coincident with internal symptoms affecting the digestive tract, the heart or the brain. Thus there may be vomiting and diarrhœa, dyspnœa, arrhythmia, even pericarditis, delirium and coma, or cerebral hæmorrhage. These symptoms are often associated with chronic interstitial nephritis, which is one of the consequences of gout.

The *Diagnosis* of acute gout is not difficult except sometimes from acute rheumatism.

TABLE XXVI.—DIAGNOSIS BETWEEN ACUTE GOUT AND ACUTE RHEUMATISM.

Acute Gout.	*Acute Rheumatism.*
In typical cases: Middle age; male sex.	In typical cases: Youth; either sex.
Preference for smaller joints; never wandering from joint to joint.	Preference for larger joints; usually wandering from joint to joint.
Swelling is usually red, tense, pitting on pressure, acutely tender.	Swelling is hot, but pale, tender only on movement of joint.
Ears show tophi.	No tophi.
Fever may be slight or transient.	Fever always marked and continuous.

Prognosis.—The duration of an attack or paroxysm—which is rarely fatal in itself, depends mainly upon the age and constitutional condition of the patient. When, however, symptoms of suppressed gout come on, the case may end fatally with great suddenness. Gout tends to shorten life mainly by the resulting kidney disease and cardio-vascular changes, and the ultimate prognosis largely depends upon the condition of the urine, which should be of good specific gravity and free from albumen. Among the complications and so-called *irregular* forms of gout (1) chronic interstitial nephritis is the most important. During an attack there is generally a certain amount of albuminuria owing to congestion of the kidney, or deposit of urate of sodium, but this passes off. Gradually, however, after repeated attacks, an interstitial fibrosis takes place in the kidney, which from this association is known as the gouty kidney. Lithuria or lithæmia (§ 249) is by some considered an irregular form of gout. Glycosuria occasionally occurs. Renal calculus occurs in persons of the gouty diathesis who may have escaped joint symptoms. (2) Cardiac and cardio-vascular diseases come next in frequency. The "gouty" heart is one which is "irritable," acts irregularly, causing palpitation and pain, and often great distress. Tachycardia is common. Gout is one of the chief causes of angina. Various valvular lesions and a thickening and degeneration of the arterial walls occur. (3) Bronchitis of a subacute or chronic form is frequent, and in treating this the gouty condition must not be forgotten. (4) Various gastric and hepatic derangements are frequent in gouty subjects. Gastrodynia or an agonising pain in the abdomen is sometimes caused by indiscretions in diet. (5) Eczema and other skin diseases of an intractable kind supervene. Hot, itchy eyeballs, migraine, and episcleritis are often present. Glaucoma and iritis also occur.

Etiology.—Among the *predisposing* causes of gout, age, sex, and heredity are extremely important. (1) As regards age, the disease is rarely met with under thirty, and the tendency increases up to the age of fifty. It rarely starts for the first time over that age. (2) The disease is almost confined to men; if it occurs in women, the attacks are generally very slight. (3) Out of 520 cases collected by Sir Alfred Garrod, 332, or nearly two-thirds, were distinctly hereditary. Like landed possessions the predisposition is transmitted mainly through the male line; but it sometimes skips a female generation and reappears in the sons. (4) Lead in the system is also a strong predisposing factor; painters, glaziers, etc., are very prone to gout and gouty kidney. (5) A fulness of habit, with feeble circulation, is a predisposing factor. (6) Attacks are more frequently met with in the changeable weather of the spring and autumn. The *exciting* causes are (1) the use of alcohol, and especially those forms which contain a high percentage of both sugar and alcohol, such as port wine, brown sherry, Madeira, sweet wines generally, and malt liquor. (2) Nitrogenous food in excess

is credited with being able to produce gout; at any rate, it is commoner in butchers and meat eaters than among vegetarians (compare remarks below). (3) Both of the foregoing causes are more potent when combined with deficient exercise in the open. (4) It is a curious and imperfectly explained fact that gout is very rare in Scotland, at least among Scottish artisans. A possible explanation of this exemption is that the beverage of the Scottish artisan is whisky, while that of the English workman is beer. An attack may be determined by (1) a debauch of alcohol, especially of certain kinds; (2) indigestion; (3) a chill to the surface of the body; (4) severe mental or bodily fatigue; (5) injury to a part, which will not only determine an attack but also the particular part affected. There is, however, a tendency for attacks to recur in the same joint.

The discussion of the pathology would be out of place here, but it may be repeated that the clinical phenomena are due to the presence of uric acid in abnormal amount. This was first demonstrated by Sir Alfred Garrod, who put 2 drachms of blood-serum obtained by bleeding or blistering into a watch-glass, added 12 minims of strong acetic acid, and left a small thread in the fluid; in a day or two it became covered with uric acid crystals. Gout is due to disordered metabolism in some way connected with diminished alkalinity of and excess of uric acid in the blood. Sir William Roberts pointed out that in normal conditions uric acid circulates in the blood, and is excreted in the urine as a quadriurate; in gout the quadriurates accumulate in the blood either from deficient elimination by the kidneys or overproduction. By taking up an atom of the sodium carbonate in the plasma this quadriurate becomes converted into a biurate of sodium. The biurate, being less soluble than the quadriurate, becomes deposited in tissues which have low temperature, poor circulation, and a high percentage of sodium chloride, such as cartilage and fibrous tissue. Dr. Woods-Hutchinson[1] maintains that "the uric acid of gout, like the phosphoric acid which invariably accompanies it, is merely a result and measure of the destructive metabolism of the nucleins of the body cells, chiefly (probably) of the leucocytes, in response to the invasion of poisons or toxins, either organic or inorganic (lead, phosphorus, alcohol, acetone). . . . As most of the toxins . . . are of intestinal origin or entry, *diet in gout should be regulated solely with regard to the diminution of intestinal fermentation and putrefaction.*" Hence, as he shows, meat in itself is not a cause of gout, and in many gouty subjects does not cause gout except when taken together with carbohydrates which decompose in the digestive tract.

Treatment during an attack resolves itself into dietetic and medicinal measures. (1) It follows from what has just been said, that a low diet of milk and farinaceous food, and complete abstinence from alcohol, should be enjoined, unless the heart be fatty, when well diluted pure spirit is the only form permissible. (2) A brisk cathartic with one or more grains of calomel should be given at the onset, followed by frequent doses of saline purgatives, such as Hunyadi Janos water, or Carlsbad. (3) Alkaline carbonates (potassium, lithium, sodium) are very efficacious in promoting the solution of uric acid.[2] (4) Colchicum (combined with

[1] "The Meaning of Uric Acid and the Urates," by Dr. Woods-Hutchinson, the *Lancet*, January 31, 1903, p. 288.

[2] Sir Wm. Roberts first showed that potassium rather than sodium salts should be given in acute gout. Dr. A .P. Luff (the *Lancet*, March 30, 1900) has shown that acute gout is due to the conversion of soluble gelatinous biurate into insoluble

alkaline carbonate) used to be regarded as a specific, and it may be given every four hours (℥ 40 for first dose, then ℥ 12, or F. 87) until the pain is gone, then it should be stopped. (5) Opium may be needed for the pain, but should not be given if there be albuminuria or other evidence of renal changes. (6) Local treatment consists of complete rest, wrapping the joint in cotton wool, and the application of sedatives to the joint, such as lotions of sodium bicarbonate ʒiv., with laudanum ʒii. in ℥x. of water. I have also found a very comforting lotion consisting of sp. vin. rect. ʒiii. ; liq. amm. acet. ʒiii. ; aq. rosæ ʒiii. ; aq. ad ℥xii. When symptoms of suppressed gout come on, employ eliminatory treatment promptly, stimulate if symptoms of collapse follow, and apply counter-irritation (mustard, turpentine stupes) and hot fomentations to the chest or abdomen as the case demands.

Treatment between the attacks—i.e., preventive treatment, resolves itself mainly into a question of diet, and the treatment of gouty dyspepsia. The dietetic treatment is of importance, as in chronic gout. Since uric acid is a derivative of nucleo-proteid, all goods rich in this substance are debarred, such as sweetbread, liver, stock soups, and meat essences and extracts. The flesh of young animals is worse than that of old ; beef is worse than mutton. Harmful vegetables are asparagus, peas, and beans. Carbohydrates must be limited, because they consume more oxygen and are apt to ferment in the intestines ; and the sugars and fats, particularly the cooked fats, because they tax the liver too much. It is probably for this reason that sweet wines, malt liquors, pastries, and all sugar, and sweet and greasy dishes should be forbidden. Plain food and abundance of pure water (to aid elimination) must be ordered. The hygienic treatment consists of regular exercise in the open air, and in aiding elimination. Among the remedies for the elimination of uric acid the most valuable in my experience is piperazine ; it may be given in 5 grain doses three times a day, or in the form of an artificial mineral water. Lysidin, urotropin, and uricedin have also been recommended. An occasional dose of mercury followed by a saline is useful. As regards drugs colchicum is of little use, except in the subacute exacerbations, when it may be given with large doses of potassium iodide until the pain is relieved. Guaiacum, given in the form of the resin, 5 to 10 grains in cachets, is a valuable preventive of the gouty paroxysms. Locally blisters or iodine may be employed near the joints ; or alkaline lotions (carbonate of lithium or sodium, 10 grains to the ounce). Massage, hot air and radiant heat treatment is also useful (see Chronic Rheumatism). Mineral waters (Carlsbad, Vichy, Hunyadi Janos, Friedrichshall) should be freely used. Potassium or sodium bicarbonate has a beneficial effect upon

crystalline biurate. This process is hastened by sodium, and delayed by potassium salts. This explains the beneficial action of potassium salts noticed by the first observer.

the alimentary canal, and it is a very good plan to order a small teaspoonful in ½ pint of warm water to be drunk every night and morning. Some advocate the use of potassium instead of sodium salts, and cases are reported which remained free of gouty symptoms when taking potassium chloride instead of common salts with meals. Visits to Bath, Harrogate, Buxton, and Strathpeffer in this country are undoubtedly beneficial because of the regulation of the life enforced there. Carlsbad, Royat, Aix-les-Bains, and a number of other foreign spas are annually visited. Gouty dyspepsia may be treated by the mineral waters just mentioned, or on the principles laid down in Chapter X. The best tonics are nux vomica, arsenic, and, if necessary, small doses of peptonate of iron.

§ **434.** II. **Acute Rheumatism** (Rheumatic Fever) is an acute febrile disease due to a toxic condition of the blood shown to be microbic by

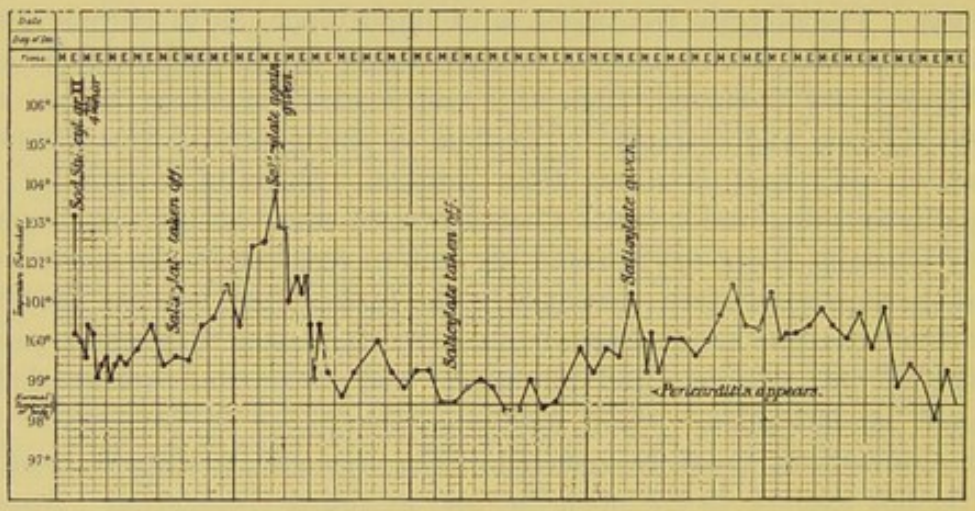

Fig. 124.—Rheumatic Fever.—Henry H——, æt. twenty-two; the chart shows efficacy of salicylates in reducing the temperature until pericarditis appears; then the controlling power of the drug is less.

Poynton and Paine and others, with erratic painful swellings of the joints and a marked tendency to disease of the heart; running a prolonged course of many weeks if untreated, and followed by a great tendency to relapse. It is a disease especially of childhood, when it is capable of many manifestations. The poison of rheumatic fever tends to affect not only the joints, but also all the fibrous, serous and muscular tissues. The joints, endocardium, and pericardium (which, it will be observed, histologically resemble each other) are the favourite situations of the inflammation. Acute rheumatism, unlike acute gout, attacks several joints, usually the larger ones—*e.g.*, the knees, ankles, shoulders. In adults it occurs only in a modified form, with polysynovitis as its most distinctive feature.

Symptoms.—(1) The fever, which may have been preceded by *tonsillitis* for a day or two, comes on in the course of twenty-four hours,

setting in before or at the same time as the joints are inflamed. It is of a continued type (Fig. 124), usually remaining about 102° or 103° F. for some days. The onset of any inflammatory complication in the pericardium or elsewhere is marked by fever, pain, and sometimes delirium, which is otherwise *extremely rare* in acute rheumatism ; in uncomplicated cases the mind remains quite clear throughout. The usual accompaniments of pyrexia are present—viz., the urine is scanty, highly coloured, loaded with lithates, with an excess of urea and deficiency of the chlorides ; the tongue is coated, the pulse quick and bounding, usually over 100. The blood exhibits leucocytosis. (2) In adults there is a profuse *perspiration* with a sour disagreeable odour and an acid reaction, but in children this is unusual ; later on sudaminal vesicles are frequently seen. Erythematous, purpuric, and other *rashes* occasionally appear. (3) The two distinguishing features of the *joint lesions* of acute rheumatism are their wandering or metastatic character, and the absence of suppuration. The effusion into a joint is not very great ; first one joint is affected, by the next day another is involved, the first joint having almost recovered ; finally several may be affected together. The joints are hot and swollen, and though not tender to the touch are acutely painful on the slightest movement. The skin over the joints is either unaltered in colour or shows a faint flush. (4) *Peri- or endo-carditis* are other manifestations of the disease ; the pericardium may be the first serous membrane to be affected. In 150 fatal cases analysed by Dr. F. J. Poynton, evidence of mitral endocarditis existed in 149. There is always some *dilatation of the heart* in rheumatic fever, which is due to the action of the toxin on the cardiac muscle. *Myocarditis* is commonly present, but rarely occurs as the sole cardiac lesion. (5) *Rheumatic nodules* occasionally occur. They are small moveable bodies, usually fibrinous, but may become fibrous. They are generally symmetrically placed on opposite sides of the body and appear on bony prominences and prominent tendons. The commonest places to find them are about the elbows, knees, malleoli, occipital curved lines, posterior spinous processes of the vertebræ, and knuckles. (6) *Chorea* is a common manifestation in childhood, and is often the first sign of rheumatism. (7) Pneumonia, pleurisy, iritis, periostitis, peritonitis, and meningitis, all occur rarely. (8) In untreated cases the fever and local inflammation may subside gradually in four to five weeks, and return again after an interval lasting, perhaps, a few days to a fortnight. In no other acute specific disease, excepting, perhaps, diphtheria, is the blood so deteriorated in so short a time ; the patient shows *grave anæmia* during convalescence. Even after recovery the liability to recurrence is very great, and special care is needed.

Two *variations* of the above symptom-group are met with clinically. In *subacute rheumatism* all the symptoms are milder, and may drag

on for months. *Malignant rheumatic fever* is a very serious form in which the heart is mainly involved, the joints little if at all. An eruption something like typhus may appear, and after a few days the temperature rapidly rises and the patient dies.

The *Diagnosis* of rheumatic fever in the adult is not as a rule difficult. Acute gout is distinguished by its sudden onset, and by the other features mentioned in the table given above. Acute rheumatoid arthritis affects chiefly the larger joints of the fingers (§ 438). The swelling is fusiform, and does not subside under treatment by salicylates. Pyæmia (when arising from some internal cause) may closely resemble rheumatic fever, but in pyæmia the joint inflammation is not erratic, the pyrexia is typical, accompanied by rigors, cerebral symptoms, and an enlarged spleen. Gonorrhœal arthritis usually affects the knees or the small tarsal or carpal joints. The condition is more chronic, and there is a history of gleet. Among the other diseases which sometimes have to be diagnosed are dengue, which has a characteristic eruption; trichinosis, in which the pain and swelling are referable rather to the muscles, and are preceded by gastric symptoms; ulcerative endocarditis, in which the joint swelling is absent, and the temperature intermittent. Osteomyelitis starting near the epiphysis is a condition always to be borne in mind.

In infants, in whom it occurs rarely, its detection may be difficult, as it may closely resemble infantile scurvy, which is known, however, by the swollen gums and failure of treatment by salicylates. Syphilitic epiphysitis and arthritis are known by the great local tenderness and œdema extending beyond the joints, and the rapid improvement under mercury. Rheumatism in infancy may also be mistaken for infantile paralysis.

Prognosis.—The disease is not dangerous to life when it attacks the joints only, but if the heart is affected the prognosis is more grave. One attack predisposes to future attacks. Other untoward symptoms are hyperpyrexia and cerebral symptoms. An attack is grave in proportion to the height of the temperature, the implication of the heart, and the presence of cerebral symptoms. The latter, happily rare, are of the gravest import unless accounted for by salicylates. The visceral manifestations of rheumatic fever are more serious than the diseases itself. The chief of these relate to the heart, which should in all cases be examined daily as a matter of routine.

Etiology.—Age is the most important predisposing factor, acute rheumatism being almost confined to persons under twenty-five, the commonest age being between ten and twenty. It is comparatively rare under ten and extremely rare in advanced life. Males seem slightly more prone to the disease, and heredity plays a considerable part. Among the determining causes may be mentioned exposure to cold or chill, and fatigue. Acute rheumatism is apt to follow an attack of scarlatina or chorea, just as these in turn may succeed an attack of rheumatism.

Rheumatic fever has for a long time been regarded as a blood disorder of *metabolic* and *chemical origin*, lactic acid or its combinations being believed to be the peccant material (Prout, Latham, and others).

Some have attempted to maintain that these metabolic changes were due to some *nervous defect* after the manner of neuropathic arthropathy. On the other hand, an epidemic prevalence in rheumatic fever has long been noticed by careful clinical observers. Dr. Arthur Newsholme in 1895[1] maintained that rheumatic fever was a specific febrile disease, due to the introduction of a micro-organism from without. By a study of death and hospital statistics, and of its seasonal and annual incidence, he showed that the disease was epidemic, its maximum incidence being in January, its minimum in August ; that it bore a direct relationship to a low level of the ground water ; that it varied inversely as the rainfall ; and, finally, that the humidity of the atmosphere had no appreciable effect. The clinical evidence on the resemblance of the symptoms of acute rheumatism to those of septic infection—especially in its involvement of the endocardium and the serous membranes—also supports the view of its being a specific infective disease. In 1900 Drs. F. J. Poynton and Alexander Paine[2] isolated a diplococcus from the blood, exudates, and cardiac valves of rheumatic cases, which answered the tests of specificity. This work has been corroborated by some, but still awaits general acceptance.

Treatment.—Absolute rest in bed is necessary. It is a good plan to fold children in blankets. The diet is that for pyrexia (§ 391). As regards drugs, salicylate of soda or salicin, first adopted by Dr. Maclagan, is rightly regarded as a specific. In most cases it brings down pain and fever within a week, but the treatment must be continued, else the symptoms will relapse and the pain return. The drug must be given in large doses—20 grains every two hours during the first day or two then every four or five hours, till the temperature subsides or physiological symptoms of the drug ensue—viz., headache, deafness, and buzzing in the ears, albuminuria, or delirium. Danger signals to be looked out for as indications to omit the drug are vomiting, acetonuria, drowsiness, and air-hunger—all indications of an acid intoxication.[3] If initiated early and before cardiac or other complications have arisen, this treatment is certain to relieve (Fig. 124). In a few cases, however, especially those in which the joints appear to be less involved than the heart, and those in which the temperature is very high, salicylates may fail. Then antipyrine or antifebrine are remedies of considerable value ; in any case, they relieve the pain and may be good for this purpose alone ; morphia should be avoided. Alkaline carbonates used to be given in large doses, and by some they are still given in combination with salicylates. Dr. Lees[4] recommends very large doses of salicylate of soda. Acid intoxication may be avoided by giving twice as much sodium bicarbonate as salicylate. It is essential that the bowels should be opened before the drug is given, and at least once a day during its administration. The joints should be swathed

[1] The Milroy Lectures, the *Lancet*, 1895, vol. i., pp. 589 and 657.

[2] Dr. F. J. Poynton and Dr. A. Paine, "The Etiology of Rheumatic Fever," the *Lancet*, 1900, vol. ii., pp. 861 and 932.

[3] F. S. Langmead, the *Lancet*, 1906.

[4] Dr. D. B. Lees, Proc. Royal Soc. Med., November, 1908.

in cotton-wool. An alkaline lotion containing opium (F. 35) may be used if any local application is necessary. If, in spite of the salicylates, the temperature remains high, quinine (10 grains every two hours till symptoms arise) may be given. Drugs failing, and the temperature being over 104·5°, a graduated bath should be immediately given, as the condition of the patient requires prompt and energetic measures. If heart complications arise iodides should be given, and many recommend small blisters (the size of a florin) to be applied over the left upper chest or over the heart itself. During convalescence prophylactic treatment is required, because of the liability to relapses and second attacks. The patient should always wear flannel, avoid exposure, and be careful in his diet (see Chronic Rheumatism).

III. **Acute Gonorrhœal Arthritis** (Gonorrhœal Rheumatism, Acute Blenorrhagic Arthritis) is an acute arthritis resembling "rheumatic fever," due not to the rheumatic diplococcus but to infection by the gonococcus from the urethra during the acute stage of gonorrhœa. It is far more frequently met with in the chronic form described in § 441. If the disease arises in the acute stage of gonorrhœa the joint mischief resembles acute rheumatism in all respects excepting: (1) Although the inflammation spreads from joint to joint those first involved do not get better as the others become involved; (2) the temperature has more of an intermittent character than ordinary acute rheumatism; (3) it does not yield to salicylates, but runs a prolonged course of many weeks or months; and (4) there is less tendency to heart complications. The joints rarely suppurate, but the disease is most intractable and may lead to extensive adhesions and distortions of the various articulations. According to Dr. G. F. Still it is by no means unknown in children.

IV. **Acute Rheumatoid Arthritis** may start in a manner indistinguishable from rheumatism, but the joint swellings persist and become more typically those of rheumatoid arthritis later. There is no cardiac affection, nor other rheumatic complication. Salicylates have little effect.

V. **Pyæmia** has already been described in § 383. In some cases of acute general pyogenic infection the joints are not at all involved (septicæmia), but in others of a pyæmic type there is a marked tendency to a suppurative inflammation in and around the joints. It is differentiated from other joint lesions by: (1) the swelling does not shift its position, as rheumatism does; (2) the joint may be red and show evidences of suppuration, though this fact may not be revealed until after death; (3) the constitutional symptoms are very characteristic, especially the wide and irregular range of temperature and the rigors and sweatings; (4) some cause may be revealed in the shape of an internal and external pyogenic focus (p. 538); and (5) there is a high leucocytosis.

VI. Other **acute specific diseases** may—though less frequently than the foregoing—lead to inflammation of joints. The joint disease can be identified only by the presence or history of the disease which it complicates. In *adults* pneumonia and enteric fever may be complicated or followed by a suppurative affection of the joints, often with a fatal issue. More rarely other acute specific fevers are so complicated. In dengue joint swelling is often part of the disease. Cerebro-spinal meningitis is almost always accompanied by synovitis. In *children* it commonly follows scarlet fever, especially when there have been severe faucial symptoms. The lesion may be suppurative. Measles, enteric, and influenza are rarer causes in children. Synovitis sometimes follows the administration of antitoxins.

VII. The three remaining generalised disorders associated with joint trouble are notable in that they are all blood disorders accompanied by some morbid condition of the skin—viz., Purpura Rheumatica, Scurvy, and Hæmophilia.

§ **435. Purpura Rheumatica** (Synonyms: Peliosis Rheumatica, Schönlein's Disease).—In this disease a synovitis resembling rheumatism is associated with a purpuric or erythematous eruption.

Symptoms.—(1) Many joints are affected with considerable pain and swelling; (2) the temperature varies between 100° and 103° F., the pyrexia usually preceding the arthritis by a day or two, and being accompanied by more or less sore throat; (3) the eruption, which usually starts upon the legs near the joints, is of a purpuric, urticarial, or erythematous character. It is attended by a good deal of anæmia. The patient usually recovers in the course of a few weeks, but the disease is apt to recur. The throat symptoms may lead to sloughing of the uvula. Males are more affected than females, and at an age between twenty and thirty. The blood change which underlies this condition is unknown.

In regard to *Treatment*, not much is known as to the appropriate remedies, but it would be worth while trying large doses of calcium chloride. Anti-rheumatic treatment is the line usually adopted.

Henoch's Purpura resembles purpura rheumatica very closely. It is chiefly met with in children, is characterised by recurrent attacks of slight pain and swelling of the joints, cutaneous lesions (for the most part erythematous and purpuric), gastro-intestinal crises, and hæmorrhages from the mucous membranes.

In **scurvy** (§ 410) non-suppurative swellings occur beneath the periosteum near the joints, but the joints themselves are not often affected. The disease is recognised by the spongy bleeding gums, anæmia, and other symptoms of scurvy (*q.v.*).

In **hæmophilia** (§ 411) the larger joints are usually affected. The joint lesion is probably always due to the extravasation of blood or blood serum into the joint cavities, and usually supervenes suddenly on a slight blow or exposure to chill. It not infrequently recurs, and may ultimately lead to ankylosis. It is diagnosed mainly by the history of hæmorrhages in the patient. The condition is met with for the first time most often between the ages of seven and fourteen.

VIII. **Acute Traumatic Synovitis** is recognised by the history of an injury, though one must bear in mind (1) that many constitutional processes, especially gout, are lighted up by a very slight injury, and (2) that in childhood the history of a traumatism may be wanting.

IX. **Extension** from epiphysitis or osteomyelitis (§ 445) or other bone disease in childhood—set up very likely by injury—may produce acute inflammation in a joint, and the serious nature of the condition may be overlooked unless the correct meaning of the pyrexia and constitutional disturbance is appreciated.

(*b*) *Chronic Joint Diseases.*

Joint disorders which may be chronic *ab initio* come clinically under eleven headings :

I. Chronic gout.
II. Chronic rheumatism.
III. Rheumatoid arthritis.
IV. Osteo-arthritis.
V. Spondylitis deformans.
VI. Chronic blenorrhagic arthritis.
VII. Other forms of chronic suppurative arthritis.
VIII. Tuberculous joint disease.
IX. Syphilitic arthritis.
X. Hysterical joint affection, which is often in reality a muscular stiffening and immobility.
XI. Neuropathic arthritis (*e.g.*, Tabes, Syringomyelia, and Raynaud's disease).

Clinically many of these joint diseases resemble each other very closely, both in their physical signs and their history, and many cases are met with which it is almost impossible to place definitely under one or other disease. Moreover, in their pathology we find the same resemblance, for with the possible exception of hysterical and neuropathic arthritis they are all due to a blood change of microbic or metabolic origin.

§ **436.** I. **Chronic Gout** usually supervenes upon a succession of acute attacks (§ 433) ; occasionally it is chronic or subacute from the beginning. The joint is stiff and painful on movement, is very tender, sometimes red, and sometimes masses of urate of soda (chalk stones) can be seen through the skin. The patient, who is usually a male over middle age, suffers also from gouty dyspepsia, irritability of temper, and frequent subacute exacerbations of joint trouble. Tophi are usually present. They consist of nodules of sodium biurate, analogous to the deposits in the joints, and are commonly situated in the cartilage of the ear, near the helix, and in bursal sacs. The urine may contain a little albumen from time to time. The arteries are generally thick, and there is a marked tendency to high arterial tension.

The *Diagnosis* between chronic rheumatism and chronic gout is by no means easy. In an infirmary, where a large number of both diseases in the chronic form are always to be seen, it is usually impossible to classify more than one-third of them. In general terms, chronic gout attacks the smaller joints, the patient is of a plethoric type, and there are concurrent symptoms such as tophi in the ears, interstitial nephritis, or the history of typical paroxysms, which give us some indication of gout. The serum test of acute gout is not of great assistance in the chronic disease (see also table, § 437).

The *Prognosis* of chronic gout is more serious than that of chronic rheumatism, though in both the same crippling of the joints occurs. Interstitial nephritis (granular kidney), is almost sure to supervene sooner or later, and the prognosis mainly depends on three factors : (i.) the condition of the kidneys ; (ii.) the degree of arterial tension ; and (iii.) the condition of the heart, especially of the heart wall. The

complications in addition to those mentioned under Acute Gout are (1) bronchitis; (2) iritis and sclerotitis; and (3) deposits of urates not only in the conjunctiva but in any other tissue of the body. Urethritis may occur in males; stone is not uncommon, and sometimes glycosuria is seen. The patient may eventually die with uræmia, pericarditis, pleurisy, peritonitis, meningitis, or apoplexy. The *Treatment* is described under Acute Gout.

§ **437.** II. **Chronic Rheumatism** is a common affection of the joints. The disease may follow one or more acute attacks, or as is more usual come on insidiously as a chronic affection from the beginning. There are some grounds for believing that chronic rheumatism is pathologically distinct from acute rheumatism. The capsule, ligaments, and tendon sheaths are thickened. The joint is stiff and creaks with adhesions, is generally more or less swollen, and sometimes tender. Sometimes many joints are affected symmetrically; sometimes only one is affected. Ultimately the joint may be considerably distorted, but not disorganised, there being a tendency towards adhesions and fibrous thickenings. When the hands are affected they are in time permanently deformed. The general health, in many cases, is not disturbed, and there is no tendency to either heart or renal affections; though there is a certain amount of anæmia and a variable degree of pain. The disease is never fatal.

The *Diagnosis* from rheumatoid arthritis and chronic gout is sometimes very difficult, and may be impossible until the later stages are reached.

TABLE XXVII.—TABLE OF DIAGNOSIS.

Chronic Rheumatism.	*Chronic Gout.*	*Rheumatoid Arthritis.*	*Osteo-arthritis.*
Either sex; middle life or over.	Generally male sex; over forty.	Chiefly female sex; usually twenty to forty.	Females more than males; forty to sixty.
Poor and debilitated. Insidious onset, unless following rheumatic fever.	Rich and plethoric generally. History of sudden onset and acute attacks with severe pain. Skin over joints red, swollen, and œdematous.	More common in the poor. Onset acute subacute, or insidious. Constitutional symptoms present.	More common in poor and debilitated. Onset, insidious; course, progressive. No constitutional symptoms.
Generally poly-articular. Temporo-maxillary joint not affected.	Only one joint affected at first; usually the metatarso-phalangeal of the great toe.	Generally polyarticular. Temporo-maxillary joint often affected. Spreads from the smaller joints to the larger; terminal interphalangeal joints usually unaffected.	Polyarticular or monoarticular. Temporo-maxillary joint affected; terminal interphalangeal joints usually affected.
Thickening of tendons and ligaments; no bone changes.	Deposits of urate of soda round the joints.	Spindle-shaped enlargement with ulnar deviation and later some fixation. No lipping or osteophytes.	Radial deviation of terminal phalanges. Lipping and osteophytes marked.

Apart from arthritic heredity the *Etiology* is obscure. The patient is generally past middle life, unless chronic rheumatism has followed acute attacks, when the patient may be younger.

Treatment.—Drugs are not nearly so potent in chronic as in acute rheumatism; among those which may be found useful are potassium iodide, guaiacum, quinine, alkalies, sarsaparilla; and in cases with much weakness and anæmia, cod-liver oil is excellent. Local treatment is often more useful—*e.g.*, iodine, blistering, turpentine or other liniments, actual cautery, massage, Scott's dressing, or the application of oleate of mercury. The diet is of considerable importance (§ 212). The patient should avoid sugar and alcohol of all kinds, excepting in very debilitated states, when a small quantity of spirits may be taken. What is known as the Salisbury treatment is certainly very efficacious in some cases.[1] One of the most satisfactory methods of treating chronic rheumatism and many other joint affections is the application of hot air. This may be given in the form of Turkish baths, or in the form of superheated air as given in the Tallerman method,[2] or by radiant heat. On the same principle passive hyperæmia of the joint, induced by bandaging firmly below and above, has given good results. Baths of many different kinds have been used, chiefly sulphur and alkaline baths—see F. 1, 3, and 6. Change of climate does much for those who can afford it. Wintering abroad to avoid cold and damp is undoubtedly most beneficial, especially in a climate like Egypt. Climatic treatment may be combined with baths, as at Aix-les-Bains, Baden-Baden, Buxton, Bath, Carlsbad, Contrexeville, Harrogate, Mont-Dore, Strathpeffer, etc.

§ **438.** III. **Rheumatoid Arthritis.**—The terms Rheumatoid Arthritis, Osteo-arthritis, Rheumatic Gout, and Arthritis Deformans have been used loosely as synonyms, whereas the first two are separate clinical entities and rheumatic gout is an inaccurate term. This opinion was clearly expressed at a discussion at the Medical Society of London in 1906, which was opened by Dr. A. E. Garrod,[3] and the classification then made will be adhered to in the following descriptions.

Rheumatoid arthritis is a general disease, producing synovitis and peri-arthritis, as shown by swelling and pain in the joints. It tends to get well, often after a protracted course, but leaves, not uncommonly, considerable deformity and crippling. It manifests also symptoms of constitutional disturbance.

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[1] A very satisfactory case of this treatment is published in the *Lancet*, 1893, vol. ii., p. 133.

[2] This is applied in the following way: The limb is placed in a specially-made copper chamber made to fit the limb, and the temperature in the interior is gradually increased up to 250° to 300° F. Each application lasts about twenty minutes. The anodyne effect is said to be remarkable, and permanent benefit is reported in some cases.

[3] Trans. Med. Soc. Lond., 1906.

Symptoms.—The onset may be acute, subacute, or chronic. In the first form the condition closely resembles acute rheumatism at first, but the joints prove intractable to the action of salicylates, and later assume the typical characters. In the subacute variety the joints are rapidly affected, but show only slight swelling at first, whilst the temperature is but little raised. The chronic form begins insidiously in one joint, and spreads slowly.

1. The joints usually affected first are the proximal row of the interphalangeal joints of the fingers, and the metacarpo-phalangeal joints ; next the wrists, ankles, and knees ; then the shoulders, and last of all the hips, so that the progression of the disease in the joints is from the peripheræ. The lesions are symmetrical. The temporo-maxillary and vertebral joints are especially liable to be attacked. The distal interphalangeal joints are usually spared. During the active stage the joints are painful, tender, and swollen, and somewhat limited in movement. The swelling is fusiform, due to the fact that the lesion is a combination of synovitis and peri-arthritis ; and there are neither lipping of bone or osteophytes to be felt, nor can grating be elicited. If the active stage is of long duration, this may be followed later by very marked limitation of movement and deformity, due to the formation of adhesions within and around the joint, and to the secondary contraction of muscles. In severe cases partial dislocation or ankylosis may occur. The most common displacement is that of ulnar deviation of the fingers (Fig. 125). The muscles above and below the affected joints are conspicuously atrophied, to a much greater extent than could be explained by disuse. The tendon reflexes are increased.

2. Subcutaneous nodules are sometimes present. Usually these are in the form of flat masses in bursæ, especially the olecranon bursa ; but more rarely they resemble the nodules of rheumatism, differing from them in being more permanent and occasionally tender.

3. The skin is glossy, atrophic, and apt to become parchment-like on the backs of the hands and fingers, which are often cold. Pigmentation is common, and may occur as circumscribed spots like freckles, or as diffuse spreading patches. It occurs especially on the face and neck, and on the backs of the wrists and forearms, but may be general. The forehead may shine like burnished bronze and various tints of yellow and brown are seen by reflected light at different angles.
4. The axillary and inguinal glands are not uncommonly swollen.
5. Constitutional symptoms. There is usually some fever during the active stage, the temperature varying from normal to as high as 102° or 103° F. The pulse is nearly always quickened, and may be from 90 to 100 for years. It may be persistently 120. The general nutrition is impaired, and the patient is usually pale and depressed.

The *Diagnosis* is considered in the table on p. 637.

In *children* certain forms of arthritis occur which resemble the lesions of rheumatoid arthritis in adults. The multiple arthritis described by Dr. G. F. Still, associated with pallor, fever, wasting, and enlargement of the lymphatic glands and spleen, differs from the description given above only in the frequency of affection of the glands and the splenic enlargement. It is probably rheumatoid arthritis modified by the age at which it occurs.

Prognosis.—It should be clearly recognised that rheumatoid arthritis is not a progressive disease, but runs a definite course. The course, however, may be very protracted, and relapses frequently occur. The outlook is serious because of the consequent crippling and deformity. The longer the active stage, the greater is the crippling. It is rarely fatal.

Etiology.—The disease may occur at any age, but is most common between twenty and forty. Females are more often affected than males, in the proportion of about three to one. A lowered resistance

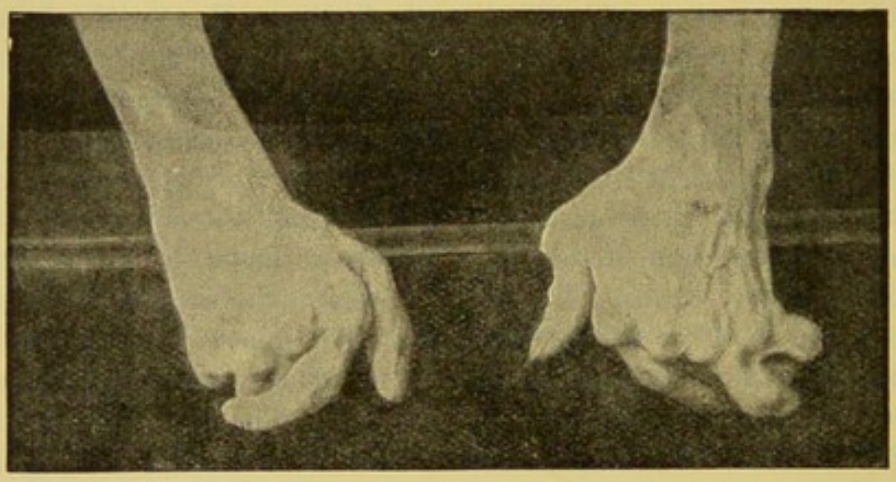

Fig. 125.—Rheumatoid Arthritis in a man about thirty-eight years of age, showing ulnar deflection of fingers.

of the individual frequently precedes an attack, which may in this way follow acute infections, especially influenza, or overwork and anxiety. Dental caries, and pyorrhœa alveolaris, septic conditions of the nose and throat, and ulcerating piles are among the conditions which are said to act as infecting foci. Some regard the disease as due to absorption of toxins from the alimentary canal.

Treatment.—During the active stage *rest* is essential, enforced by the aid of splints if necessary. At the same time, when the pain has diminished, the muscles should be massaged, to counteract wasting and fixation.

The diet, according to Dr. A. E. Garrod, should be abundant and nourishing. When no longer active, spa treatment is beneficial, especially combined with douches and massage. Such is obtainable at Bath, Buxton, Harrogate, and Aix-les-Bains. Brine baths, vapour, hot air, and electric baths also relieve. Bier's method of passive hyper-

æmia, induced by applying a firm elastic bandage above the joint for a few hours, several times a day, is sometimes useful. If the joints are painful, the local iodine vapour bath, as recommended by Dr. Luff, almost always relieves. The joint is washed and dried, and then the skin over it is painted with tincture of iodine. Over this a thin layer of butter muslin is placed. A linseed poultice is applied outside this, and the part is swathed in cotton-wool. *Drugs.*—A combination of guaiacol carbonate and potassium iodide in gradually increasing doses up to the limit of toleration, and persisted in for several months is the most valuable medicinal remedy. Salicylates and aspirin relieve painful joints, or guaiacol, or linimentum potassii iodidi cum sapone, locally applied. Equal parts of guaiacol, menthol, and linimentum camphoræ painted on is very useful. Ionic medication, using potassium iodide, may be tried. Any possible focus of infection such as oral sepsis should be attended to.

§ **439.** IV. **Osteo-arthritis** is a chronic degenerative disease of joints, progressive in character, and occurring chiefly in the elderly.

Symptoms.—The special features of the joints are as follows : The ends of the bones are thickened and lipped. The synovial membrane is also thickened, and thickened fringes can be felt, in some of which cartilaginous bodies are recognisable. These may be pedunculated or free, forming the so-called melon seed bodies. Bony outgrowths or osteophytes are formed, often in great quantity, so that if the joint is moved, scrunching or grating is audible, and by their interlocking, movement is much restricted. True ankylosis rarely occurs. The joint is often distended with fluid, as are also the bursæ, around it. Sometimes the encysted collections of fluid near the joint are unconnected with bursæ, but are lying in spaces bounded by muscles and areolar tissue. Pain is not usually severe, but the joints often feel hot and tingling, and occasionally numb. In severe cases considerable deformity results from absorption of the ends of the bones, so that shortening or displacement is produced. There is no constitutional disturbance in this disease. Muscular atrophy occurs, but is less marked than in rheumatoid arthritis, and does not lead to the same crippling by contracture.

The disease manifests itself in several forms, and may be localised or general :

1. *Heberden's Nodes* is the commonest and best known variety. These are bony outgrowths, which occur at the sides of the distal interphalangeal joints. They are usually painless, but may be painful, and produce numbness and tingling in the fingers. Little bursal swellings occasionally accompany them. In advanced cases the terminal phalanges are bent acutely toward the radial side. The hands are symmetrically affected. This condition may exist alone as evidence of osteo-arthritis, but often accompanies other varieties.

2. *The Carpo-metacarpal Joints of the Thumbs* are not infrequently affected alone, or with Heberden's nodes. The joints are loose and grate, and the bones can be felt to be lipped.

3. *The Knees*, as Dr. A. E. Garrod[1] pointed out, are frequently affected in women at the menopause. Pain and stiffness is noticed on walking or going downstairs, and the knees give way, letting the patient down. If the joint be moved when the patella is depressed by pressure with the thumbs, a fine velvety scrunch can be felt and heard. Later the joint assumes the ordinary deformity.

4. *The Temporo-maxillary Joint*, may be first or solely affected, and the osseous outgrowths may lead to locking so that chewing is impossible. This joint is not so often affected as in rheumatoid arthritis.

5. *The Hip-joint of Elderly Men.*—This is the most important local form of the disease, since it leads to considerable crippling. It is usually unilateral. There are pain and rigidity of one hip-joint with difficulty in adduction. The pain is felt most severely in the groin, but may radiate down the front of the thigh to the knee. From sciatica, with which it is often confused, it is distinguished by the position of the pain, and the fixity of the joint. Wasting may occur later, but is limited to the buttock and thigh. The limb may be shortened. It occurs chiefly in men over fifty.

6. *The Generalised Form.*—In this condition most of the joints in the body may be attacked, including those of the spine. In the hands, the distal interphalangeal joints and the carpo-metacarpal joints of the thumbs are usually selected, and show the characteristic grating and lipping, not the fusiform swelling of rheumatoid arthritis.

Prognosis.—If treated early, temporary improvement may occur, but speaking broadily, the disease is progressive. The form occurring in the hip-joint of old men is very intractable, but that in the knees of women at the menopause more remediable. The crippling is not great, but patients with the joints of the lower extremities affected will often be afraid to get about, because of the fear of the knees giving way.

Etiology.—It occurs most often in women between forty and sixty years of age. It is doubtful whether the joint lesions are in any way specific, since similar changes occur as the result of traumatism of prolonged pressure, as by a tight boot, in hæmophilia from repeated hæmorrhages, and in tabes dorsalis and syringomyelia.

Treatment.—The *diet* should be plentiful and nourishing. Local treatment, by means of hot baths, hot air, or electric baths combined with massage, is very useful. The douche-massage treatment sometimes produces great improvement. The joints should be moved, but not forcibly. These forms of treatment are often more satisfactorily complied with at spas, such as Bath, Harrogate, or Buxton. In the early stages iodide of iron and arsenic should be freely employed, and

[1] Dr. A. E. Garrod, Trans. Med. Soc. Lond., 1906.

are often very beneficial. Aspirin relieves the pain. The local iodine vapour bath recommended by Dr. Luff (*vide supra*) is valuable if pain be present. Ionic medication with potassium iodide may be tried.

§ **440.** V. **Spondylitis Deformans** is a disease formerly classed under rheumatoid arthritis, but now recognised as a morbid entity. The vertebral column and the shoulder and hip joints are most often affected.

Symptoms.—The spine may be quite rigid, so that the name " poker back " is aptly applied. This is due to a synostosis of the vertebræ and ossification of the intervertebral ligaments. A similar change at the hips and shoulders may produce fixation, partial or complete, of these joints also. There is marked kyphosis of the upper part of the spine. The chest is flattened, and the breathing is sometimes entirely abdominal, due to fixation of the costovertebral joints. Nipping of the nerves at their exit between the vertebræ may lead to referred pains around the chest or abdomen, areas of impaired sensation, paræsthesia, and local atrophy of muscle.

Etiology.—It occurs chiefly in adult males, and has been described in children. Three children in one family have been attacked. Syphilis, gonorrhœa, and injury have all been suggested as the cause.

Treatment is symptomatic, and on the same lines as that for osteo-arthritis.

§ **441.** VI. **Gonorrhœal Rheumatism** (Synonyms : Blenorrhagic Arthritis, Urethral Arthritis) is a synovitis associated with a gonorrhœal or other urethral discharge resembling chronic rheumatism in some respects, chronic pyæmia in others. An acute form has been referred to on p. 634, but the disease is nearly always chronic. In this, the chronic and commoner form, the joint affection comes on insidiously during the gleet (often about the fourth or fifth week). According to surgeons who see many of these cases, it is particularly apt to supervene in those cases of gonorrhœa in which the prostatic portion of the urethra is affected, and the extreme vascularity of that part lends probability to this view. When the joint becomes involved the gleet sometimes disappears, a circumstance which may give rise to an error in diagnosis. In the chronic variety there is only slight elevation of temperature of an intermittent or septic kind ; but the general health is always more or less disturbed, and may be so greatly that the patient becomes anæmic and emaciated. Some say that the affection is usually monarticular, the knee being its favourite seat ; but in all the cases I have seen many of the joints have become progressively involved. It never shifts its position, but is progressive. It is apt, moreover, to attack many of the joints usually spared in other diseases, such as the sacro-iliac, sterno-clavicular, and temporo-maxillary, and to settle down the smaller joints of the carpus or tarsus. The affected joint becomes swollen, stiff, and tender, and gradually becomes permanently damaged, resulting, perhaps, in ankylosis or dislocation. The fibrous tissues also are often affected, especially the plantar fascia; pain in this position or in the tendo Achillis is an important diagnostic feature of the disease.

For the *Diagnosis* one has to rely mainly on the history of blenorrhagia, the inveterate character, the tendency to ankylosis, and the fact that it is wholly unrelieved by salicylates.

The *Prognosis* as regards life is favourable, but not as regards recovery. The heart is seldom affected, but there may be pleurisy or iritis. It is more hopeful in younger people and in attacks of recent date. The joint destruction may go on for months or years. The probability of cure depends a great deal on the curability of the urethritis.

As regards *Etiology*, both men and women may be affected. It has been definitely shown that the gonococcus may be present in the joint, with or without strepto- or staphylococci. Special exposure to chill during a gonorrhœa will sometimes determine the disease.

Treatment.—The first indication is to cure the urethritis. This is sometimes extremely difficult, especially when, as generally happens, the gleet takes on a fresh development after its temporary disappearance. The gum resins and ordinary injections are useless in most cases. Urotropin, helmitol, and other urinary antiseptics may be tried. A course of local applications to the urethra is usually required. For this purpose silver nitrate, argyrol, protargol, and potassium permanganate are useful. For the joint mischief one of the most useful applications is Scott's dressing with ung. hydrargyri or oleate of mercury; and the treatment suggested for chronic rheumatism may be tried. The late Mr. Christopher Heath[1] treated cases with belladonna and glycerine applications, and 5 grains of sulphate of quinine every six hours internally, and stated that the patients were usually better in three or four weeks. Iodide of potassium in large doses is recommended by some. A sea voyage is, in my experience, one of the most efficacious remedies. Climatic and bath treatment is also advantageous. It is of the greatest importance to improve the patient's general nutrition. The patient should avoid thereafter any possibility of a fresh attack of gonorrhœa, as this would most certainly be followed by a recurrence of the joint symptoms. Surgical treatment by irrigation of the joint in severe cases is often efficacious. Inoculation with small doses of dead gonococci by the opsonic method is under trial.

VII. Other forms of **Chronic Septic Arthritis.**—Chronic infective arthritis (chronic pyæmic arthritis) is not a form of chronic joint affection generally recognised by authors, so far as I am aware, unless the chronic form of gonorrhœal rheumatism is so regarded. A case which I had under my care for many months (and one other of a similar nature) is described and illustrated on p. 538, which is best explained on the supposition that the joint mischief was due to secondary infection from an internal pyogenic focus—namely, in the appendix. Cases of arthritis have been recorded[2] which occurred during convalescence from dysentery.

§ **442. Tuberculosis, Syphilis, Hysteria, Tabes Dorsalis,** and other **nervous disorders** also affect the joints.

VIII. **Tuberculous Joint Disease.**—Tuberculosis affects chiefly the synovial membrane, but it may commence in the articular ends of the bones. This is *par excellence* the mon-articular joint disease of children.

[1] The *Lancet*, November 25, 1899, p. 1467.
[2] Dr. Paul Remlinger, *Revue de Médécine*, September, 1898, p. 685.

Symptoms.—The onset is insidious, though not infrequently the symptoms date, or are supposed to date, from an injury. The favourite situations are hip and knee-joint, though any joint may be affected. The child may complain of slight pain, which gives rise to limping, for weeks or months before anything is apparent. Generally the disease is in the knee, but sometimes it is in the hip, although the pain may still be referred to the knee, one nerve supply of which is also a branch of the obturator nerve. By-and-by the affected joint swells ; it is pale, and has a pulpy or doughy feel beneath the finger, and fluctuation may be felt. If untreated, the case goes on to abscess formation. The constitutional symptoms consist of an intermitting pyrexia, and general debility which are present even from the very beginning.

The *Causes* are the same as those mentioned under phthisis. The symptoms may date from or be first noted after an injury. The disease nearly always attacks children, though a more destructive form of tuberculous joint mischief does occur in advanced life. It may last for many years, and the prospect of recovery depends very much upon the stage at which it first comes under treatment. If neglected, extensive destruction of the joint may occur, and very frequently tuberculous mischief is found in other organs. The *Treatment* is mainly surgical, but a good deal can be done in the early stages by rest, fresh air, and cod-liver oil.

IX. **Syphilitic Joint Disease.**—Syphilis has of late years been recognised as a cause of chronic joint disease. The differential features of this form of arthritis are as follows : (1) One or several joints may be affected. The synovial membrane may be attacked, leading to a doughy swelling ; or the ligaments or cartilage. There is no " lipping " of the bone ends. (2) The joint manifests no signs of acute inflammation, but there is occasionally some effusion. (3) The pain is very moderate during the day, but subject to nocturnal exacerbations. (4) Other evidences of syphilis are generally present. (5) All but one of the cases I have seen have been in children. (6) The condition is very chronic, and is only partially amenable to iodides.

A Pseudo-Paralysis of Syphilitic Origin occurs in infants, due to the separation of the cartilage from the diaphysis, and is apt to be mistaken either for joint disease or for infantile paralysis. The affected part is, however, acutely tender.

X. **Hysterical Joint Disorder** usually affects the hip or the knee, and it often dates from some trifling injury. The joint is fixed, tender (often more tender to light touches than to deep pressure), and sometimes swollen, and the local temperature of the joint may also be raised. Sometimes there are no physical signs referable to the joint at all. The loss of function may be entirely due to muscular rigidity, and in the case of the hip-joint the condition may very precisely resemble (mimic, as Sir Jas. Paget[1] says) tuberculous disease of this joint. This *Diagnosis*, which is often extremely difficult, rests mainly on (1) the absence of evidence of serious disease in the affected joint when examined under chloroform ; (2) the disproportionate loss of function ; (3) the patient being a female, and the subject of other manifestations of the hysterical diathesis.

The *Treatment* should be mainly directed to the hysteria (*q.v.*). The joint mischief may sometimes be cured by chloroform anæsthesia ; on coming round the patient finds that she can use the joint, and continues to do so. In the author's view[2] these cases are due to a vascular change in the synovial membrane, probably of vaso-motor or toxic origin, albeit slight, and perhaps temporary and evanescent. Many cases of undoubted hysterical joint disease are amenable to salicylates, or alkaline carbonates on the one hand, or to bromides or vaso-motor remedies on the other.

XI. **Neuro-trophic Arthritis** (Synonyms : Neuro-Arthropathy, Tabetic Arthropathy, Arthritis in connection with spinal lesions).—Two diseases of the spinal cord are sometimes, though comparatively rarely, attended with chronic mischief in the

[1] " Lectures and Essays of Sir James Paget," edited by Mr. Howard Marsh, London, 1879.

[2] " Lectures on the Pathology of Hysteria," the *Lancet*, January 20, 1904, and " Lectures on Hysteria," Glaisher, London, 1909.

joints—viz., *Tabes Dorsalis* and *Syringomyelia*. In both it may occur in an early stage of the disease, when nervous symptoms are few or absent, and in both extensive disintegration of the joint may take place, without pain, heat, or redness, and without giving rise to much inconvenience. In *tabes dorsalis* the associated joint lesion is known as tabetic arthropathy, or **Charcot's joint disease,** because it was he who first identified the connection. This lesion may occur without the patient suffering any pain, and but little inconvenience, although the bone ends may be enlarged, and it may go on to extensive disorganisation with increased mobility and new bony formations before the patient seeks a doctor's advice. A case of *tabes dorsalis* is narrated by Prof. J. M. Charcot[1] of a soldier, in whom actual dislocation of both hips was found to have occurred without the patient being aware of any mischief in the joints. Indeed, it had happened while he was on the march. In all such cases the pupils and knee jerks should be examined. The knee is the favourite situation, and therefore it is sometimes difficult to test the tendon reflexes.

Syringomyelia is characterised by muscular atrophy and anæsthesia at the ends of the extremities. Any joint may be involved; in two cases I have seen very extensive disease affected the joints of the upper extremities.

In *Raynaud's Disease* a subacute or chronic synovitis sometimes occurs which is possibly of vaso-motor origin.

GROUP III. MUSCULAR DISEASES.

We are here concerned with lesions situated in the muscular substance as evidenced by pain in the muscle (myalgia) and tenderness, accompanied, perhaps, by some swelling. The causes of pain in the limbs were discussed in § 422. The causes of muscular weakness will be dealt with in the chapter on nervous diseases.

I. Muscular rheumatism or gout.
II. Tumours.
III. Trichinosis.
IV. Idiopathic myositis.

§ **443.** I. **Muscular Rheumatism** is certainly the most frequent cause of muscular pain and tenderness in this country. It is difficult, if not impossible, to separate gouty from rheumatic muscular inflammation. *Symptoms.*—(1) The pain usually comes on quite suddenly; so suddenly indeed, in the case of lumbago, that it is often mistaken for a sprain or rupture of the muscular fibre. It is greatly aggravated by movement and relieved by rest. In the more acute cases it is attended by localised tenderness. (2) Little or no swelling can be detected in the affected muscles, a point of distinction from trichinosis and new growths. (3) It is usually accompanied by a furred tongue and disordered digestion, with constipation and a copious deposit of lithates in the urine. There may be slight pyrexia.

The commonest *variety* of muscular rheumatism is **lumbago,** where the pain is situated in the muscles and fascia of the small of the back. It is usually of very sudden onset, often when in the act of stooping. Rheumatic torticollis is a rheumatic affection of the sterno-mastoid, and is met with chiefly in children. Intercostal rheumatism is a similar

[1] "Nouvelle Iconographie de la Salpêtrière," about 1887.

affection of the intercostal muscles. Lumbago has to be *diagnosed* from other causes of lumbar pain (§ 269). In aneurysm of the dorsal aorta the pain is more continuous, not so easily relieved by muscular rest. In myelitis and meningitis there are other symptoms referable to the nerve trunks, sensory, or motor. Muscular rheumatism, though not lethal, is very painful and incapacitating, and is very prone to recur.

Etiology.—Muscular rheumatism generally arises in gouty and rheumatic subjects who present other evidences of lithæmia (§ 249). It is usually determined either (i.) by a chill, especially after prolonged exertion accompanied by profuse perspiration, or (ii.) a muscular strain. It comes on especially in cold and damp weather. Cold or damp alone do not seem able to produce it; it is when the two occur together, and especially when combined with errors of diet, that the disease is chiefly produced. Sugar, rich foods, and sweet heavy wines are most potent for evil. Professor Ralph Stockman[1] has found fibrous nodules in the muscles, tendons, fascia, and nerves in chronic rheumatism, which swell and become more painful in cold, damp weather.

Treatment.—The treatment must be directed on the lines laid down for gout and rheumatism in other parts, and consists mainly of free purgation with calomel and salines, of alkalies, sodium salicylate, or guaiacum (F. 96); quinine and iodide of potassium are useful in protracted cases. Rest is necessary for the pain; and even morphia may be required. Locally, counter-irritants are best. Lint soaked in a mixture of equal parts of liniment of belladonna and chloroform should be kept over the muscles (or in hospitals, liniment of turpentine does equally well, and is very much cheaper); it should not be covered up with oil silk, or it will blister. Dry heat is very efficacious. The galvanic current allays the pain and promotes recovery in some cases. Ionic medication with sodium salicylate is sometimes efficacious. Warm underclothing and a flannel belt may prevent recurrence. The diet must be very simple (milk chiefly), and sugar and alcohol should be avoided (see also § 437). Sometimes an attack may be aborted by massage and a Turkish bath. Dr. Stockman (*loc. cit.*) advises that the nodules be massaged perseveringly, in spite of the pain caused, for six or eight weeks; and that this should be followed by exercises to stretch the muscles and aponeuroses.

II. **Tumours** in the substance of the muscles may give rise to pain and tenderness, usually associated with swelling. The pain and tenderness are in this case strictly localised, at any rate at first, to the seat of the disease, and there is a thickening or tumour discoverable on careful palpation. In some cases—*e.g.*, syphilitic and malignant growths, the lymphatic glands in the neighbourhood are enlarged. The chief tumours affecting muscles are (*a*) innocent—syphilitic gumma; abscess, which may arise from a gumma, or be of inflammatory origin; innocent neoplasms such as fibroma, lipoma, angioma, and hydatid or cysticercus cysts. (*b*) Malignant growths, sarcoma, and carcinoma (by extension). First determine whether the

[1] *Brit. Med. Journ.*, February, 1904, p. 477.

swelling is inflammatory or non-inflammatory, malignant (and rapidly growing) or non-malignant, by an investigation of the swelling, the glands, the history, and the concurrent symptoms. The diagnosis and treatment is mainly surgical.

§ **444.** III. **Trichinosis** is a disease due to the presence of a nematode worm (the trichina spiralis), in the intestinal canal, and the dissemination of the embryos in the blood and the muscular system, consequent on the ingestion of "measly" meat (usually pork) insufficiently cooked. It is rarely met with in this country.

The female *adult* or intestinal worm measures about $\frac{1}{8}$ inch, the male slightly less. In fæcal examinations for the parasite it should be remembered that the characteristic feature is the "cell body" at the anterior part of the intestine of the parasite. The *larvæ* (Fig. 126) or muscle trichinæ are found in infected muscle, where they are visible by the aid of a $\frac{1}{2}$ or 1 inch lens. Each consists of an ovoid capsule (translucent, or infiltrated with lime salts, according to the length of time it has existed) containing two or more embryos coiled up within it. The *embryos* are 0·6 to 1 mm. long, with pointed head and rounded tail. It is the presence of these larvæ which give to pork or other infected meat a characteristic "measly" appearance which is visible to the naked eye. Trichina is chiefly conveyed to man by "measly" pork, insufficiently cooked; the capsules are then digested, and the embryo set free in the intestinal canal of the patient. During the ensuing week the embryos grow and attain sexual maturity; each female being capable of producing several hundred embryos. After fecundation the female worm penetrates the walls of the intestinal canal, and hundreds of embryos are discharged into the lymph spaces, or directly into the blood stream, and are carried chiefly to the muscles, where after two or three weeks they become encysted and assume the larval form. In the course of months or years the larvæ become calcified, but they have been found to be alive and capable of developing ten or more years after their entrance.

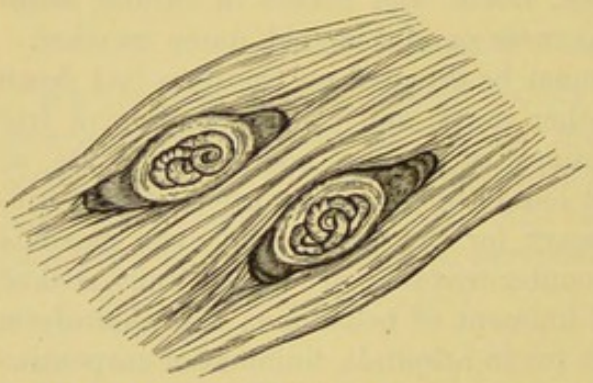

Fig. 126.—Larvæ of the TRICHINA SPIRALIS encysted in muscle.

Symptoms.—The disease runs a course of several weeks, and consists of three well-marked stages. The *first* stage, before the larvæ begin to migrate, lasts usually about a week or ten days, during which the symptoms are those of gastro-intestinal disturbance attended by abdominal pain, with more or less diarrhœa and vomiting. The *second* stage, which lasts from two to three weeks, is coincident with the active migration of the embryos. This gives rise to acute universal muscular pain, tenderness, and sometimes swelling. The wandering of the embryos in the muscles produces shortening and rigidity; the biceps seem specially apt to be affected, resulting in a typical flexion. In severe cases, movement of the affected muscles—*e.g.*, turning the eyeball, moving the limbs, chewing, swallowing, etc., aggravates the pain. Pyrexia, of a remitting or intermitting type, is present, with profuse perspiration, inability to sleep, and possibly delirium. In some cases there is extreme dyspnœa from implication of the diaphragm. There may be general œdema, starting in the face, and later on emaciation. In slight cases the muscular and other symptoms may be so insignificant as to be altogether overlooked. In the *third* stage the acute symptoms gradually subside, great muscular weakness ensues, and recovery is slow. This stage is apt to be interrupted by various complications, especially pneumonia, pleurisy, or persistent and intractable diarrhœa.

In slighter cases the *Diagnosis* from muscular rheumatism or other diseases in this group may present difficulty, though the widespread muscular tenderness of

trichinosis, the history of gastro-intestinal symptoms, and the epidemic occurrence in a whole family should aid us. The stools after a large dose of calomel may be searched for the adult worm, and it has been suggested to remove a small portion of muscle by means of a harpoon for microscopic examination. Some cases are mistaken for enteric fever, and *vice versa*. In trichinosis, however, Dr. T. R. Brown has shown that there are characteristic and distinctive changes in the blood, in the shape of a very marked leucocytosis—reaching 30,000 per cmm. or more—due mainly to an enormous increase in the eosinophil cells, which may amount to 50 per cent. of all the leucocytes as compared with the normal 2 to 5 per cent.

Prognosis.—The disease not infrequently ends fatally between the third and sixth week ; the mortality varying from 2 to 30 per cent. The intensity and duration of the symptoms is a fair measure of the prospect of recovery. Death may occur from (i.) diarrhœa, (ii.) asphyxia (from involvement of the respiratory muscles) ; (iii.) from exhaustion ; or (iv.) from hæmoptysis or pneumonia. In any case health may not be restored for several months.

Etiology.—The disease is due entirely to the ingestion of " measly pork " or other meat, and occurs in an epidemic form in families and towns. It is much more frequent in Northern Germany, where underdone pork or ham is a popular food, than in England and France. Thorough cooking will destroy the parasite, but in large joints the cooking temperature may not be sufficiently high to destroy the parasite in the interior. All meat, particularly sausages and pork, should be thoroughly well cooked. It is said that 170° F. will destroy the larvæ, but that 107° F. will do if maintained long enough. This temperature must permeate to the interior of the meat.

Treatment.—If the patient is seen within two or three hours after the ingestion of infected meat an emetic should be given. If the disease is discovered within twenty-four or thirty hours the gastro-intestinal tract must be thoroughly cleared out. Glycerine in large doses has been recommended in the first stage for its hygroscopic properties to destroy the nematode. Filix mas, kamala, santonin, thymol, and turpentine are also recommended. If, however, the second stage is reached, and the embryos are migrating, the treatment must be symptomatic, because nothing will destroy them. For the pain and tenderness, opium and other anodynes may be required.

§ **445.** IV. **Myositis**, or inflammation and swelling of the voluntary muscles, is a rare condition ; only a few cases have been placed on record. Three forms are recognised by authors : (*a*) A *localised* form, in which pain, tenderness, swelling, and impaired movement are localised to one muscle or a group of muscles ; (*b*) an *acute generalised* form in which these symptoms, accompanied perhaps by œdema and redness of the skin, are more widespread ; (*c*) a *progressive generalised* form of myositis in which the disease runs a prolonged course spreading from muscle to muscle. An example of this variety is reported by Dr. J. K. Fowler.[1] The disorder began in the left thigh, and in about two years became general. It was characterised by painful knotty swellings, and followed by general wasting of the muscles. In *myositis ossificans* the process goes on to the formation of bone.

The *Diagnosis* of myositis appears to offer some difficulty, especially from trichinosis. A microscopic examination by means of a harpoon would afford aid in the acuter cases, and the prolonged progressive course of myositis aids the diagnosis of chronic cases.

The *Causes* of idiopathic myositis are very obscure. It seems probable that Syphilis may have been in operation in some cases. Glanders and Actinomycosis also affect the muscles. The acute localised form is generally the result of injury, or spread from surrounding structures. Some think that the generalised varieties are essentially of nervous origin. Dr. F. Parkes Weber, who has kindly supplied me with several references,[2] regards such cases as neuro-dermatomyositis.

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[1] Clin. Soc. Trans., April 23, 1897.

[2] (1) Max. Levy-Dorn, " Polymyositis and Neuritis," *Berl. Klin. Woch.*, September 2, 1895 ; (2) E. Wagener, " Ein Fall von Acuta Polymyositis," *Deut. Arch. f. Klin. Med.*, 1887, p. 241 ; (3) Unverricht, " Polymyositis Acuta Progressiva,"

In regard to *Treatment*, iodides may be tried. In Dr. Fowler's case the only treatment which proved of service consisted of warm baths, which relieved the pain and stiffness.

GROUP IV. BONE DISEASES.

It would be out of place to deal at any length with diseases of the bones, which belong in a special manner to the surgeon. Nevertheless, these diseases frequently come under the notice of the physician, especially in their early stages. Pain and deep-seated tenderness are often their chief and sometimes their only symptom. Pyrexia and constitutional derangement may be present. Deep-seated swelling and deformity may appear later, and if the bone is superficial, œdema and redness of the skin. The majority of bone diseases are chronic.

Acute Bone Diseases.

I. Acute osteomyelitis.
Acute periostitis.
Acute epiphysitis.

Chronic Bone Diseases and Deformities.

I. Rickets.
II. Chronic periostitis, osteitis, caries, and necrosis.
III. Tumours of bone.
IV. Acromegaly.
V. Achondroplasia.
VI. Pulmonary osteo-arthropathy.
VII. Osteitis deformans.
VIII. Mollities ossium.
IX. Leontiasis ossea.

§ **446. Acute Infective Osteomyelitis** (Synonyms: Acute Periostitis, Acute Necrosis).—This disease, which used to be known as acute necrosis, and later as acute periostitis, is an acute inflammation affecting one or more of the bones, starting usually beneath the periosteum, rapidly involving the whole bone, accompanied by severe constitutional disturbance—on which account the case comes under the notice of a physician. This is the only really acute bone disease, though acute symptoms very closely resembling those of osteomyelitis may arise in association with a LOCALISED PERIOSTITIS such as results (especially in children) from an injury.

The *Symptoms* of acute osteomyelitis are (1) pain of a very severe character coming on suddenly, and attended by extreme tenderness, starting usually at the articular end of the bone—very often the tibia—attended in the course of a day or two by swelling of the limb, at first pale, and afterwards red, as the inflammation makes its way towards the surface. (2) The constitutional symptoms come on suddenly, and are very marked. The temperature is high, and there are rigors and great prostration. The *Diagnosis* from acute rheumatism, which it may at first resemble, because of the pain starting near a joint, is made by the fact that in rheumatism the pain and swelling are confined to the joint, by the early involvement of other joints, and by signs of cardiac complications. Acute epiphysitis is mentioned below. The *Prognosis* is always very grave. If the patient survive

Zeit. f. Klin. Med., vol. xii., p. 533; (4) Unverricht, "Dermatomyositis," *Deut. Med. Woch.*, 1891, p. 41; (5) Hepp, "Ueber Pseudotrichinose," *Berl. Klin. Woch.*, 1887, p. 297, etc.; (6) Senator, in *Deut. Med. Woch.*, 1893, p. 933; (7) Senator, in *Zeit. f. Klin. Med.*, 1889, xv., p. 61.

the initial constitutional disturbance the malady leads to abscess, necrosis and tardy convalescence. The most frequent complications are septicæmia and the extension of the inflammation to a joint. *Etiology.*—Acute osteomyelitis is more frequent in children under the age of puberty. It sometimes dates from an injury, though due to microbic infection. It may occur as a sequel to the continued fevers. *Treatment.*—A surgeon should be called in at once to consider the advisability of free incision, drainage, and other surgical measures.

Acute Localised Periostitis may arise from traumatism, and if not infected it soon subsides. If infected either from a wound or from the blood, suppuration and necrosis take place, and the condition becomes chronic (§ 448), which is more common than acute periostitis.

Epiphysitis is inflammation beginning in the growing line, which in early life separates the epiphysis from the shaft of the long bone. The *acute* form is met with in very early infancy. Suppuration soon sets in, and spreads to the joint, forming abscesses. It may resemble acute osteomyelitis, but the profound constitutional disturbance is lacking. It is distinguished from acute rheumatism by the age of the patient, and by the development of abscess. In the *chronic* form the process is much slower, and is of interest chiefly in relation to the diagnosis of rickets, from which it differs in being localised to one joint. As regards causation the acute form is generally due to an injury and sepsis ; the chronic form is generally associated with syphilis or tubercle.

TABLE XXVIII.—CHRONIC BONE DISEASES AND DEFORMITIES.

	Occurrence.	*Age Period most Affected.*	*Number of Bones Affected.*
Rickets.	Very common.	Infancy (six months to two years).	Many bones symmetrically.
Ch. periostitis, osteitis, caries, and necrosis.	Common.	Early life.	One bone ; may be several in syphilis.
Tumours.	Relatively uncommon.	All ages.	Starts in one bone.
Acromegaly.	Rare.	Twenty-five to forty.	Extremities and face.
Achondroplasia.	Very rare.	Congenital.	Whole skeleton.
Pulmonary osteo-arthropathy.	Rare.	After middle age.	Arms chiefly.
Osteitis deformans.	Very rare.	After middle age (men).	Long bones.
Mollities ossium.	Very rare.	Twenty-five to thirty-five (women).	Whole skeleton.
Leontiasis ossea.	Very rare.	After middle age.	Facial and cranial bones.

§ **447.** I. **Rickets** (Synonym : Rachitis) is a constitutional disorder of childhood attended with epiphyseal enlargements and other deformities of the skeleton. It was described by Glisson in 1675.

The *Symptoms* for which we are consulted, coming on between the sixth and twelfth month, are delayed dentition and walking, or the child cannot "sit up" ; gastro-intestinal disorders ; bronchitis ; sweating about the head ; or a generalised tenderness and restlessness. In the limbs the disease is typically shown by the *enlarged epiphyses*,

affecting most, if not all, of the long bones. The rib-ends are the first to show the enlargement at their junctions with the costal cartilages and thus produce an appearance of " beading "—the " rickety rosary." The long bones often curve, especially near the epiphyseal junction, a condition most commonly seen in the tibiæ and fibulæ. The convexity of the curve is outwards, and greenstick fractures may be produced by slight injuries. The spine has a general backward curvature when the child sits up ; scoliosis may ensue later. The head is square shaped, both the frontal and parietal eminences are prominent. The anterior fontanelle may remain open after the second year (normally it should close between fifteen months and two years of age). There may be craniotabes (thinning of the skull bones) especially of the occipital region. The body may be emaciated or plump and flabby. The chest is deformed, due to sinking in at the costochondral junctions, so that the sternum and cartilages stand out prominently in front, and are united to the ribs along a deep lateral groove (see Fig. 35, § 74). Another groove (Harrison's sulcus) runs transversely across the chest, just above the lower costal margin. The liver and spleen are both enlarged in advanced cases ; the costal margin is everted, and the belly is prominent. The joints are loose, permitting of hypermobility. There is instability of the nervous system, sometimes evidenced by convulsions, tetany, or laryngismus stridulus ; nodding spasm is more rarely present.

Diagnosis.– The disease may have to be diagnosed from hereditary syphilis, in which there may be enlargement of the epiphyses, but this occurs usually only in one bone, and is accompanied by other undoubted signs of syphilis. It may also need to be distinguished from the other diseases of this group which affect children. Infantile paralysis soon exhibits muscular wasting. Achondroplasia (§ 449) is a rare condition which has only recently been distinguished from rickets. In infantile scurvy (§ 413) the swellings affect the shaft rather than the epiphysis, and are painful. The diagnosis of rickets, hereditary syphilis, and hydrocephalus is given in the form of a table on the next page. It is chiefly in regard to the form of the head that the diagnosis between hydrocephalus and rickets presents any difficulty.

Prognosis.—The disease when taken in hand before osseous changes are marked is readily amenable to treatment. If untreated it leads to deformity. If death occurs it is due to some of the common complications, notably bronchitis, or gastro-intestinal disorder, laryngismus stridulus, or convulsions. Spinal, pelvic, and other deformities or hydrocephalus may result, and the growth is stunted. Genu-valgum (knock-knee), genu-varum (bow-leg), and flat-foot often occur.

Etiology.—Rickets rarely appears earlier than six months or later than the second year, though Sir William Jenner described a late rickets arising up to the ninth or twelfth year. Dr. Samuel Gee col-

lected 635 cases, and found it seldom began later than twelve months, and his latest case was only twenty months old.[1] Both sexes are equally affected. The disease is more frequent in cities, and impure air may play a part in its production, but it may also be found in the country and among the wealthy. Too prolonged lactation, suckling during pregnancy, and too rapid pregnancies in the mother predispose to rickets in the offspring. However, it is now generally admitted that the essential cause of rickets is a defect in the diet. The diets on which rickets is produced all show, according to Dr. Cheadle, a deficiency in two constituents—animal fats and proteids. A deficient assimilation of lime salts also appears to aid. Too early or excessive administration of carbohydrates and particularly the proprietary infant foods rich in starches is in actual practice a frequent cause.

TABLE XXIX.—DIFFERENTIAL DIAGNOSIS.

	Rickets.	*Hereditary Syphilis.*	*Hydrocephalus.*
I. History.	Gastro-intestinal irritation, sweating about head. Improper feeding.	Snuffles and rash. Miscarriages in mother.	Congenital, or may be acquired after some meningeal inflammation, or due to tumour pressing on veins.
II. Age of patient.	Commences in infancy and begins to show itself during the first or second year.	Symptoms first appear third week and the third month.	Congenital or acquired.
III. Shape of head.	Often compressed antero-posteriorly. Frontal eminences marked.	Irregular prominence on each frontal and parietal bone. Skull has been called natiform. Depressed bridge of nose.	Bulges in all directions. General tendency to assume a globular form.
IV. Fontanelles.	Close late.	Appear to be depressed in the hollow between the four prominences.	Bulging, separation of the bones at the sutures.
V. Other peculiarities.	Epiphyseal enlargements, delayed dentition, etc.	Pegged and notched teeth. Scars about mouth, palate, etc.	Stunted growth, mental deficiency.

The *Treatment* must be mainly dietetic (compare children's dietary, § 212*a*). Carbohydrates must be reduced, and raw meat juice, milk-casein, or other proteid food-stuffs, and good milk and cream added. Cod-liver oil is the best drug to give, either alone or combined with lime preparations and with iron. Phosphorus $\frac{1}{120}$ grain three times a day in oil is recommended. One of the best remedies for unhealthy stools is F. 64. The child must not be allowed to walk lest the bones yield and produce permanent curvatures. Fresh air and sunlight are necessary adjuncts to dietetic and tonic treatment. Should the bones have

[1] Quoted by Dr. Hilton Fagge, "Prin. and Pract. of Med."

already some degree of curvature further yielding is prevented by placing the legs in restraining splints, which if carried beyond the feet will efficiently prevent any attempt at walking or standing when the mother's back is turned.

§ **448.** II. Under **Chronic Osteitis** and **Periostitis** are included a number of tuberculous, syphilitic, and other conditions leading to caries, necrosis, and other anatomical changes in the bone. Osteitis and periostitis may be dealt with together, for although the disease may start in the bone or the periosteum it soon spreads to the other.

The *Symptoms* of osteitis and periostitis may have come on with acute pain, redness, and swelling ; but more frequently they come on insidiously with hardening, thickening, or enlargement of the bone. These symptoms may be followed by softening (caries) or death of a portion of the bone (necrosis) with signs of abscess formation.

Causes and their differentiation : (1) *Traumatism* alone, without sepsis or toxæmia of some kind, is a rare cause of chronic periostitis or osteitis. Traumatism is recognised by its history, and by the fact that only one bone is affected. (2) The favourite seat of *tubercle* is the epiphysis, where it induces a chronic epiphysitis, especially in the neighbourhood of the hip or knee. Sometimes it gives rise to osteitis, and when this occurs in the fingers it results in a characteristic thickening of the phalanges known as " strumous dactylitis." In any position it may go on to caries or necrosis. Tuberculous affection of the bones is recognised by (i.) the youthful age of the patient ; (ii.) a tuberculous history ; (iii.) by the characteristic intermitting pyrexia ; (iv.) signs of tubercle in the lungs and elsewhere ; (v.) by the chronicity of the process ; and (vi.) the frequent limitation to one bone. (3) *Syphilitic* affections of the bones are very common both in the acquired and the hereditary disease. (*a*) *Acquired syphilis* may take the form of a chronic diffuse or localised periostitis (nodes), or, on the other hand, a diffuse or a gummatous (localised) osteitis. It is recognised by (i.) the characteristic flying pains in the limbs ; (ii.) the nocturnal pains in the bones, which are such a frequent manifestation of syphilis ; and (iii.) other evidences of syphilis. (*b*) *Hereditary syphilis* may give rise in childhood and early life to the same lesions as the acquired disease. In infancy (in addition to the foregoing) chronic suppurative osteochondritis (chronic epiphysitis) is apt to arise and to be mistaken for rickets. In this condition one or several bones may be affected, but it never presents the same symmetry as rickets. The deformities resulting from hereditary lesions (§ 404) and the physiognomy are very often characteristic—the bosses on the frontal and parietal bones (Parrot's nodes), the depressed bridge of the nose, scars about the angle of the mouth, Hutchinson's teeth (Fig. 3, § 11), and perhaps keratitis. (4) Rheumatism and gout may give rise to chronic periosteal thickening, or periosteal nodes.

For the adequate *Treatment* of most of these different conditions, rest and surgical aid are necessary. The treatment of the tuberculosis, syphilis, rheumatism, and gout have already been dealt with.

III. **Tumours of Bones** may commence with pain, tenderness, and swelling like chronic periostitis. The chief *innocent* tumours are EXOSTOSES, which may occur on almost any bone, and ENCHONDROMATA, which are commonest on the metacarpals and phalanges. Both are usually multiple. The *malignant* tumours are either SARCOMA (especially myeloid sarcoma) or CARCINOMA. In both the swelling of the bone is more rapid, and reaches a greater degree than in any of the other causes of swelling above mentioned, and as a rule they are limited, at any rate at first, to a single bone. Sarcoma of bone may occur at any age ; carcinoma is limited to advanced life, and accompanied by the usual cachexia.

§ **449.** There are six rare forms of chronic bone disease which need to be mentioned.

IV. **Acromegaly** is a rare disease first described by Dr. Pierre Marie, leading to enlargement of the skeleton. The patients generally apply for treatment for some

other malady, though sometimes they apply on account of the awkwardness of their movements, and sometimes they complain of obscure pains in the limbs. The aspect is very characteristic. The bones and other tissues of the hands and feet become markedly elongated and hypertrophied, though the growth is so gradual as to escape the patient's notice. The cranium is increased, but not so much in proportion as the face, which is egg-shaped, the lower jaw representing the large end of the egg. The lower jaw especially is enlarged, and may project beyond the upper jaw. The nasal bones are also enlarged, whilst the thickening of the soft parts causes hypertrophy of the ears, eyelids, nostrils, and tongue. Later in the disease there may be a similar enlargement of the bones of the limbs and the thorax, and kyphosis of the spine. Sometimes there is temporal hemianopsia, with gradual optic atrophy.

Diagnosis.—Myxœdema resembles acromegaly, but it is known by its round or " moon-shaped " face, the dry skin, and the absence of all bony enlargement or muscular weakness. Pulmonary osteo-arthropathy (see below).

Acromegaly occurs rather more frequently in women, generally beginning about the twenty-fifth year. Changes in the pituitary body, either hypertrophy or tumour, have been found in all the fatal cases. It has been suggested that gigantism and acromegaly are one and the same disease, and that both are due to disordered function of the pituitary gland, and it is worth noting that in the skulls of certain giants the sella turcica has been found to be considerably enlarged. In some cases there have also been changes in the thymus or the thyroid glands, and mediastinal dulness has been made out during life in the position of an enlarged thymus.

Treatment.—Acromegaly runs a very prolonged course of many years, and no known treatment seems to affect it. Extracts of pituitary gland and thyroid have been tried. The patient generally dies of some intercurrent malady.

V. **Achondroplasia** (Synonyms : Fœtal Rickets, Chondrodystrophia Fœtalis) is a rare condition of infancy leading to dwarfism and generalised deformity, which until a few years ago was probably confused with cretinism on the one hand, or with the deformity resulting from rickets on the other. Fig. 127 represents a case exhibited at the Clinical Society of London by Mr. W. Turner, to whom the author is indebted for the photographs. There is a generalised symmetrical shortening of the diaphyses (producing characteristic shortening of the limbs) with considerable thickening of the epiphyses (producing enlargement of the articulations), due to hyperplasia of the cartilaginous ends of the bones. Consequently the stature is stunted, the fingers and toes taper and are abducted from one another, the cranium is large, the face small, and the bridge of the nose depressed. There is a characteristic waddling gait. The disease is congenital, and dates from birth. The mental deficiency, facial aspect, and the changes in the hair and skin characteristic of cretinism are absent, and cases do not exhibit the constitutional symptoms or characteristic changes of rickets in the skull. It is ascribed to a premature union of the diaphysis and epiphysis, so that lengthening of the large bones is arrested.

VI. **Pulmonary Osteo-arthropathy** is a chronic hyperplasia sometimes associated with chronic pulmonary disorders. There is enlargement of the hands and feet, and of the lower ends of the long bones of the legs and forearms, but the face and head are not enlarged. The nails are curved over the enlarged terminal phalanges, " filbert nails."

VII. **Osteitis Deformans** (Synonym : Paget's Disease) is a somewhat rare disease coming on after middle life, mostly in males, and consisting of a very chronic enlargement of the bones, both in diameter and in length. The histological change is a rarefying osteitis with enlargement of the Haversian spaces. It affects the cranium (not the face), spine, limb, bones, and clavicle. It becomes manifest to the patient by the fact that he frequently has to change the size of his hat. Sometimes rheumatic pains in the bones are complained of. The head is projected forwards, associated with kyphosis in the dorso-cervical region, so that the attitude is characteristic. The base of the chest is expanded, the abdomen diamond-shaped,

and crossed by a deep transverse sulcus, the hips are widened, and the legs are bowed outward and forward.

VIII. **Mollities Ossium** (Synonym : Osteomalacia) is a progressive disorder of the bony system, due to gradual decalcification and weakening of the skeleton, which results in considerable deformities and contortions, owing mainly to muscular action. It occurs in women (91 per cent., Arthur Durham) between twenty-five and thirty-five years of age, mostly after pregnancy. The early

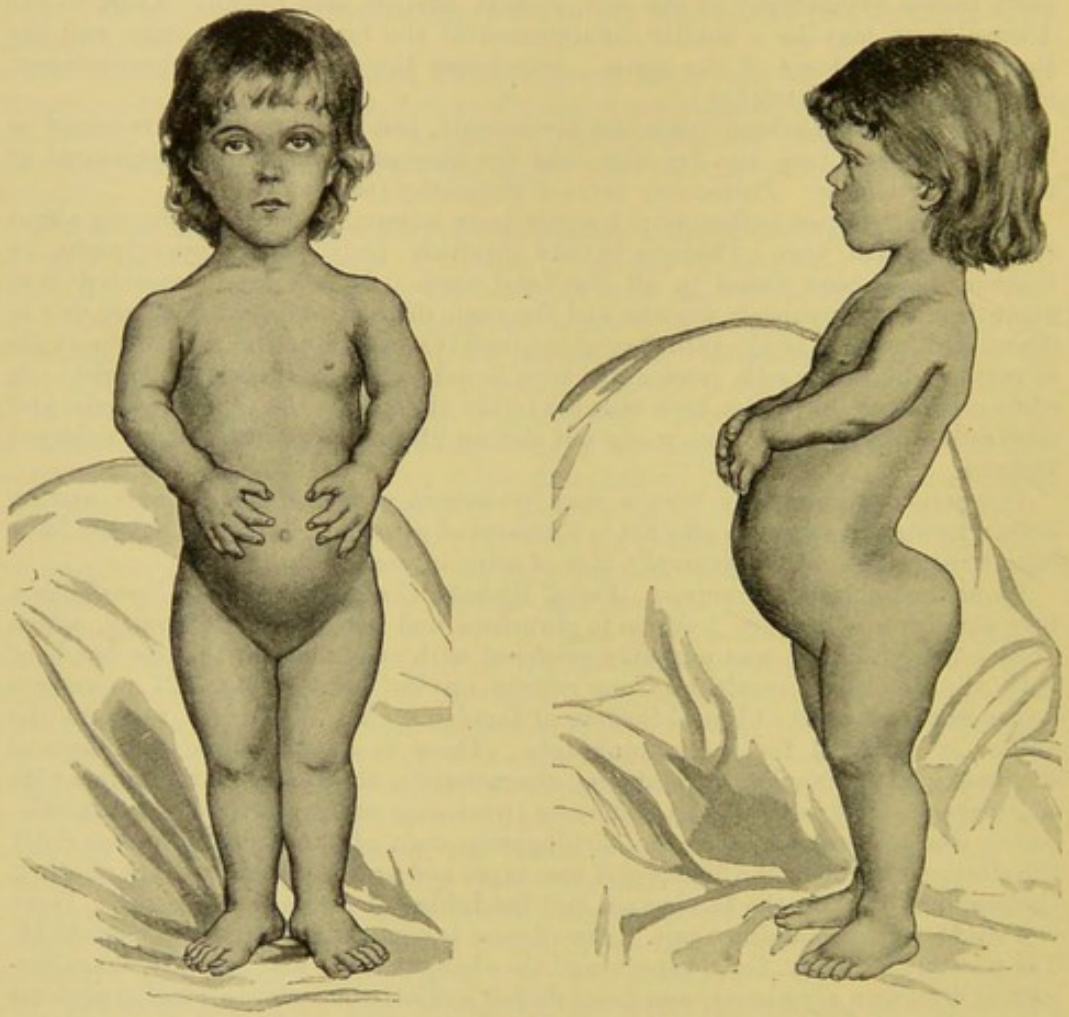


Fig. 127.—A case of ACHONDROPLASIA exhibited by Mr. W. Turner at the Clinical Society of London (Trans. Clin. Soc. Lond., vol. xxxii., 1899).

symptoms consist of wandering pains in the limbs and trunk, worse at night, with weakness of the limbs. In the course of a few months there is bending of the bones ; spontaneous fractures and distortions may occur. The stature is diminished from the involvement of the spine. Death usually occurs from respiratory complications owing to fracture of the ribs.

IX. **Leontiasis Ossea** is the term given to a rare condition in which there are symmetrical hyperostoses of the facial bones and skull, which encroach upon the cranial cavity, and so may lead to death.

CHAPTER XVIII

THE SKIN

THE skin is subject to the same diseases as other epithelial structures, but the circumstance which strikes the thinking student is that, although various skin diseases have, as it were, written their own characters on the surface of the body in full view of the observer, we have hitherto learned really less about the pathology of morbid processes which occur in the integument than we have about those which take place in most of the other parts of the body.

Another circumstance which increases the mystery surrounding skin disease is the immense number of names adopted by dermatologists for the same or but slightly different diseases. It has not been possible to give all the synonyms for the various recognised types of disease in the following pages, but a glance at the disease now usually known as dermatitis herpetiformis will give some idea of the mysteries and difficulties of this nomenclatural maze. It would almost seem as though every dermatologist was compelled to rename some old disease or to rediscover some " new " disease in order to justify his pretensions to the title.

I am happy to say that this tendency to cut up a disease into an infinitude of varieties and subvarieties seems at last to be on the wane, and that a more scientific tendency to trace several differently named varieties to one morbid process has set in.

PART A. SYMPTOMATOLOGY.

The cardinal symptom of skin affections consists of an ERUPTION with or without SUBJECTIVE SYMPTOMS. The subjective symptoms of skin diseases are of relatively less importance for diagnostic purposes, because the morbid process itself is before us. There is, however, one subjective symptom which attends a great many skin diseases — namely, PRURITUS (itching). Anæsthesia and other disorders of sensibility will be dealt with among diseases of the nervous system.

§ **450. Pruritus** is the Latin word for itching, and that is the sense in which it is used here.

There are three groups of *Causes* of itching :

(*a*) Pruritus may be *secondary* to some visible skin disease, and in

that case the itching is localised to the neighbourhood of the eruption. Some eruptions are invariably attended by itching, such as urticaria, eczema and most acute conditions which progress rapidly. Other diseases are generally unattended by itching, such as syphilis, psoriasis, and most chronic conditions which evolve their course slowly.

(*b*) Various *local conditions* may produce more or less localised itching: (1) *Discharges* or secretions from nasal, buccal, or anal orifices, *e.g.*, pruritus ani, pruritus scroti, pruritus pudendi. In many of these cases there is also a certain amount of localised eczema, which is possibly also the result of the discharge or sweat. (2) A *rough garment*, such as a new flannel shirt or certain dyed articles, may produce intolerable itching in delicate skins. (3) Various *parasites* give rise to pruritus. With *scabies* there is also a characteristic eruption localised chiefly to the flexures of the joints. In *phtheiriasis* (due to pediculi corporis) the eruption is generalised, though most intense across the shoulders; the *flea*, the *harvest-bug*, *pediculus pubis*, and other parasites cause intense itching.

(*c*) With *idiopathic* or internal causes the itching is generalised, and may or may not be accompanied by a certain amount of generalised eruption of papules (see Prurigo, § 464). Among the causes may be mentioned gout, certain articles of food (*e.g.*, shell-fish, eggs, cheese, excess or deficiency of salt), jaundice, digestive disorders, lithæmia, diabetes, kidney disease, pregnancy, nervous irritability, constipation, and old age. The pathological condition on which all of these depend is in the author's view[1] some blood change. The *Treatment* of pruritus is given under Prurigo.

PART B. PHYSICAL EXAMINATION.

The APPARATUS required for the investigation of skin diseases is simple, and consists of a good lens some 3 inches in diameter, a microscope with accessories, and the means of histological examination. A pair of flat forceps is useful for removing scales, hairs, or parasites. A flat glass slide may be used or the skin may be stretched to ascertain if the spots disappear on pressure.

HISTOLOGICAL EXAMINATION is of great use in many cases, and frequently enables one to diagnose a lesion with certainty; a small piece of the diseased skin can be removed without causing appreciable pain if the part be first frozen by ethyl chloride. As the skin freezes pinch up the fold required with the fingers so that it retains its shape, then take hold of it with a forceps, and cut with curved scissors a tiny piece (including all the layers of the skin), which can be put into alcohol, then celloidin or paraffin for section cutting.

§ **451.** The points to investigate in any given case of skin eruption are: I. The size and appearance of the prevailing elements; II. What it feels like, and whether it disappears under pressure; III. The distribution and symmetry of the eruption; IV. Subjective symptoms; V. The duration and evolution of the eruption; and VI., Its etiology.

[1] The *Lancet*, August 1, 1896, p. 300.

I. The **Character and Size of the Prevailing Elements.**—The spots are *never all quite alike*, being modified by the age of each spot, the locality affected, and the conditions to which it has been subjected (*e.g.*, scratching or pressure). It is therefore of the highest importance to *examine every part* of the eruption. Patients may object to undress and the physician may grudge the time, but these considerations should never be allowed to weigh. The most convenient clinical classification of skin diseases is based upon the nature of the elementary lesions, a list of which is given below.

§ **452.** The principal **elementary lesions** which appear on the skin are as follows : There are three varieties of primary lesion, and three which arise secondarily to these.

1. A *macule* (or macula) is a spot of congestion ; *roseola* is a generalised eruption of macules ; *erythema* is a larger area of congestion with fading edges. A *wheal* is a spot of congestion accompanied by slight exudation beneath the skin ; it is also called *urtica*, and a generalised eruption of wheals is called *urticaria* or " nettle-rash," because they resemble nettle stings.

2. A *papule* (or pimple) is a small solid elevation of skin, conical, round-topped, or flat. A *lenticular* papule is a large flat-topped papule. A tubercle, or *nodule*, as it may be better called, to avoid confusion with the lesion of tuberculosis, is larger than a papule, but not large enough to be called a tumour.

3. A *vesicle* is a collection of serous fluid beneath the cuticle. A *bulla* is a large vesicle. A *pustule* is a collection of purulent fluid beneath the cuticle.

The SECONDARY lesions are :

1. A *scale* or squame is the exfoliation of cuticle which occurs after a congestion or inflammation of the skin, or it may be the product of pathological processes special to the skin, such as cornification, or hyperkeratosis. In a sense a scale may be a primary lesion.

2. A *crust* or scab is dried serum or pus.

3. *Fissures*, ulcers, cracks, excoriations are breaches of the surface. *Cicatrices* or scars may result from these when a sufficient extent or depth of skin is involved.

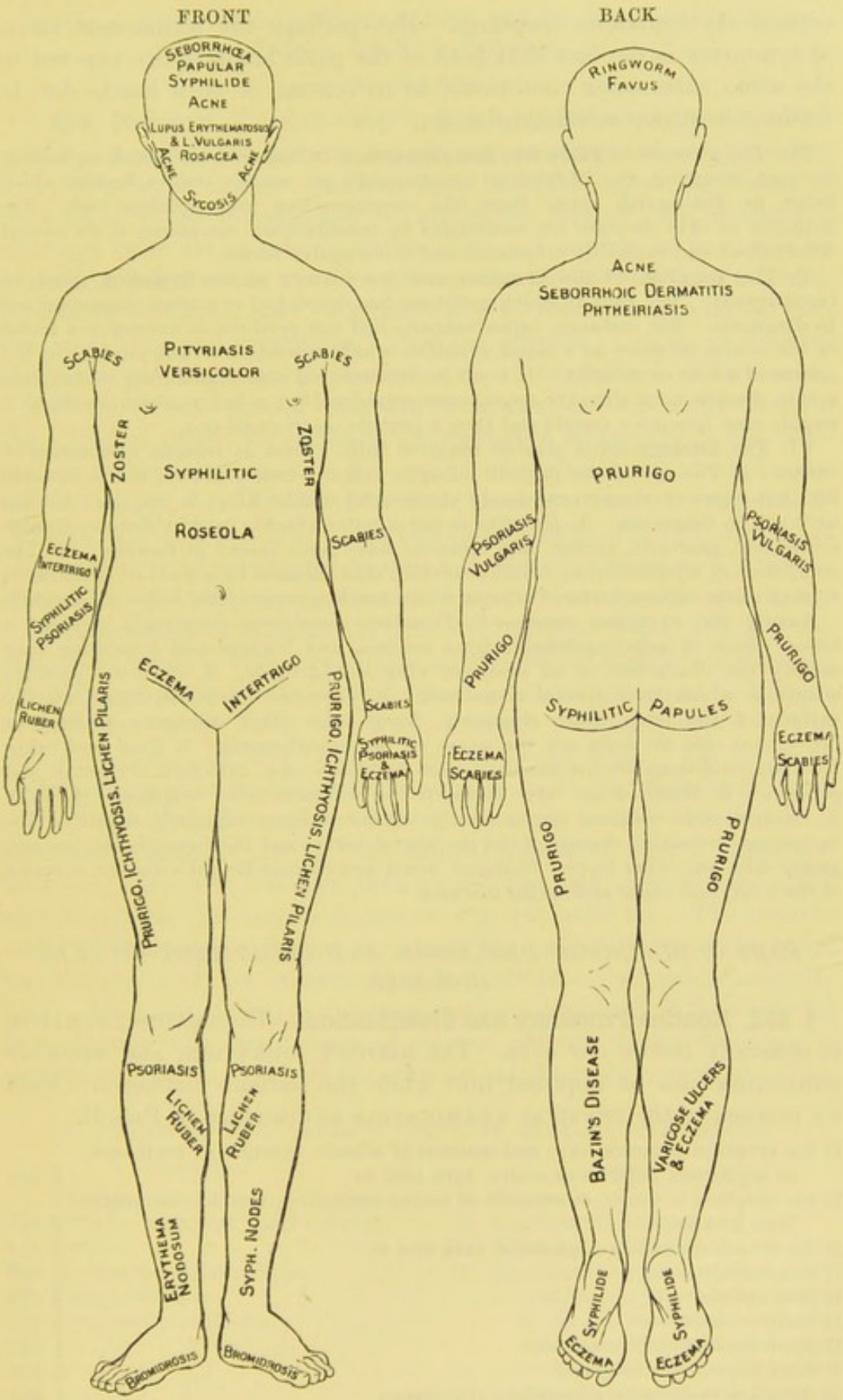
PIGMENTARY ALTERATIONS are known as *chloasma* when there is a broad streak of excessive pigment ; *leucoderma*, when there is an area of skin devoid of normal pigment ; *melanoderma*, when there is an area of increased pigmentation. *Ephelis* is a freckle. *Nævus* is a mole or birthmark, either pigmented, hairy, or vascular. A dilatation of the superficial vessels of the skin is known as *telangiectasis*. *Petechiæ* are small spots of hæmorrhage into the skin. *Ecchymoses* are larger patches of extravasated blood which go through the changes of colour characteristic of a bruise. A *comedo* or " blackhead " is a little black plug of inspissated sebum blocking the orifice of a sebaceous gland.

The fundamental **histological changes** of the skin are congestion (hyperæmia) with or without exudation, inflammation, and infiltration. If the lesion consists of *congestion*, such as roseola, or urticaria, or simple *inflammation* without infiltration, such as eczema, it disappears on pressure. If, on the other hand, there be definite *infiltration* or neoplastic deposit, as in lupus and syphilis, or if there be *hæmorrhage* into the skin, the colour does not disappear when the skin is pressed by the finger or a glass slide, or stretched. This is a point of much significance in the diagnosis of skin diseases. The secondary consequences of inflammation in the skin are, as elsewhere, three in number. If the inflammation does not undergo resolution, there may be (1) *suppuration*, leading to the formation of pustules, ulcers, etc. ; (2) *necrosis*, as in the centre of boils and carbuncles ; or (3) *organisation*, as in the case of the various scars, hypertrophies, or scleroderma. In addition to the primary lesions just referred to—congestion, inflammation, and infiltration and their consequences—which occur in the skin as elsewhere, there are at least four processes special to the skin. 1. *Hyperkeratosis* is an increased deposit of keratohyaline material leading to an increased cornification of the surface cells of the epidermis, and a scaliness of the surface as in pityriasis and psoriasis. 2. *Parakeratosis* is the irregular or deficient cornification which occurs, for instance, in eczema. Here the prickle cells, instead of going through the regular process of cornification by the deposit of kerato-hyaline granules in their interior, and their gradual conversion into dry, horny, non-nuclear cells, remain moist and succulent (though dry on their exterior), and retain their nuclei. They adhere to one another, being moister, and are shed in masses of crusts and scales instead of being shed singly and imperceptibly. 3. *Acanthosis* is a term applied to the increased proliferation of the prickle cells by increased mitosis (karyokinesis), resulting in an increased thickness in the epithelial layers of the cuticle. 2 and 3 are found in all kinds of eczema, 2 chiefly in dry eczema ; 3 is met with in moist eczema.

II. What does the **eruption feel like,** and does it **disappear on pressure** ? Infiltrating lesions feel hard, and do not entirely disappear on pressure, as is evident from the histological characters (*vide supra*). A faint purpuric eruption may thus be diagnosed from an erythema.

III. The **distribution, position** and **symmetry of the eruption** is important for purposes of diagnosis, and it is therefore most essential to examine the whole of the eruption. Many diseases may be recognised by the position in which the elements predominate, and Figs. 128 and 129 will aid the student to remember the parts most frequently affected by certain eruptions. Some diseases are always more or less generalised—*e.g.*, urticaria and the exanthemata, and this generalised distribution usually indicates a toxæmic or idiopathic cause. Others, while sometimes affecting the whole body, have a preference for certain parts—*e.g.*, psoriasis for the knees and elbows, seborrhœic dermatitis for the head and shoulders. Various words are used to describe the distribution, thus, *punctate* when the eruption is dotted about, *discrete* when the elements are separate, *confluent* when they run together, *gyrate* or crescentic when they are arranged in wavy lines or segments of circles, *circinate* or annular when they are in circles, *corymbose* when grouped into clusters.

Any **symmetry** of arrangement on the two sides of the body should be carefully observed, though its significance must not be overrated. It may indicate that some constitutional or blood change is in operation as in the earlier eruptions of syphilis. Symmetry may also indicate that some nervous or neuro-vascular cause is in operation, as in



Figs. 128 and 129.—DIAGRAM showing the parts most frequently affected by certain eruptions.

certain erythematous eruptions. But perhaps the commonest cause of symmetry is the fact that both of the parts involved are exposed to the same extraneous conditions, as in eczema of both hands due to washing in strong solutions of soda.

IV. The presence of **Subjective Symptoms** must be inquired into, such as itching, burning, smarting, etc. Syphilitic eruptions do not usually itch, a feature which helps to distinguish them from the corresponding non-syphilitic rash. The majority of skin diseases are *unattended by constitutional symptoms*, if we except tuberculous and syphilitic eruptions, and the eruptive fevers.

V. The **Duration of the Eruption** and the history of its **Evolution** must be investigated. The rate at which a disease has developed is a most important aid to diagnosis. For instance, lupus vulgaris will not produce so extensive a lesion in the course of years as a facial syphilide which resembles it will produce in the course of weeks or months. It must be remembered also that during its progress a skin disease may alter its appearance considerably; a lesion which starts as a papule may become a vesicle and then a pustule, as in small-pox.

VI. The **Etiology** must also be inquired into. First as regards PREDISPOSING causes: 1. The *age* of the patient. Lupus vulgaris nearly always starts in early life, but lupus erythematosus rarely starts until middle life. 2. *Sex* does not aid us much in diagnosis. 3. *Heredity* is not a potent factor in skin disease, though ichthyosis, psoriasis, cancer, and albinism have been traced in families. 4. The *occupation* of a patient may result in certain skin diseases by a want of cleanliness, or may cause various forms of eczema of the hands (grocer's itch, baker's itch, etc.).

Among the EXCITING causes—1. *Traumatic conditions* frequently produce a lesion which is indistinguishable from eczema, and friction and scratching may modify the characters of an eruption very considerably. 2. *Parasites* produce eruptions which have special characters. 3. *Vegetable organisms*, fungi, on the surface of the body cause ringworm, favus, and other diseases. 4. *Bacteria* introduced *into the body* are the cause of the exanthemata. 5. *Gout* and other toxæmic conditions are the causes of some eruptions—*e.g.*, urticaria, erythema, and prurigo. 6. Many *drugs* are attended by characteristic eruptions (§ 457). 7. *Diseases of the internal organs* may produce eruptions, especially digestive disturbances (urticaria), disease of the peripheral nerves and their ganglia (herpes and glossy skin, and other trophic changes), acute and chronic Bright's disease, diseases of the liver, and other abdominal diseases.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF SKIN DISEASES.

§ **453. Routine Procedure and Classification.**—The LEADING SYMPTOM is generally before our eyes. The HISTORY, DURATION, and MODE OF EVOLUTION can be inquired into while the patient undresses. Then we proceed to the PHYSICAL EXAMINATION as described in Part B.

If the eruption is QUITE DRY, and consists of wheals, macules or erythema, or erythema, papules or scales, turn first to .. .. .. § 454
If the eruption is MOIST, or consists of serous exudation, vesicles, or crusts, turn first to .. .. § 478
If the eruption consists of *pustules*, turn first to .. .. .. § 484
If it is *multiform* .. .. § 489
If it is *nodular* .. .. § 490
If there is *ulceration* .. .. § 492
If there are *warts* or *excrescences* .. .. § 493
If there are *scars* or *atrophies* .. .. § 494
If there are *vascular* or *pigmentary* alterations .. .. .. § 495
If there is disorder of the *sweat* .. .. § 497
If the *hair* or scalp is affected .. .. § 498

GROUP I. ERUPTIONS USUALLY DRY (Wheals, Macules or Erythema, Papules, and Scaly Eruptions).

a. Wheals.

§ **454. Urticaria** (" nettle-rash ") is a generalised eruption which consists of wheals, of more or less evanescent character, rarely lasting more than a few hours. Patients come complaining of the *history* of such an eruption accompanied by intolerable itching ; and sometimes although there are no wheals visible, these can readily be produced by drawing a point across the skin (dermatographia or urticaria factitia). This latter condition may very frequently be found in association with the exanthems and erythemas. It is also present in states of cerebral congestion (meningitis) and constitutes the tâche cérébrale of Trousseau.

Etiology.—Some people, especially neurotics, develop urticaria upon the slightest cause, for instance, on meeting a stranger, but the commonest determining cause is the eating of shell-fish, or unwholesome tinned food, or some intestinal toxin or gastro-intestinal derangement.

Varieties.—1. There is an acute and chronic form of the affection ; the first named consists of a transient attack lasting a few hours or days ; in the chronic or more properly the *recurrent* form (urticaria perstans) there are constantly recurring attacks. (2) Urticaria papulosa (§ 464), and (3) Urticaria pigmentosa (§ 495). (4) In giant urticaria or Quincke's disease the eruption is more persistent and consists of circular white œdematous spots or swellings in the skin. (5) Angio-neurotic œdema consists of still larger swellings affecting the loose subcutaneous tissue. (6) Urticaria bullosa is a rare variety with vesicles or bullæ, met with chiefly in children.

Prognosis and Treatment.—The disease as usually met with is essentially transient, and a brisk saline purge is all that is necessary. Relapsing cases are, however, difficult to cope with, and in these strict attention to the diet and the digestive organs is called for. The remarks under Erythema Multiforme concerning the coagulability of the blood and the treatment by calcium chloride apply in my experience equally here. After any digestive defects have been corrected, it is wonderful how efficacious 20 grain doses of calcium chloride thrice daily are in some cases, though in others the eruption reappears on ceasing the drug.

b. Eruptions which usually consist of Macules or Erythema.

Generalised.	*Localised.*
I. Exanthemata.	I. Rosacea.
II. Roseola (simplex and syphilitica).	II. Lupus erythematosus.
III. Erythema scarlatinoides.	III. Erythema nodosum.
IV. Drug eruptions.	IV. Erythematous eczema, X-ray dermatitis, Erythema paratrimma, E. faciei, E. traumaticum, E. caloricum, E. Pernio, and other varieties of E. multiforme ; Macular Leprosy, and Pellagra.
V. Erythema multiforme.	

The early stages of eczema and of other eruptions to be mentioned hereafter may take the form of an erythema.

I. The **Exanthemata** or eruptive fevers are fully described in Chapter XV., where they form Group I. of the acute specific fevers.

§ **455.** II. **Roseola** is a term employed to designate a generalised eruption consisting of patches of congestion, more or less marginated, varying in size from a pin's head to a lentil. Two varieties are described.

Roseola Simplex may resemble measles, and, indeed, its chief importance is in connection with the diagnosis from this disease (*q.v.*): it gives rise to a considerable amount of itching and irritation, with usually a slight degree of constitutional and gastro-intestinal disturbance. It may occur in childhood under the same conditions as urticaria, and is therefore predisposed to by gastric disturbance. The occurrence of such an eruption when small-pox is prevalent should make one suspect the initial eruptions of that disease. It is one of the commonest rashes associated with vaccination. Drugs, such as copaiba, may cause it. *Treatment* consists in administering an aperient, salines, and diuretics, and in correcting any concurrent digestive disorder.

Roseola Syphilitica is the earliest of the syphilitic skin eruptions, occurring three to six weeks after infection. It appears upon the trunk, chiefly its anterior aspect, the chest, the flexures of the limbs and the palms and soles, as rosy or *dusky* red macules, disappearing on pressure, rounded, oval or irregular in shape with fading edges, varying in size from a pea to a shilling. A degree of pigmentation may be left behind. Sometimes the eruption is so faint that it is overlooked. It becomes better marked after a bath or when the skin is exposed to cold. It may last from a few days to a few weeks. It is *diagnosed* by the history and other signs of syphilis. *Non-syphilitic roseola* undergoes rapid changes in size and shape; *pityriasis versicolor* can be scraped off and is fawn-coloured; *seborrhœic eczema* develops greasy scales on the surface, and forms spots of irregular size and outline.

§ **456.** III. **Erythema Scarlatinoides,** as its name implies, consists of a widespread rash, resembling scarlet fever, preceded and accompanied by fever and constitutional disturbance, and followed by desquamation. So-called "surgical scarlatina" is probably identical with this condition. The chief causes are septicæmia, intestinal disorders, enemata of soap or other substances, the ingestion of ptomaines and other toxins, certain drugs (see below), rheumatism, gonorrhœa, and sewer-gas. The *Diagnosis* from scarlet fever is difficult only in severe cases. In erythema there is less constitutional disturbance, no strawberry tongue, and there is a tendency to relapse.

§ **457.** IV. **Drug Eruptions.**—An idiosyncrasy with regard to certain drugs, whether taken by mouth or applied externally, is shown by some individuals, and is manifested by the appearance of a rash, which disappears on the withdrawal of the drug. Two things will be noticed in the list below: (1) That by far the commonest eruptions are erythema and its congeners; and (2) that the iodides and bromides are among the commonest drugs to produce eruptions; they may even produce a frambœsial eruption resembling gumma. The chief eruptions produced by the internal administration of drugs are as follows:

Papulo-Pustules: Bromide and iodide of potassium (chiefly on the face), occasionally sulphide of calcium, antimony, and arsenic.

Erythema : Antipyrin, antitoxins, copaiba, cubebs, turpentine, belladonna, atropine, quinine, salicylic acid and sodium salicylate, boracic acid; iodoform and carbolic acid by absorption from wound dressings. And in the author's opinion certain toxins absorbed with milk.

Urticaria : Quinine, copaiba, turpentine, valerian, santonin, sodium salicylate, benzoic, salicylic and tannic acids.

Erysipelatoid (erythema with infiltration or œdema of the skin) : Bromide and iodide of potassium, quinine, iodoform, boracic and carbolic acids (aconite, oil of cade, chrysarobin, and carbolic acid applied externally).

Herpes : Arsenic.

Purpura : Iodide of potassium, chlorate of potash, chloral hydrate, chloroform, copaiba.

Pigmentation : Silver nitrate, arsenic.

Epidermic Thickening : Arsenic and borax.

§ **458.** V. **Erythema Multiforme** is an affection characterised by erythematous efflorescences, varying in form and size, localised usually upon the backs of the

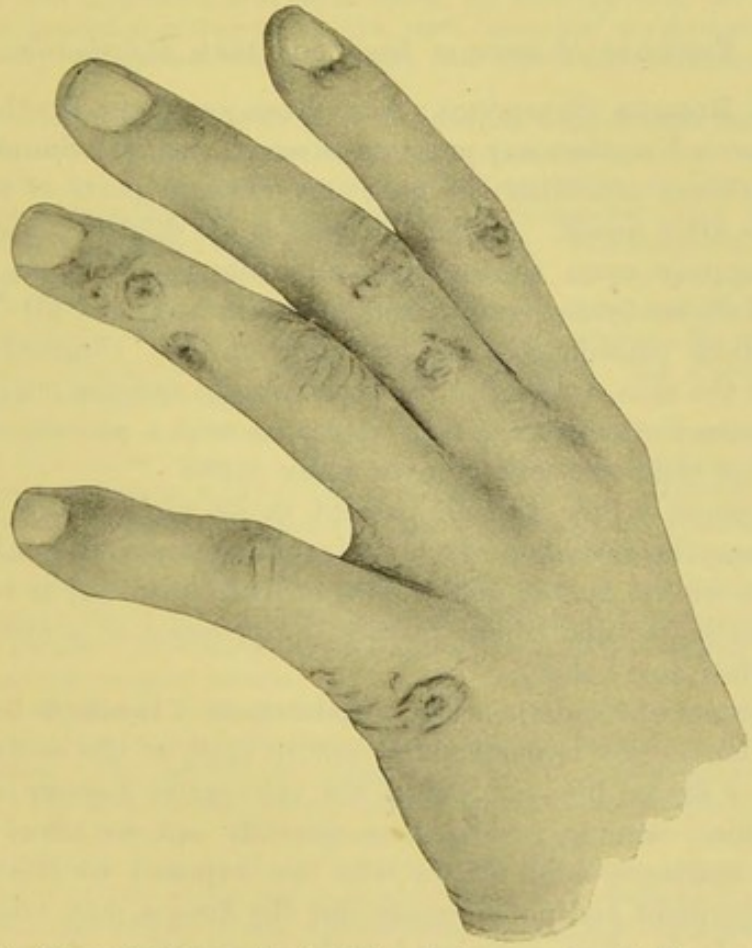


Fig. 130.—Erythema Iris on the hand of a single woman twenty-three years of age.

hands and forearms, dorsum of feet and legs, and sometimes on the face, neck, and the trunk, and accompanied usually by lassitude and ill-health. The lesions vary in size from a lentil to the palm of the hand. They belong to the *congestive* form of eruption, being slightly raised, with fading edges. The centre is the highest part, is usually livid, and sometimes hæmorrhagic. There is usually a feeling of burning or formication. Many varieties have been described, such as *E. gyrata*, when the blotches fade in the centre, coalesce with neighbouring ones, and form wavy or gyrate lines. *E. iris* (herpes iris, herpes circinatus) is a form in which a

vesicle forms in the centre, with concentric rings of purple and white and red around it (Fig. 130). In *E. bullosum* bullæ form. *E. nodosum* is described in § 461. Erythema is known from urticaria by its deep red coloration, by its more localised distribution, the larger size and more permanent character of the lesions, less itching, and more marked constitutional symptoms. Young people and males are more prone to the affection. It is commoner in the spring and autumn. The *course* of the disease varies, but each erythematous patch lasts eight to ten days, and they continue to appear for two to six weeks. Each may leave temporary brown pigmentation, and desquamation may occur as they fade.

Treatment.—That the disease is due to some alteration of the blood is shown by the marked effect which certain remedies have upon the condition, especially quinine, and, as Sir A. E. Wright and the author have shown, calcium chloride.[1] This alteration appears to consist of a diminished coagulability of the blood, such as occurs when calcium salts are deficient. Quinine should be given in 4 or 5 grain doses, t.i.d. and gradually diminished, or gr. xx. calcium chloride. If given after meals these doses do not impair the digestion. The diet and digestive system require careful regulation.

Erythema of more or less LOCALISED *distribution.*

§ **459.** I. **Rosacea** (Synonyms: Acne Rosacea; Acne Erythematosa; Gutta Rosacea Kupeferrosa) presents three stages: (1) Simple congestion or erythema attacking the nose and adjacent parts of the cheek, often worse after meals. (2) In the next stage dilated vessels (telangiectasis) appear upon the surface, and after some time, inflamed sebaceous follicles (acne papules and pustules) appear. (3) The third stage involves considerable hypertrophy of the connective tissue elements of the skin, leading to the formation of nodules (rhinophyma) which are sometimes of great size. Rosacea runs a prolonged course; the first stage alone may extend over many years.

The *Diagnosis* is not difficult, except in its early stage, when the erythema may be mistaken for lupus erythematosus and other kinds of erythema of the face (§ 462). The former, however, is recognised by slight scaliness; and a lens reveals the presence of a fine "tissue-paper" surface (see below).

The *Causation* of the disease is not understood. It affects both sexes, but in the female sex is particularly apt to start at the evolution and involution of sexual life—*i.e.*, when the vaso-motor system is particularly irritable. Among males, it is specially apt to affect cabmen, coachmen, mariners, and others who are exposed to the weather. Drink is a frequent and potent cause, but the disease may arise in total abstainers. It is often associated with constipation, dyspepsia, and pyorrhœa.

Much of the *Treatment* of acne vulgaris (§ 463) is available for rosacea. The cause if possible, should be removed. In the first stage stomachics, alkaline carbonates and other internal remedies are indicated. Several cases under my care have benefited by large doses of calcium chloride. Calamine lotions and soothing remedies should be applied. In the

[1] *Lancet*, August 1, 1896.

later stages, diligent application of phenol at intervals, and for rhinophyma scarification, will undoubtedly ameliorate even very bad cases.

§ **460.** II. **Lupus Erythematosus** (Synonyms: Ul-erythema Centrifugum, Seborrhœa Congestiva of Hebra) is the most chronic of the erythemata. The eruption has a spreading erythematous border, which as it spreads leaves a very thin permanent scar in the centre. In the first stage the disease begins with one or more small, red, slightly-raised spots. By spreading at the margin and increasing in number the little patches form, in the course of many months, an irregular bluish red area, with thin cicatricial centre and erythematous margin covered with scales, and sometimes with crusts. In another variety (seborrhœa congestiva of Hebra) there is a marginated erythema with numerous black specks, or large gaping openings of the sebaceous glands; the central part of the skin appearing depressed, and covered with adherent dry scales, interspersed with venules. The favourite seats of the eruption are the checks and bridge of the nose (butterfly distribution); then other parts of the face and forehead, the lips, ears, scalp (where the scar leaves permanent bald patches), the extensor surfaces of the hands, fingers, and toes (l. pernio), and more rarely on other parts of the body. The patches are generally symmetrical. In rare cases the erythematous patches become rapidly widespread over the body, and severe constitutional symptoms are present.

Etiology.—The disease is much more frequent in women than men, and very rarely occurs under twenty, a most important circumstance in the diagnosis from lupus vulgaris, which invariably appears before or during adolescence. Lupus erythematosus, according to some observers, is connected with the tubercle bacillus, but this has never been found in the lesions. The *Diagnosis* from lupus vulgaris is given in tabular form (§ 490). *Prognosis.*—L. erythematosus extends over ten or twenty years; always terminates in cicatricial changes in the skin, and permanent baldness of a hairy part. Beyond the disfigurement the disease is not serious, and is unattended by constitutional symptoms. The acute generalised variety usually terminates fatally.

Treatment.—In the early stage we must employ soothing remedies which act superficially (*vide* acute eczema). If these fail, stimulating ointments and caustics may be necessary, such as salicylic and carbolic ointment, or salicylic collodion, or plaster mull. Painting with carbolic or carbol-camphor, or sulphur pastes, etc., give rise to a reactionary inflammation and swelling which runs its course in a few days, then results in considerable improvement. Linear scarification, X rays, high frequency, zinc and copper cataphoresis, and Finsen treatment have also given satisfactory results. Internally, quinine, salicylic acid, and intestinal disinfectant treatment have given good results.

§ **461.** III. **Erythema Nodosum** is an eruption with an acute onset, consisting of erythematous lumps about the size of a pigeon's egg, occurring most frequently over both shins. The patches are round, oval, raised, non-marginated, painful, and tender. The centre is most deeply coloured, whence the purplish tint gradually fades away to the margins. There is usually some malaise and elevation of temperature; sometimes pain in the joints and other rheumatic symptoms. Each nodule lasts one to two weeks, and successive crops may continue for a month or two. Patients are usually young women with a rheumatic diathesis. The condition is known from other forms of erythema by the position of the lesion and the acute pain and tenderness. In periostitis the lesion is usually single. The disease usually runs a benign course to spontaneous recovery in a month or two. The *Treatment* consists in the administration of salicylates, saline aperients, and after the acute symptoms have subsided, iron and quinine internally. Lead and opium lotion applied locally allays the pain.

§ **462.** IV. Certain specially-named forms of erythema may be mentioned.

Erythematous Eczema.—Eczema is usually vesicular, but there is an erythematous variety which may run its course without presenting any vesicles. The

surface of the skin is red, dry, and rough, with slight scaling. It frequently attacks the face, when the eyes may be almost closed, and is attended by burning and itching. For treatment, see Eczema, § 478.

X-ray Dermatitis may be acute, consequent on a single large dose, or chronic, after repeated small doses of X rays. In the acute form there is erythema, swelling, sometimes bullæ, and sensations of burning or intense pain, according to the degree of the mischief. In mild chronic cases there is temporary loss of hair and pigmentation. If exposures are continued, telangiectasis develops; atrophy, cracking of the skin, warts, and indolent ulcers follow. Treatment is prevention by ensuring greater protection to those engaged in X-ray work by wearing lead foil or other material impervious to the rays. Sedatives, lotions, and pastes hasten the recovery of acute dermatitis. Antiseptics should not be employed. For chronic forms it is necessary to order complete rest from exposure to the rays.[1]

Bedsores (E. paratrimma) are due to pressure over prominent parts, such as the sacrum, trochanters, heels, or ankles of the bedridden, or to the pressure of a badly-adjusted splint. A local patch of erythema appears, followed by abrasion of the skin. If the cause continues sloughs form. They are due to three causes: pressure or irritation from rubbing, perspiration and excretions in cases of incontinence, the lowered vitality of the sick and aged. In certain nerve diseases, especially myelitis, the sloughs form so rapidly that the condition is attributed to a trophic neurosis. Extreme fœtor attends the decomposition of the slough, and septicæmia may set in.

Treatment.—Good nursing can prevent bedsores. Three principles should be kept in mind: cleanliness, dryness, and relief of pressure. (i.) The parts should be carefully cleansed night and morning, and the draw-sheet pulled through immediately it becomes soiled. (ii.) After washing, the skin should be thoroughly dried by rubbing over a little methylated spirit or brandy, and well powdered. (iii.) Relief of pressure is obtained by a water-bed, ring pads, and by frequently turning the patient from side to side. If an ulcer or slough forms these measures should be combined with the plentiful use of antiseptic lotions, and occasionally a charcoal poultice to relieve the pain and promote healing.

Erythema Faciei vel *e. fugax* is a flushing of the face which occurs chiefly in association with dyspepsia. It may form the first stage of rosacea. *E. traumaticum* develops on any part subject to long-continued pressure—*e.g.*, the garters and tight waist-bands. *E. læve* is the erythema found on the legs of dropsical persons. *E. caloricum* appears on the face from exposure to the sun and wind. *E. intertrigo* is found in parts which are opposed, such as the thighs and armpits, in infants, corpulent people, or those who perspire much. It may pass on to eczema. *E. pernio* (Synonyms: dermatitis congelationis, frostbite, chilblain) is a painful inflammatory condition of the skin of the fingers, toes, heels, or other portions of the feet or hands, caused by exposure to cold, and attended with itching and tenderness, sometimes by vesication, ulceration, or gangrene. In the form of the so-called chilblain it is prone to occur in those whose circulation is poor, and constitution feeble. Thus children and old people frequently suffer from this complaint during successive winters. *Treatment* consists in local applications of stimulating liniments, such as iodine and camphor, and internally, iron and strychnine.

Pellagra is a non-contagious endemic disease occurring in adults in parts of Italy, Spain, and the Tyrol, due to eating diseased maize, and characterised by erythema, vesicular, and bullous eruptions, pigmentation, atrophy, and progressive constitutional weakness, terminating fatally in five to fifteen years.

Macular Leprosy appears as brownish or mahogany red patches of erythema of various sizes (§ 491).

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[1] Hall Edwards, *Brit. Med. Journ.*, vol. ii., 1908, p. 726.

c. Eruptions which usually consist of Papular Elements.

Common.

I. Acne vulgaris and other forms of acne.
II. Prurigo.
III. Scabies.
IV. Papular syphilide.
V. Skin diseases, sometimes papular at *one stage :*
  (i.) Papular eczema.
  (ii.) Psoriasis and other scaly eruptions.
  (iii.) Exanthemata.
  (iv.) Pustular and vesicular diseases.
  (v.) Erythematous eruptions.
  (vi.) Nodular eruptions.

Rarer.

VI. Lichen planus.
VII. Keratosis pilaris and follicularis.
VIII. Milium.
IX. Lichen scrofulosorum.
X. Adenoma sebaceum.

§ **463.** I. **Acne Vulgaris** is an eruption consisting of " blackheads " or comedones and indurated papules which may go on to suppuration, confined to the face, shoulders, and back, and sometimes the chest. Acne papules are pale red to crimson, and hard (A. indurata), varying in size from a pin's head to a small pea. These are invariably accompanied by a number of black points (comedones), which are black plugs of sebum. The papules come out one after another, and are remarkably persistent ; some go on to pustulation, or the formation of small abscesses, leaving scars if untreated. The skin of the face in individuals affected with acne is usually greasy, coarse, and dusky-looking.

Causes.—The disease in a considerable proportion of cases starts soon after puberty, and fresh papules may continue to appear for some years, occasionally to middle life or old age. Inspissated sebum blocking the follicles is the histological cause of the papules, and the change which the hair and sebaceous follicles undergo at puberty appears to be one of the factors in the causation of disease. Sabouraud and other observers have found a bacillus in the papules, which they consider the causative factor. Indigestion undoubtedly aggravates the condition.

Varieties.—(1) *Acne punctata*, *A. indurata*, and *A. pustulosa* are stages in the diseased process, not varieties in the true sense of the word. (2) Associated with pityriasis of the scalp a crop of papules often appears on the face, almost identical with acne spots, excepting that they are smaller, softer, and not totally accompanied by comedones. Its favourite positions seem to be the chin, the furrows below the angles of the mouth, and sometimes between the scapulæ. (3) *Bromide* and *iodide acne* are indistinguishable from each other, and the individual spots resemble acne vulgaris very closely. Come-

dones, however, are absent, and there is a greater frequency of distribution over the chest and back, though the face is always first affected. (4) *Acne Rosacea* is mainly an erythema, at any rate in its earlier stages (§ 459).

5. *Acne varioliformis* is a severe variety of A. pustulosa. It occurs chiefly on the brow, and is apt to leave deep scars. 6. *A. cachecticorum* (Hebra), or A. scrofulosorum (Calcott Fox [1]), or A. necrogenica is another pustular variety affecting chiefly the back and the extensor surfaces of the limbs, and to a less degree the face, in debilitated or scrofulous children. It is now regarded as a tuberculide. It differs markedly from A. vulgaris in its distribution, and though it may last for many years, is generally amenable to tonic treatment and cod-liver oil. It is liable to be mistaken for a syphilide, but the latter is less indolent, and there is a firmer infiltration.

The *Diagnosis* of acne from the other eruptions in this group is not usually difficult on account of (1) its characteristic position, and (2) the presence of comedones. Papular, pustular, and tubercular syphilides affecting the face are usually copper-coloured, and grouped in a serpiginous manner. Lupus vulgaris generally occupies one side of the face and presents no comedones.

Treatment.—If there be much irritation soothing applications are best applied at first—*e.g.*, calamine lotion. Of all remedial agents sulphur is the most efficacious. A sulphur ointment (20 to 40 grains to the ounce) should be rubbed on night and morning, or a sulphur lotion, or resorcin in strengths varying according to the skin of the individual. For pustular acne mercury is indicated, as in all suppurating affections—*e.g.*, an ointment of 10 to 30 grains to the ounce. To prevent scarring an antiseptic lotion should be used, each pustule must be lanced, and its interior wiped out with a pointed stick or match dipped in pure carbolic or camphor phenique. Staphylococcal vaccine injections are of value when crops of pustules resist ordinary treatment, and acne bacillus vaccine is on trial. The scalp should always be examined for seborrhœa, and, if necessary, treated, for unless this be cured, the acne may recur. Indigestion and constipation aggravate acne, and must be corrected. Washing the face frequently with warm water and soap, rinsing off the soap, and rubbing with a rough towel is a valuable domestic remedy. X rays and high frequency currents have been found useful in some cases.

§ **464.** II. **Prurigo** is a disease in which the leading and sometimes the only symptom is generalised itching (pruritus), but it is frequently accompanied by an eruption of papules, urticarial patches, and scratch-marks. The papules of *idiopathic prurigo* are hard, shotty, acuminate, pale red, frequently better felt than seen (giving the sensation of a nutmeg-grater), come out in crops chiefly on the extensor surfaces of the thighs and arms, the trunk, especially the back and buttocks, and only occasionally the face. Each crop lasts a week or two, and is

[1] *Lancet*, 1895, vol. ii., p. 499.

sometimes accompanied by urticarial blotches; dermatographia can generally be elicited. The intense itching leads to scratch-marks. In the course of time prurigo is followed by a dry, rough, thickened, pigmented skin. The *Prognosis* is unfavourable, especially in the very poor, the disease recurring for years, and sometimes lasting for life.

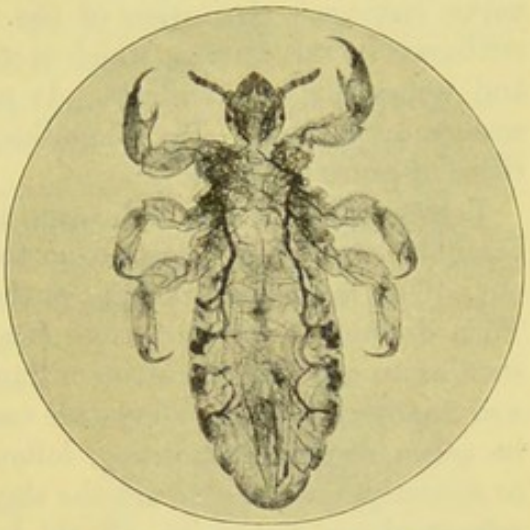

Fig. 131.—Pediculus Corporis, magnified about ten times.

The *Diagnosis* is simple in well-marked cases by the intensity of the itching and the condition of the skin. The eruption of prurigo very closely resembles that of scabies (§ 465) and pediculi corporis (Fig. 131), but in scabies it is almost confined to the *flexures of the joints* instead of the entensor surfaces; in pediculosis to the *shoulders*, where is is attended by typical staining. The pruriginous eruption of pediculi pubis (Fig. 132) is more or less localised. The diagnosis from papular eczema is not always easy; eczema, however, generally prefers the flexures and flexor surfaces, while prurigo predominates on if it is not confined to the extensor aspects.

Varieties.—(1) **Prurigo infantalis** (Synonyms: Urticaria Papulosa, Lichen Infantalis, Lichen Urticatus, Prurigo Mitis) should, in the author's opinion, be classified as a variety of prurigo. The papules are small, chiefly on the back, and the urticarial element moderate. It starts about the fourth month of life, and recurs until about the fourth year. (2) In **P. adolescentium** the papules are larger, the urticaria prominent, the skin brown and thickened. (3) In **P. senilis** (Synonym: Pruritus Senilis) the eruption may be insignificant or absent, and the irritation intractable, with a tendency to induration and purpuric complications. (4) In "*pruriginous*" *eczema*, an eczema complicates a prurigo; (5) In *P. agrius*, the prurigo of Hebra, all the lesions are on a larger scale, the inguinal glands involved, and the general health deteriorated, (6) *Summer prurigo* (Synonym: Lichen

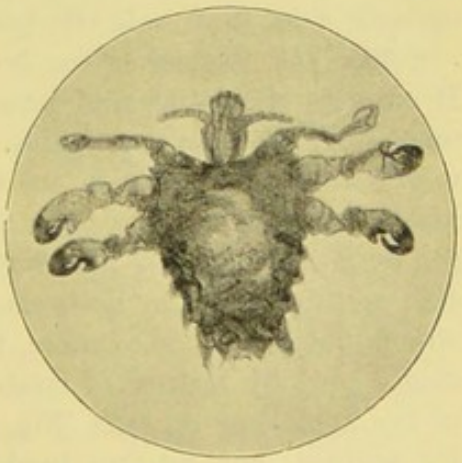

Fig. 132.—Pediculus Pubis, magnified about ten times.

æstivalis) develops in hot weather, and the papules tend to vesiculation and scarring (hydroa vacciniforme); and (7) *P. hiemalis* occurs in cold countries or in the winter only.

Etiology.—Prurigo is especially apt to occur at the two extremes of life, and is probably a blood disorder with irritability of the cutaneous nerve endings.[1] Irritation of the alimentary tract is liable to start outbursts of the disease, which is greatly influenced by improper food, and unhealthy surroundings. It may be idiopathic, or secondary to scabies or pediculi. The idiopathic causes of prurigo are the same as those of pruritus (§ 450c).

Treatment.—The first indication is to discover the cause, for without carefully sifting this question no treatment can be successfully carried out. One must first exclude phtheiriasis, scabies, or other parasites. Then determine whether there is any other source of local irritation, such as an unbleached cotton or flannel garment, or an acrid discharge; and finally turn to the idiopathic causes. Warm weak tar baths should be taken night and morning, followed by the inunction of a mild tar or mercury ointment, to get the skin into a healthier condition, and the softest of undergarments should be worn. For local itching, carbolic acid 1 in 40, alkaline lotions, vinegar, chloral-camphor (℥i. of each liquified and added to ℥i. starch powder) are all useful. The diet should be plain; sugar, alcohol, and excess of meat must be avoided. Any digestive errors should be corrected, and the possibility of mucous (catarrhal) colitis borne in mind. Calcium chloride in large doses has been very efficacious in my experience. Other remedies which are worth a trial are—cannabis indica, antipyrin, valerian, bromides (especially in nervous subjects), pilocarpin, liq. am. acet., atropine, carbolic acid, salol, salicylic acid, and other intestinal antiseptics.

§ **465.** III. **Scabies** is the eruption produced by the acarus scabiei. It consists of papules and vesicles of varying sizes; the latter may go on to the formation of pustules. In addition to its multiform character, it is readily diagnosed in its typical form (1) by the short white or black burrows; (2) by the excessive itching, and scratch-marks; (3) by the fact that it always commences and predominates where the skin is thinnest—*i.e.*, between the fingers or toes, the flexures of the wrists and elbows, the axillæ, the penis, the inner side of the feet and thighs; (4) by history of contagion; and (5) lastly, by the discovery of the animal or its eggs (Fig. 133). Prurigo is likely to be mistaken for scabies, but prurigo predominates on the extensor surface and outer side of the limbs. Eczema is perhaps the disease with which, in the adult, it is most frequently confused. Many experienced physicians have overlooked the parasitic origin of eczema thus produced, and it is a good rule to suspect scabies in inveterate cases of eczema.

[1] "The Pathology of Itching, and its Treatment by Large Doses of Calcium Chloride," the *Lancet*, August 1, 1896, p. 300.

Scabies untreated may go on for an indefinite time, but it readily yields to treatment. Sulphur ointment should be thoroughly applied after a warm bath with plenty of soap, for at least four successive nights, and the underlinen and sheets should be boiled. Balsam of Peru, either pure or mixed with equal parts of vaseline, is a more expensive but equally efficacious remedy. If the sulphur produce a secondary eruption, mix it with equal parts of zinc ointment.

§ **466.** IV. **Papular Syphilide.**—Syphilitic eruptions are often multiform, but papules generally form the most prominent feature of all syphilitic rashes, especially in the secondary stage. The papule, indeed, forms the prototype of all syphilitic eruptions. These papules are firm, glistening, and project above the surface of the skin with a hard, infiltrated margin, and *vary in size* from a pin's head to a bean. They are of a brownish-red colour (like copper or raw ham) which does not entirely disappear on pressure. The wide variability in the size of the papules is a feature distinguishing this from other papular diseases. As they increase in size the centre often becomes depressed, or cupped. The distribution is more or less generalised, often symmetrical, but the favourite sites are the forehead, around the mouth, the *flexor aspects* of the arms, and the trunk. When near the corners of the mouth or the anus their surface may be moist, and the *secretion is highly infectious*, no matter how long after the contraction of the primary malady. Itching is rare. Other constitutional signs of syphilis may be present. The presence of shotty glands in the groin and neck and elsewhere is a valuable aid in the diagnosis of all syphilitic eruptions. They are present even when no other signs are present, and may last throughout the patient's life. Two *varieties* of papular syphilide are described according to the prevailing size of the elements, *papular syphilide* if the spots are small and numerous, *lenticular syphilide* if they are large and scanty. The former is met with more in the early, the latter in the later stages of the diseases. A rarer form is the *corymbose syphilide*, in which there are clusters of small red rounded papules scattered over the body and limbs, which leave corresponding groups of atrophic scars (see also §§ 404 and 489).

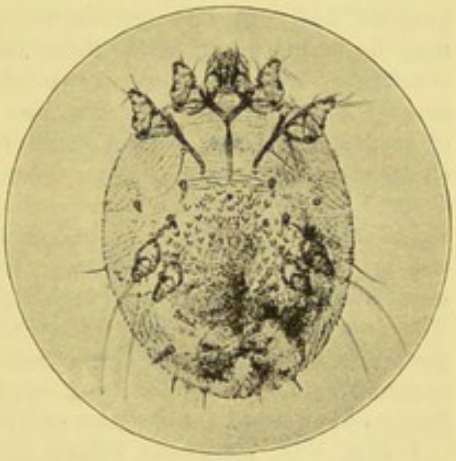

Fig. 133.—Acarus Scabiei (female), magnified about twenty times. The female burrows under the skin to lay her eggs, the burrows thus formed being quite typical and black with the fæces of the insect; most frequently situated on the wrist. The male roams about over the body and clothes, and is rarely captured.

V. **Skin Diseases Sometimes Papular.**—Eczema Papulosum (Papular Eczema, Pruriginous Eczema) is a term which should be confined to

papules which rapidly pass on to vesiculation, or which are associated with definite patches of eczema. Papules frequently form a stage, generally an early stage, in PSORIASIS, SEBORRHŒA, PITYRIASIS RUBRA PILARIS, SYCOSIS, in the EXANTHEMATA and ERYTHEMATA, and will be dealt with their proper place.

Rarer Papular Diseases.

§ **467.** VI. **Lichen Planus** (Synonym : Lichen Ruber Planus) is an eruption consisting of flattened, angular, shiny, dull-red papules, often presenting a central depression, and a small silvery scale on the surface. These tend to coalesce and form irregular patches of a peculiar purplish hue. Occasionally rings are formed (lichen annularis). There is no exudation. When the papules disappear much pigmentation may be left behind. The eruption is frequently symmetrical, and by far the most characteristic positions are the flexor aspect of the wrists and forearms, and the inner side of the knee. Sometimes the distribution is more generalised, and the mucous membrane of the mouth may be affected. Itching as a rule is present, and may be troublesome. By the fusion of several papules large plaques may be formed, and when these take on a warty growth, as about the ankles, the condition, which is very intractable, is called *lichen verrucosus*. Lichen occurs mostly about middle age, and has been noticed to appear when there is a degree of nervous debility. In Vienna two-thirds of the patients attacked are males (Kaposi), aged ten to forty ; whereas in England the majority of cases occur in women. There should be no difficulty in *diagnosing* lichen planus from a *papular syphilide* or an *eczema*, on account of its typical position, angular shape, purple colour, and flat waxy surface. The *Prognosis* as to the cure of the condition is good under treatment, although this may have to be extended over many months. The eruption remains localised to definite regions for years, but may become generalised. The *Treatment* consists in the administration of arsenic and tonics internally, and local soothing applications, such as F. 90 or 93 (see also § 499). Some recommend small doses of hydrarg. perchlor.

§ **468.** VII. **Keratosis Pilaris** (Synonyms : Pityriasis Pilaris, Lichen Pilaris or Spinosum) is an affection of the skin, generally of young adults, in which the orifices of the hair follicles of the thicker portions of the skin—*i.e.*, on the extensor and outer surfaces of the limbs—are occluded with corneous plugs. Hard friction, with almost any ointment at night, and a rough towel in the morning, will generally remedy the condition in a few weeks.

Keratosis Follicularis (Synonyms : Darier's Disease, Psorospermosis) is a very rare disease, due to overgrowth and degeneration of cells in the mouths of the pilo-sebaceous follicles. The papules are at first of pin-head size, resembling keratosis pilaris. They contain in the centre a horny plug, which is difficult to remove. Some become enlarged and hyperæmic ; others become confluent, presenting a papillomatous surface covered by hard yellowish crusts. These may ulcerate, and the area may be covered with a mucopurulent discharge. The disease affects first the face and head, and after the gradual development of years appears over the sternum, spine, loins, hypogastric and inguinal regions, and the extremities with symmetrical disposition. The *Diagnosis* may be difficult, in the early stages, from keratosis pilaris and ichthyosis, and in the later stages from acanthosis nigricans. *Treatment* consists in the use of salicylic, sulphur, or other keratolytic applications.

§ **469.** Three rare conditions—Milium, Lichen scrofulosorum and Adenoma sebaceum—come under the papular rashes.

VIII. **Milium.**—The term milium is applied to an eruption of small whitish or yellowish pearly granules about the size of a pin-point, which affect chiefly the delicate skin under the eyes, the eyelids, cheeks, temples, scrotum, and labia. It is due to retention within the sebaceous follicles, owing to an overgrowth of the

stratum corneum closing their openings. Some say they consist of embryonic tissue. The *Treatment* consists in making a small incision over the granule, and squeezing out the contents. Or electrolysis may be employed.

IX. **Lichen Scrofulosorum** is an eruption of minute yellowish-red papules, isolated or grouped, and occurring usually on the breast, abdomen, or back of tuberculous subjects. They are painless, and do not itch. It is not common after the age of twenty. It is not serious, and may last, hardly noticed by the patient, for months or years, and then subside without leaving a trace behind it. Its treatment is that of other tuberculous affections. It is probably the same disease as acne necrotica.

X. **Adenoma Sebaceum** is a rare disease, consisting of numerous small hemispherical elevations, discrete, grouped usually about the middle of the face. In size they vary from a pin-head to a split pea. Their surface is crimson or pinkish yellow, and associated frequently with telangiectases. They have no visible orifice. Some disappear spontaneously, leaving a small scar. The disease is almost always congenital, though it may not be observed till puberty, when it takes on fresh activity. It is said to be associated with intellectual inferiority, but this is not always the case. It is due to overgrowth of the sebaceous glands and hair follicles. The knife, electro-cautery, or electrolysis are necessary to destroy the growths where spontaneous involution does not occur.

d. Eruptions usually Scaly or Scurfy.

Common.	*Rarer.*
I. Psoriasis.	V. Exfoliative dermatitis.
II. Seborrhœic dermatitis.	VI. Pityriasis rosea.
III. Scaly syphilide.	VII. Pityriasis rubra pilaris.
IV. Skin diseases sometimes scaly at one stage—*e.g.*, eczema, lichen, erythematous diseases.	VIII. Ichthyosis.
	IX. Erythrasma.

§ **470. Psoriasis** is a common disease, occurring as irregular patches, slightly raised, covered with copious silvery scales, unattended by any exudation, and situated chiefly on the elbows and knees. The lesion starts as a tiny papule which from the first has a scale on the top (P. punctata) ; this gradually becomes larger, and is called P. guttata. In a short time it reaches the size of a coin (P. nummularis). The disease generally then remains stationary for some weeks or months, and may tend to undergo spontaneous involution. The healing process usually starts at or near the centre, and gives to the eruption a circular or serpiginous appearance (P. circinata, P. gyrata). The lesion is scaly and elevated from the first, and always dry, three features which at once distinguish it from eczema, and if the tops are scratched off bleeding hyperæmic papillæ are exposed. The distribution is extremely characteristic, being found always on the knees and elbows, frequently on the scalp, trunk, and other parts of the limbs, and only very rarely on the face, palms, or soles. There is little or no itching or subjective symptoms.

Causes.—As regards age, the disease almost always occurs in early life, though rarely before seven years old. Both sexes are equally affected. There is a considerable hereditary predisposition in some families. The seasonal influence varies in my experience, but those who are subject to the malady often complain that it recurs each winter or spring.

Diagnosis.—It is important to distinguish psoriasis from *scaly syphilide*, and in many cases the character of the lesion affords no true guide. A syphilide has more infiltration; rarely affects the elbows and knees and generally prefers the flexor aspects, and palms, and soles; the centre of the patches are usually depressed, stained, and healing; the scales are scantier, less silvery, and on being scraped off, do not leave bleeding points. *Seborrhœic dermatitis* is sometimes difficult to differentiate from psoriasis, and, indeed, Unna (the describer of this disease) holds that they are identical. The seborrhœic patches are less crimson, the scales smaller, scantier, greasier, and more orange coloured; they occur by preference on the shoulders and upper parts of the trunk, and if affecting the limbs are usually on the flexor aspects. The scalp may be affected in both diseases.

Prognosis.—Psoriasis is apt to disappear and to recur spontaneously at certain seasons. The patient may take little heed of the disease unless it affect the uncovered parts. In severe cases the eruption may spread over the whole body.

Treatment.—Psoriasis is essentially a chronic malady. Frequent bathing, followed by removal of the scales, is an essential part of the treatment. Chrysarobin (¼ to 1 drachm to the ounce) is the most valuable remedy, but the objection to its use is that it stains the linen and the skin, and frequently sets up a scarlatiniform dermatitis. For intractable cases chrysarobin paint—20 per cent. in chloroform—may be painted on once a week, and covered with a layer of collodion. Other stimulating applications, such as tar, carbolic acid, and salicylic acid, are often quite as efficacious. Plaster mulls of the same ingredients, or of mercury, are useful. In protracted cases the patient should be detained in bed for a month during treatment. X-rays often clear up a few obstinate patches, but cannot be used for widespread disease. Internal medication often does good. Thyroid extract has not fulfilled the expectations of its advocates. Arsenic, cod-liver oil, salicylates (Crocker), and copaiba (M'Call Anderson) are recommended.

§ **471.** II. **Seborrhœic Dermatitis** (Synonym: Eczema Seborrhœicum —Unna) was first described by Unna. It occurs in irregular ovoid patches, greasy, covered with brownish-red scales; quite superficial, sometimes slightly raised, with sloping margins. They sometimes heal in the centre, forming a ringed eruption which may be mistaken for ringworm. The patches vary in size from a small pea to a crown piece, or larger. Single papules may be present, brick-coloured, and soft, especially on the face. The disease is found *chiefly on the scalp* whence it may spread on to the forehead and face, then on the sternum and back of the neck and shoulders. It is often limited to the upper part of the body, but the lower parts of the body may be affected, and the legs occasionally present typical patches. The affection is more frequent than is generally thought, being often diagnosed as ordinary eczema when

from irritation or other causes it passes on to an eczema with vesicles and crusts. It is frequently associated with psoriasis ; Unna and others consider these two diesases identical (*vide supra*). The author does not share this view. The disease is more frequent in, though not confined to, young people. It is undoubtedly contagious in the author's experience, and is particularly apt to spread to those who occupy the same bed and whose pillows are apt to be exchanged. Professor Unna ascribed it originally to the bottle bacillus, Dr. Sabouraud to the staphylococcus griseus acting in conjunction with the bottle bacillus.

The *Treatment* must not only be directed to the cure of the patches which are on the body, but to the scalp, for until this is cured the eruption will constantly recur upon the body. A pomade of hyd. ox. rub. (7 to 10 grains to the ounce) should be rubbed in twice a week, or a lotion of hyd. perchlor. (5 grains to the ounce). Sulphur is equally efficacious, but is a disagreeable application (see also § 498).

Epidemic Peri-oral Eczema.—In 1895 the author had the opportunity of investigating an eruption which occurred on the faces of a large number of children in one school in the East End of London. The school contained nearly a thousand children, and quite half of these were affected in succession during three or four months. The patches occurred mostly around the mouth, on the face and neck, and rarely elsewhere. They were superficial, reddish, dry, scurfy, ovoid, somewhat resembling a superficial seborrhœic dermatitis. They were supposed to be ringworm, but this was positively excluded. The disease readily yielded to a mild tar and mercury ointment. It certainly spread from child to child, but only one of the teachers was slightly affected (*British Medical Journal*, 1896, vol. i.).

§ **472.** III. **Squamous Syphilide** (Syphilitic Psoriasis).—The squamous syphilide occurs as a later stage of the papular or the tubercular syphilitic eruptions (*q.v.*), and does not constitute a separate form of eruption of itself. It is recognised by the fact that the scales are thin, scanty, and greyish, lying upon patches of stained or infiltrated skin (*i.e.*, the syphilitic papule) which are deep brown or copper coloured, usually round, or in the shape of *segments of circles*, having raised serpiginous scaly borders. A squamous syphilide may occur on any part of the body, but the flexor aspects and the palms or soles are particularly characteristic situations, the converse of psoriasis vulgaris. A scaly syphilide of the palms is diagnosed from dry eczema by its raised serpiginous border, with sometimes an area of normal, atrophied, or pigmented skin in its centre.

IV. Certain skin diseases are scaly at one stage. A *scaly* or scurfy condition of the skin, especially of the face, is produced by hard water and exposure, and in certain states of ill-health. It is also met with after scarlatina, measles, and some of the other eruptive fevers. In *eczema*, which is a somewhat protean disease, scales and crusts form, but the presence of exudation is its essential and differentiating quality. *Pityriasis capitis* is a scaly or scurfy condition of the scalp, sometimes called also seborrhœa sicca, or dandruff (see Diseases of the Scalp). In several varieties of *lichen*, a thin silvery scale is constantly found, although they belong really to the papular eruptions. This occurs also in papular syphilide, and it is difficult sometimes to draw the line between a papular and a scaly syphilide. *Lupus erythematosus* is attended by adherent scales and crusts.

§ **473.** V. **Exfoliative Dermatitis.**—Much discussion ranges around the use of this term and the term Pityriasis Rubra, which has been employed by some as a synonym. It is best to regard the term Exfoliative Dermatitis as implying any chronic or subacute generalised inflammatory disease of the skin, whether primary or supervening upon other cutaneous disturbance of long standing, which is characterised by hyperæmia of the entire surface, and *abundant and repeated exfoliation* of the cuticle, accompanied usually by shedding of the hair and nails. There is usually some constitutional disturbance, and the itching may be severe.

Etiology.—Occasionally, as a *secondary* affection, it may follow psoriasis, eczema, pityriasis, rubra pilaris, pemphigus foliaceus, and seborrhœic dermatitis. As a *primary* condition the disease is of considerable gravity. It starts in several different ways, though a rapidly spreading hyperæmia of the integument is common to all. My belief is that the different varieties which have been described as separate diseases only differ in their mode of onset and etiology. In the *Treatment* of the malady general tonic and other internal treatment, as a rule with stimulants, occupies a prominent position. Externally soothing baths and ointments should be used.

An **epidemic exfoliative dermatitis** which the author first had the opportunity of observing in 1891[1] illustrated very well the wide varieties both in the severity and other features of this malady. One hundred and sixty-three cases occurred among the patients in the Paddington Infirmary, with a case mortality of 12·5 per cent. That the disease was epidemic and contagious was certain, that it was microbic seemed probable, though the author's later observations on epidemics at other workhouses and infirmaries tend to show that the milk consumed by the patients was in some way the means of propagating the disease amongst them, possibly having undergone some toxic or fermentative change.

§ **474.** VI. **Pityriasis Rosea** consists of numerous pink patches, slightly raised, varying in size from a pea to a florin. There is no infiltration ; slight scaling is often present at the margins. They spread peripherally, and many become circinate, with a fawn-coloured centre. It starts usually on the sides of the trunk, and may spread to the neck, upper arms, and thighs. It is rare on the face. Slight itching may be present. Pityriasis rosea runs a course of a few weeks to a few months, and disappears spontaneously. *Etiology.*—The disease occurs in both sexes, and at any age, but is most frequent in young adults. It is believed to be of parasitic origin. *Seborrhœic eczema* has greasy scales and different sites. *Tinea circinata* occurs in fewer patches, and the fungus can be found. *Psoriasis* has more infiltration and diffuse scaling. *Syphilis* has a darker colour, and is infiltrated.

Treatment.—Mild ointments of sulphur or salicylic hasten the course of the disease, and soothing lotions are useful if itching is present.

§ **475.** VII. **Pityriasis Rubra Pilaris** (Devergie), Lichen Acuminatus or Lichen Ruber (Hebra), is a somewhat rare disease in which the eruption commences as tiny papules involving the hair follicles, which become fused together into one reddened surface, and shed a succession of flaky scales. The distribution is fairly characteristic, as it starts where the lanugo hairs are mostly found—namely, the backs of the hands and forearms. In this way it often presents a glove-like distribution on the upper and lower extremities, which is very characteristic. It may spread over the whole body. The progressive margin is always marked by the same tiny scale-capped papules. The disease has to be *diagnosed* from psoriasis on the one hand, and dermatitis exfoliativa on the other. It is differentiated from *psoriasis* by its distribution and by the presence of the little papules at the spreading margin of the eruption, but is indistinguishable from dermatitis exfoliativa when the whole body is involved, except by the large flakes of epidermis in the latter. In the earlier stages dermatitis exfoliativa does not present the small acuminate papules which constitute the elementary lesion of P. rubra pilaris.

The *Causes* are obscure ; the disease usually occurs before the age of twenty-one. The malady may occur in varying degrees of acuteness. Some cases are ushered in with a certain degree of constitutional disturbance, vomiting, and some pyrexia,

[1] "Monograph on Epidemic Skin Diseases," H. K. Lewis, London, 1892 ; Med. Soc. Trans., 1891; *Brit. Med. Journ.*, Dec., 1891.

and after lasting a few months will tend spontaneously to subside. In mild cases, there are no constitutional symptoms, and the disease runs a prolonged course of many months. *Treatment.*—When it occurs in the chronic form arsenic is a remedy of some value. The same rules guide us as in the treatment of eczema.

§ **476.** VIII. **Ichthyosis** (Synonym : Xeroderma) may be defined as a congenital condition of the skin, characterised by an undue dryness and scaliness of the epidermis, and in some cases by the formation of wart-like outgrowths. Though congenital, the condition may not be identified till the child is some years old.

There are three *clinical types* or degrees of the affection. In the first or mild type (*Xeroderma*) there is simply an undue harshness or roughness of the skin, and consequently through life a great tendency to the supervention of "chaps," eczema, and other skin affections. It occurs chiefly on the extensor aspects. In a second type (*I. vera*) the superficial layers of the epidermis are thickened, and appear stretched ; the hardened cuticle presents fissures and cracks which, bounding polygonal areas, give to the patient the appearance of a fish or crocodile skin. The everted eyelids and nostrils, the atrophied hair and nails, and the hardened, scale-like condition of the skin are characteristic. In the third variety (*I. hystrix, I. sebacea, Papilloma lineare*), the skin presents a thickset aggregation, of little horny. wart-like processes which entangle the dirt, and present a brownish-black coloration. These are arranged in streaks, which were believed to follow the course of certain nerves, but a closer observation shows that this is not so, the disease being a developmental one. The diagnosis is not difficult owing to the congenital nature of the malady. Apart from the inconvenience and the liability to eczema, the first type is not serious. In the second type the disease progresses to the age of puberty, and then remains stationary. The third type rarely shortens life, but is a disfiguring malady. No known remedy influences this disease. Vaseline, lanolin, baths, and various ointments may soften the skin, and remove the superficial scales to some extent. Thyroid seems to control milder cases to some extent.

§ **477.** IX. **Erythrasma** consists of defined scaly discs of a pale red, yellow, or dark brown colour. The scales can be scraped off, and are found to contain a fungus, the microsporon minutissimum. The patches are extremely chronic, and are found on the opposed surfaces of the scrotum, thighs, axillæ, and mammæ. They itch when perspiration is excessive.

GROUP II. VESICULAR AND WEEPING ERUPTIONS.

Moist eruptions, in which the elements are usually vesicular and the exudation serous, are commonly classed into those with small vesicles (I. to VIII. below), and those with vesicles of larger size, bullæ (IX. and X. below).

I. Eczema.
II. Impetigo contagiosa.
III. Herpes.
IV. Varicella.
V. Scabies.
VI. Tinea circinata (sometimes).
VII. Sudamina.
VIII. Hydrocystoma.

IX. Dermatitis herpetiformis.
X. Pemphigus.

XI. Pustular and other diseases in which vesicles and bullæ may occur at some stage.
NOTE.—Syphilides are practically never vesicular.

§ **478.** I. **Eczema** is a catarrhal inflammation of the skin, running sometimes an acute, sometimes a chronic course, presenting a red excoriated surface denuded of its epithelium and more or less covered with crusts, associated in its acute stages with non-marginated swelling. "Weeping"—*i.e.*, a serous exudation which stiffens linen, is the chief

characteristic of eczema. Although eczema has been defined as a vesicular disease, it may present different appearances at different stages of its course. In eczema are seen, at different stages, the three primary and three secondary lesions of the skin—erythema, papules, vesicles, crusts, scales, and fissures. In the first stage, or *acute* eczema, there is erythema, with papules and tiny vesicles, which readily rupture, causing a serous exudation. By the time the physician sees the case—*e.g.*, in a day or two, the second or *subacute* state is usually reached, with excoriations and crusts, and the involved patch of skin presents a more or less swollen surface, denuded of its epithelium. If the disease passes on to the third or *chronic* stage, the discharge decreases or disappears, leaving a thickened, irregular, scaly patch, fading at its margins. Any part of the body may be affected, and to any extent; but eczema has a predilection for the flexor aspects of the limbs and the flexures of the joints. The patient complains of a burning, smarting, throbbing, or itching, in proportion to the acuteness of the process.

The *Diagnosis* of eczema is not difficult. *Seborrhœic dermatitis* has no serous exudation, and is covered by greasy yellow scurf. *Syphilides* never resemble acute or subacute eczema, or, indeed, any vesicular disease, a fact of considerable value in practical diagnosis. It is difficult sometimes to distinguish patches of dry chronic eczema from *psoriasis*, but the latter affects characteristic localities, preferably the extensor aspects, and is covered with silvery white scales. The diagnosis of the numerous varieties will be given below.

The history or evidence of a *cause* should be sought. (1) *Local Causes.*—Eczema is the lesion most frequently following the dermatitis produced by local irritants, such as a mustard plaster, turpentine, the leaves of certain plants (*e.g.*, the primula obconica), or soaking the hands in water containing soda. Eczema forms around the eyes treated with eye-drops; and excessive sweating may produce the condition. It often occurs around the mucous orifices from which an irritating discharge issues (the nose, ears, anus, etc.), and under these circumstances is very intractable. The local hypostatic congestion attending varicose veins is a frequent predisposing factor. Unrecognised scabies or pediculi lead to protracted cases of eczema of multiform character. (2) *Constitutional Causes.*—Eczema may occur as a complication of dyspepsia, gout, diabetes, or renal disease. In cases of eczema of the vulva or prepuce, the urine should be examined for sugar, and the presence or absence of leucorrhœa ascertained. It often accompanies albuminuria especially if dropsy be present. It may appear during every pregnancy, or after the cessation of lactation, when neuropathic influences are at work.

Varieties.—In addition to the typical acute and chronic forms described, there are several varieties of eczema. (1) In *E. papulosum* the process stops at a papular stage. (2) *E. pustulosum* or *impeti-*

ginodes is due to a secondary invasion by pyogenic cocci, occurring frequently on the heads or faces of children, on the hairy parts of delicate and tuberculous persons, or when any local irritation is present ; it is attended by the formation of crusts. (3) *E. rubrum* occurs usually on the legs, chiefly in old people, where the deficient circulation produces a livid colour, and prevents repair. (4) In *Vesicular E.* the vesicles are prominent, tend to come in crops, and become confluent. Its favourite localities seem to be the face, the ears, and the flexures of the limbs, fingers, and toes. Some cases of this disease resemble dermatitis herpetiformis. (5) *E. squamosum* is a chronic scaly stage into which many erythematous and papular varieties develop, and is found most frequently on the palms, legs and scalp, and is apt to be mistaken for psoriasis and squamous syphilide. (6) Occurring in different parts of the body, eczema is often named from the locality—E. capitis, E. ani, E. intertrigo, but it is unnecessary to invent special terms for these varieties, except, perhaps, in the case of *E. palmaris*, which is often due to the sufferer's occupation (baker's itch, washer-woman's itch, etc.). (7) In the palms it is apt to become chronic, dry, thickened, and fissured (*E. rimosum*).

(8) *Cheiro-pompholyx* is the term given to a vesicular and bullous eruption affecting the hands *symmetrically*, and sometimes the feet at the same time. The thickness of the epidermis in this situation prevents the rupture of the vesicles. These are especially prone to appear in the clefts between the fingers and toes, like boiled sago grains, and creep on to the palmar and dorsal surfaces—an important diagnostic feature between this disease in its later stages and a scaly syphilide of the palms. Some of the vesicles coalesce into bullæ, their contents become absorbed, and exfoliation of the epidermis occurs.

(9) *E. Marginatum* is tinea circinata of the groins and genitals.

(10) *Paget's disease* of the nipple is really a malignant form of eczema. It starts on the nipple of one or both mammæ, and spreads centrifugally, sometimes with a slightly raised margin, leaving a reddened, congested, and sometimes weeping surface. It is met with mostly in females of advancing life, and it consists of a slowly-growing cancerous process.

(11) *Peri-oral Eczema* (§ 471) is a term which is applied by the author to an epidemic condition which he observed in a large board school in 1895.

The *Treatment* of eczema differs materially according to the stage of the disease. The principles of treatment are those underlying the treatment of all diseases of the skin. In the acute inflammatory stages, when there is much erythema or vesiculation, the indication is rest indoors or in bed if necessary, with such soothing applications as zinc oxide, calamine, lead, or bismuth, especially in the form of lotions, powders, and occasionally ointments. Lead or weak creolin lotions are comforting ; and for acute eczematous conditions I have found nothing more successful than prescription (F. 42), or lot. calamin. co. Saline aperients are useful ; and vin. antimon. ℥x. with mag. sulph. ½ dr. t. d., if used early, seems to sometimes cut short the disease. A daily bath (say a teaspoonful of creolin to 15 gallons of warm water) is indicated whenever the eruption is widespread, and among out-

patients in whom want of cleanliness is a potent causal factor. In subacute stages a stimulating agent should be added to the soothing remedy; and in the chronic stages change of air (to hilly country in preference to seaside) and stimulating remedies. These are tar, mercury, resorcin, creosote, carbolic acid, salicylic acid, and sulphur. But a mere knowledge of the drugs to be employed is not sufficient; it is necessary to follow certain rules in their application. Thus for weeping surfaces lotions, not ointments, should be chosen; for thickened surfaces ointments should be not merely smeared on, but spread on a piece of lint, and firmly applied to the skin, so that a macerating effect may be obtained. The same results may be obtained and the part protected by the use of medicated plaster mulls, the most useful of which, perhaps, is a weak tar and mercury plaster (4 per cent. of the former and ¼ per cent. of the latter). It is of no use to apply remedies over thickened crusts or scabs; these must first be removed by means of bread or starch poultices, or wiped away with olive oil. When the surface is thus cleaned it must be kept aseptic by excluding the air and renewing the applications every four hours for acute, and once or twice a day for chronic, conditions. Lotions should rarely be covered with gutta-percha tissue to keep them moist, because the part becomes sodden. Eczematous parts must never be washed with soap and water, but when ointments are used, they may first be cleansed with sweet oil or creolin lotion (1 drachm to 1 pint). In the latter stages an ointment containing mercury or tar may be used (*e.g.*, F. 104), the stimulating agent being cautiously increased. If there is much secretion an astringent lotion (such as lead or creolin) may be first employed to cleanse the part, while if the part is dry and scaly, the tar and mercury may be increased without danger. In cases of long duration in which considerable thickening exists, our chief object should be to remove the products of disease so that the healthier underlying structures may resume normal growth. This may be done by the active application of exfoliating remedies such as salicylic acid (20 to 30 grains or more to the ounce) and sulphur. In very inveterate cases the previous application of strong plaster mulls (*e.g.*, salicylic acid 10 to 30 per cent. and creosote 10 to 40 per cent.) facilitates the process, though it may at first appear to make it worse by setting up an acute inflammation. Tonics and internal remedies should be administered, such as iron, alkalies and gentian, etc., to correct any morbid general condition; arsenic should seldom be used except in chronic non-inflammatory cases. In the intractable eczema affecting the extremities of the aged, I have found small doses (1 to 3 minims) of opium of great use. Constitutional treatment is specially indicated when the eruption is widespread or generalised. The digestion must be put in order, and when there is any marked tendency to erythema or congestion three remedies are of great use—alkaline carbonates, quinine, and calcium chloride, all in fairly large doses.

Treatment of Varieties.—E. of the *eyelids* requires careful treating, lest the ointment should set up conjunctivitis. The following ointment —hyd. ox. flav. and acid borici, āā gr. ii., aq. dest. and ol. amyg. dulc. āā gr. xxx., lanolin 1 oz.—may be found useful. For eczema around *mucous orifices*, the great indication is to keep them dry, and a powder consisting of equal parts of zinc oxide, bismuth carb., and calamine frequently dusted on gives relief. Calamine lotion is also useful. For *E. papulosum* a creolin bath and the removal of the offending garment suffices ; for *E. impetiginodes* ung. hyd. am. is almost a specific ; *cheiropompholyx* is treated on the same lines as acute E. ; in *E. palmaris* gloves saturated with the ointment should be worn at night, and if possible by day, and a small quantity of ointment should be smeared on after every washing ; *Paget's disease* is an indication for removal of the breast (see also § 499).

II. **Impetigo Contagiosa** consists of discrete vesicles of varying size which soon become pustular (§ 484).

Streptococcal and staphylococcal skin infections give rise to many different kinds and sizes of vesicular eruptions of the impetigo class, which may be mistaken for eczema or herpes on the one hand, and pemphigus or urticaria bullosa on the other.

§ **479.** III. **Herpes** (Synonyms : Herpes-Zoster, Zona, Shingles) may be defined as an acute non-contagious disorder, consisting of one or more *clusters of vesicles* on a crimson base, associated with neuralgic pain, and due to an irritative lesion of one of the ganglia of the posterior spinal roots or their analogue the Gasserian. Herpes commences with a red patch or a group of flat papules, on which vesicles very rapidly appear. The vesicles are larger than those of eczema, round, hemispherical, and uniform in size, about as big as a millet seed ; and as there is no tendency to spontaneous rupture there is usually no oozing such as occurs in eczema. They smart or burn, and the neuralgic pain which precedes and follows the affection is often very severe and intractable. The vesicles contain clear serum, and after lasting a few days, dry up and form little crusts. The whole attack lasts on an average three weeks. The vesicles leave no ulceration and no scars. The affection is nearly always unilateral. Formerly a patch of herpes was thought to correspond with a sensory nerve distribution, but the groups correspond more precisely with what is known as a sensory area ; that is to say, an area which has been shown by Mackenzie, Thorburn, and Head to represent the terminal distribution of the pain-appreciative (algetic) fibres connected with each spinal segment (§ 558). Many *varieties* are named according to their position—*e.g.*, H. frontalis, ophthalmicus, brachialis. H. febrilis (facialis or labialis) often accompanies inflammations of the respiratory tract. H. progenitalis, or preputialis, which occurs on the genital organs of both sexes, sometimes alarms patients with the dread of syphilis. Serious varieties are *zoster*

hæmorrhagicus, in which hæmorrhage occurs into the vesicles, and *zoster necrogenica* in which the skin sloughs, and is followed by scarring or keloid. *Diagnosis.*—Herpes may be distinguished from all other vesicular conditions by the occurrence of the vesicles in *clusters* or constellations and their erythematous base. In regard to *Prognosis*, herpes tends to spontaneous recovery in the course of a few weeks, except in the two grave forms above mentioned. The neuralgia which succeeds, however, is often very intractable, especially in the aged.

The *Treatment* is quite simple. Protect the vesicles by starch or zinc powder, or paint with collodion, or use some soothing ointment. Quinine in large doses, 5 grains three or four times a day, is reputed to be the best remedy for the neuralgia. For herpes preputialis give lead lotion.

IV. **Varicella,** the description and differential diagnosis of which has been dealt with (§ 352 and § 355).

TABLE OF DIAGNOSIS.

Varicella.	*Small-pox.*
No symptoms before rash.	Three days before rash, sudden onset of illness with backache.
Soft pink papules becoming vesicular.	Shotty papules becoming vesicular or pustular.
Chest, neck, and trunk, rarely face and limbs.	First on face and wrists, then trunk and legs in regular order.
Successive crops, and thus find small papules besides vesicles of various sizes.	**All one stage** (papular or vesicular, or pustular) at one place.

V. **Scabies** is chiefly a papular eruption (§ 465). But in children the vesicular element is apt to predominate, and it may then be mistaken for varicella. The burrows, and the marked tendency which scabies has to affect the soft skin at the bends of the joints and between the fingers should obviate such a mistake.

§ **480.** VI. **Tinea Circinata,** or ringworm of the body, is occasionally vesicular, especially when occurring on the wrists of adults, the arrangement of the vesicles in the form of a definite ring being so characteristic as to be unmistakable (Fig. 134). The usual naked-eye appearance of this lesion is a pale red ring with a scurfy margin. With a lens the margin is seen to be slightly raised with minute papules or vesicles. The favourite localities are the face, neck, and arms. When occurring in the genito-crural region it used to be (improperly) called **eczema marginatum.** On examining scrapings under the microscope, the mycelium (Fig. 135), and perhaps a few spores of the tricophyton tonsurans (the large-spored fungus, § 498) can be seen. The treatment consists of rubbing in ung. hyd. amm. chlor., or some other parasiticide.

§ **481.** VII. **Sudamina** are clear, scattered, non-inflammatory vesicles, like droplets of water, about the size of a pin's head, occurring in conditions such as acute rheumatism, which are attended by very profuse perspiration. They do not give

rise to any inconvenience, and disappear in a few days. They are a non-inflammatory disorder of the sweat glands, whereas *miliaria* (commonly called prickly heat) are a mildly inflammatory condition of the same glands characterised by similar papules and vesicles on a red base.

VIII. **Hydrocystoma** is a rare disease characterised by deep-seated, tense, translucent vesicles occurring on the face, varying in size from a pin-head to a pea, very persistent, lasting for months. They are formed by a cystic swelling of the duct of the sweat-gland, and never become purulent. They disappear spontaneously, chiefly in cold weather. The disease occurs chiefly in middle-aged women, especially in those whose life is spent in a warm, moist atmosphere, or who perspire much. It frequently comes on as warm weather sets in. The *Treatment* consists in puncturing the vesicles.

§ **482.** IX. **Dermatitis Herpetiformis** is the term used by Duhring[1] for a large and widely varying group of eruptions which were formerly classed under pemphigus and various other headings.[2] Dermatitis herpetiformis may be defined as a relapsing disorder of prolonged duration, characterised by the appearance of successive crops of erythematous or papular elements, always in clusters, which usually go on to the formation of vesicles, pustules, or bullæ, are always attended by intense irritation, and sometimes by pigmentation. Different varieties are described according to the element which predominates. In some (the *erythematous* variety) the preponderating eruption consists of circumscribed patches of bright red erythematous or semi-urticarial inflammation, which spread by raised edges, and leave a pigmented centre. In another, and perhaps a commoner variety (the *papulo-vesicular*), vesicles which vary from the size of a pin's head to a split pea predominate. Sometimes these vesicles become bullæ as big as a walnut (the *bullous* variety). They are always in clusters of two or three, and may coalesce, and sometimes the vesicles become pustular. Sometimes the predominating element is a papular one, and because of the itching the heads

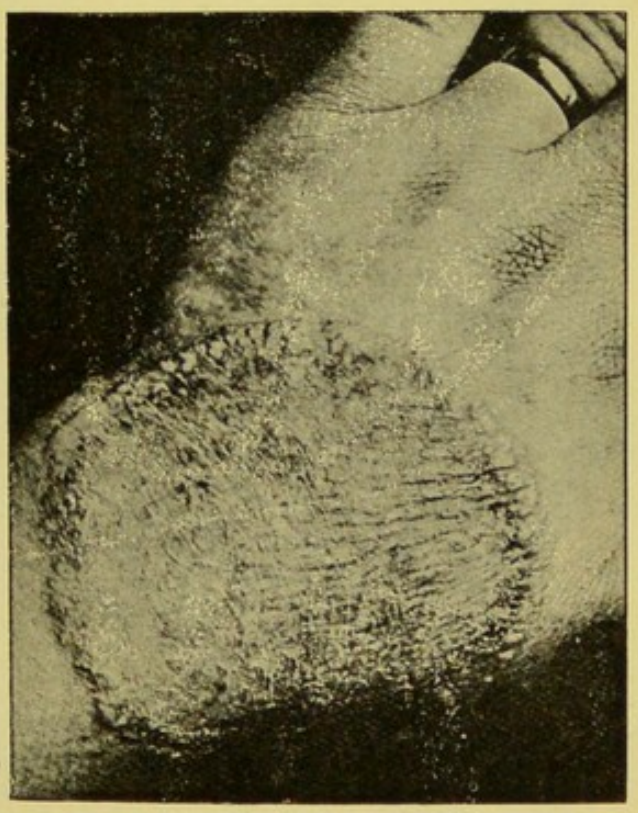

Fig. 134.—Hand of a woman suffering from TINEA CIRCINATA, in which the vesicular element is unusually prominent. A large ring of vesicles encloses a scurfy area. Verified by microscopic examination.

[1] *American Journal of the Medical Sciences*, February, 1891, and elsewhere; also New Sydenham Society's publications.

[2] Synonyms of Dermatitis Herpetiformis: Pemphigus pruriginosus; pemphigus circinatus (Rayer); pemphigus composé (Devergie); herpes gestationis (Milton, 1872); herpes circinatus bullosus (Wilson); herpes phlyctenodes (Gilbert); erythema bullosum; dermatitis multiformis (Piffard); hydroa gestationis (Liveing); hydroa herpetiforme; hydroa bullosus; pemphigus arthritique (Bazin); Duhring's disease; impetigo herpetiformis of Hebra; dermatite polymorphæ prurigineuse, ou douloureuse, chronique à poussées successives (Brocq).

of those become scratched, and covered with blood-stained scabs. Still more often these various elements are commingled, for the different lesions apparently represent different stages of the same pathological process. The intense itching is a very notable feature in all the varieties, especially the papulo-vesicular one. Scars and temporary pigmentation may ensue. In many cases the general health seems undisturbed, but often the appearance of each crop is attended by pyrexia, and occasionally gastro-intestinal disturbance. As regards its distribution, the elements have a tendency to be symmetrical, and to favour the flexor surface of the wrists, the axillæ, groin, abdomen, and buttocks. The mucous membrane of the mouth and pharynx may also be involved. Each successive crop lasts from one to four weeks; each attack being separated by longer or shorter intervals of comparative freedom. In this manner the disease may go on for months or years.

Diagnosis.—The disease differs from pemphigus vulgaris in the following respects: (1) The smaller size of the vesicles or bullæ, which are (2) constantly arranged in clusters; (3) the presence of erythematous patches beneath the vesicles and elsewhere, and the multiform character of the eruption; and (4) the presence of itching. Nevertheless it will be seen that all these differences are more questions of degree than of kind. From *eczema*, *urticaria bullosa*, and *erythema multiforme* the disease is distinguished by consideration of the above features.

Fig. 135.—Mycelium of TINEA CIRCINATA (ringworm of the body).—A scraping from the skin, stained by Gram's method. Mycelium of equal segments having truncated ends bifurcating in places; spores almost absent. Compare Fig., p. 705.

Etiology.—The disorder is more common in men than in women, and between the ages of sixteen and thirty. Many regard it as a dermato-neurosis, by reason of its symmetry and tendency to attack neurotic individuals.

Treatment.—Arsenic is of great service, and should be given in full doses. Quinine and salicin, and phenacetin for the irritation are useful. Locally, sedative lotions and ointments may be prescribed, much the same as those recommended in acute eczema (*q.v.*), while Duhring advocates sulphur ointments, 2 drachms to the ounce, and tar lotions—*e.g.*, liquor carbonis detergens, 5 drachms to the pint.

§ **483.** X. **Pemphigus** is one of the rarer diseases of the skin, characterised by the presence of bullæ and constitutional symptoms of a mild or severe nature. *P. chronicus* or *vulgaris* is the more common and typical variety in which the bullæ develop in crops, each bulla varying in size from a pea to a hen's egg, being tense with clear fluid, which becomes turbid, purulent, and occasionally hæmorrhagic (P. hæmorrhagicus). The bulla is characterised by having no ring of erythema round its base. The fluid is either absorbed with formation of crusts, or the blebs burst, leaving a raw surface on which new epidermis soon develops. Almost any part of the skin may be affected, as well as the mucous membrane of the mouth and nose. While each bulla only lasts a few days fresh crops may continue to come out for several months. The constitutional disturbance depends largely upon the number of bullæ and the frequency of the crops, the prognosis for recovery being

good when the number is small, and grave when abundant. Many cases are apt to recur throughout life. In *P. foliaceus* the bullæ are very thin and flaccid, and rupture early ; but the epidermis, instead of re-forming, continues to peel off until large areas of red, raw, exuding surface are exposed, with epidermis folded at the margins—a point which distinguishes it from eczema rubrum. This process slowly extends for a year or two until the whole body may be involved, and a fatal issue ensues. *P. vegetans* is a rare variety, which develops papillomatous vegetations on the base of the bullæ, and usually ends fatally in a few months. The *Etiology* is obscure. It is more frequent in infancy, and is about twice as common in the male (Kaposi). At different times pemphigus has been associated with renal disease, anomalies of the female sexual organs, and lesions in the spinal cord or sympathetic system. Contagion has often been suspected, and various observers have found bacteria in the bullæ, but the etiological value of such findings is not established. The treatment of pemphigus is usually regarded as rather hopeless ; but arsenic in gradually increasing doses in some cases seems to have quite a specific action. Quinine and other tonics and a generous diet are called for, especially in cachectic cases. Among local remedies the continuous bath in severe cases, and dusting powders or boracic ointment to protect from septic influences in milder cases are the best.

XI. The description of the other diseases occasionally characterised by vesicles or bullæ—erythema multiforme, urticaria, drugs, congenital syphilis, leprosy, etc., will be found under their respective headings. Occasionally streptococcic dermatitis gives rise to large bullæ.

GROUP III. PUSTULAR ERUPTIONS.

Eruptions in which the elements are mainly pustular naturally fall into three classes.

a. Superficial Pustules.

I. Impetigo contagiosa.
II. Ecthyma.

b. Pustules on an Indurated Base.

III. Pustular syphilide.
IV. Sycosis.
V. Pustular acne.
VI. Pustular folliculitis.
VII. Bromide and other drug eruptions.
VIII. Variola.
IX. Acute glanders.
X. Pustular tuberculide.

c. Furuncular Eruptions with a Slough.

XI. Boils.
XII. Carbuncles.
XIII. Kerion.

Hebra wrote in 1870 "all pustules must be regarded as secondary morbid products, and hence are not fitted to form an independent series of cutaneous diseases." Curiously enough almost the only exception to this dogma is to be found in that rare condition Impetigo Herpetiformis, which was first described by Hebra. *Eczema and all the diseases mentioned in Group II. may become pustular*, owing as we now know to infection by pyogenic cocci. Conversely, nearly all the pustular diseases just mentioned may start as vesicles.

According to Dr. Sabouraud (the *Lancet*, August 10, 1901, p. 403, and *Brit. Med. Journ.*, August 3, 1901), there are three prevailing cocci found in the skin (1) The

streptococcus which is the cause of the impetigo contagiosa of Tilbury Fox, and for this he recommends 1 or 2 per cent. zinc sulphate; (2) staphylococcus aureus, which invades the follicular orifices and causes pustules, being the cause of all primary and secondary pustular lesions, and for this he recommends sulphur 19 grammes, alcohol 30 grammes, aq. rosæ 100 grammes; and (3) staphylococcus griseus, which causes pityriasis simplex and seborrhœic dermatitis, and is the same as the morococcus of Unna. For this he recommends oil of cade 10 grammes, yellow binoxide of mercury 1 gramme, and pet. moll. 30 grammes. All these three organisms Dr. Sabouraud suggests are probably polymorphic varieties of one and the same microbe.

a. Superficial Pustules.

§ **484.** I. **Impetigo Contagiosa** (Synonym: Porrigo) is frequently met with on the faces of children, and is so called because it is readily conveyed from one child to another. At first the spots are vesicular, but they become pustular in a few hours. The pustules vary in size, and are discrete, but may run together if near each other. In the course of a few days they dry into yellow crusts, which, falling off, leave a flat congested mark covered by new cuticle. They do not leave scars unless scratched. The favourite positions are the face, especially round the mouth, scalp, and hands of children, but they may occur on any part of the body. If untreated, fresh pustules appear in other places for a week or two; or the disease may die out spontaneously in a few weeks. It is usually trivial, without constitutional disturbance, and with only slight itching. The disease may be conveyed by contagion to other parts of the same or to another individual.

Etiology.—Impetigo will spread through a school or family of children attacking weak and strong alike. Adults enjoy remarkable immunity, but occasionally an adult will contract it from a child. The essential cause is a streptococcus. Coccal infections of this class may give rise, as just mentioned, to vesicular and vesico-pustular eruptions of several kinds.

Diagnosis.—Impetigo pustules are readily distinguished from acne, sycosis, pustular syphilide, and all other pustular eruptions by (i.) their superficial character, and (ii.) their typical localities.

The *Treatment* is extremely simple. The crusts must first be removed with warm water, a little sweet oil, or a starch poultice; and then a few applications of sublimate solution, or ung. hyd. am. chlor. are sufficient. Mercury is almost a specific in pustular affections.

Eczema impetiginodes is really a pustular form of eczema as already described.

Impetigo Herpetiformis, or, as it might be more properly called, Pyæmic Impetigo, is a rare disease described by Hebra, the differentiation of which from dermatitis herpetiformis is often difficult. It is a pyæmic process of the skin, characterised by the appearance of clusters of miliary pustules, usually starting on the inner surface of the thighs, whence they spread to almost the entire integument, generally associated with the pregnant or puerperal state, and terminating fatally. The tongue, palate, pharynx, and even the œsophagus have been the seat of pustules and superficial ulceration. There is considerable fever of pyæmic type, with

delirium and vomiting, and each fresh crop of pustules is attended by rigors and increasing prostration. All but two of the recorded cases have been connected with the later months of pregnancy, and if the patient recovers from the first attack the disease seems to be apt to recur in a subsequent pregnancy. Nearly all the recorded cases have terminated fatally sooner or later.

The *Treatment* should be conducted on the lines of other septicæmias. Vigorous local measures should be employed ; probably the acid nitrate of mercury vigorously applied in the early stage might cut the process short. But when the disease is established it would perhaps be better to make hourly applications of 1 in 1,000 corrosive sublimate (after the manner of cancrum oris, § 147). Any vaginal or uterine discharge should be attended to by frequent irrigations.

II. **Ecthyma** is a term which is gradually falling into disuse, but is still sometimes used to describe *large isolated pustules*, or the superficial sores which form part of impetigo, scabies, pediculosis, etc., in children and aged persons broken down in health, and wanting in personal cleanliness. They may have to be distinguished from scabies in children and from suppurating syphilides.

b. Pustules on an Indurated Base.

§ **485.** III. **Pustular Syphilides** are of two types : (1) *Small Papulo-pustular Syphilide* (Acneform Syphilide, or Lichen Syphiliticus Pustulosus), consists of spots about the size of a pin's head, upon a hard base, which in a week or ten days scab off, leaving the characteristic indurated papules with depressed centres. They are arranged in groups, circles, or circular lines. (2) *Large Pustular Syphilide* (Rupia, Ecthymatous Syphilide, Variola Syphilitica) consists of pustules varying in size from a split pea to a halfpenny, flat or hemispherical, and surrounded by a raised brick-red infiltrated margin. They may be grouped, ringed, or isolated. The pustule bursts, the pus escapes, and crusts are formed with ulceration beneath them. The ulceration tends to spread serpiginously, and leaves permanent scars, rings, and pigmentation. Both varieties may occur on any part of the body, and both indicate a malignant type of syphilis. The smaller pustular syphilide may have to be *diagnosed* from *acne* by the presence of comedones, and slower course in the latter. When on the face pustular syphilide may be hard to distinguish from *lupus vulgaris*, but the youth of the patient in the latter complaint, and the extremely slow rate of progress, may aid us. When pustular syphilide is diffuse, it may be mistaken for *variola*, but in the latter there is a history of a vesicular stage, of backache, and constitutional symptoms.

§ **486.** IV. **Sycosis** is a term applied to a slowly growing papulo-pustular eruption affecting the sebaceous glands and hair follicles of the beard and sometimes the moustache. Indurated papules at first appear, and some of these undergo suppuration. The disease is usually contracted at the barber's shop, but may be taken from horses and other animals suffering from ringworm. Clinically there are three conditions which present the appearance which we describe as sycosis (συκον = a fig). (1) True Sycosis (Synonym : Folliculitis Barbæ) in which the pustules become large and indurated, and in this later stage

the hairs can be easily drawn out with their macerated and softened sheaths entire. This is due to staphylococci, and on that account is sometimes called coccygenic sycosis. It is usually contracted at the barber's, or it may be due to a nasal discharge. (2) Tinea Sycosis (Synonym : Hyphogenic Sycosis) is ringworm of the beard due to the trichophyton tonsurans. The large spored ringworm in children, and the ringworm of horses, cows, cats, and dogs may produce this disease. There are two varieties : (*a*) *Superficial*, characterised by scaly red rings, in which the hairs are only slightly involved ; and (*b*) *deep-seated*, in which hard nodules and lumps are formed with suppurating hairs. In this form the hairs are easily pulled out from the onset. (3) *Eczema barbæ* taking on suppuration consists primarily of a general inflammation of the skin with pustules around some of the hair follicles secondarily. In true sycosis the pustules are the predominating element, and the intervening inflammation is secondary. In eczema barbæ the eczematous condition affects the intervening parts, and spreads on to the face as well. All three conditions may, if untreated, last for a considerable time.

It is sometimes very difficult to *diagnose* which of the three conditions named we have to deal with ; unless the trichophyton be found. In *impetigo contagiosa* the pustules and crusts are quite superficial and readily distinguished from sycosis. The *Treatment* is prolonged. The hair should be kept short. Salicylic and carbolic acids and mercury are, in my experience, the best reagents to use. Epilation by X rays is the most rapid method of cure. The eczematous variety is treated like other eczemas (§ 478).

§ **487.** Various other pustular eruptions may be mentioned :

V. **Pustular Acne** is recognised at once by the presence of comedones, papules, and pustules on the face, and sometimes the upper part of the back. This and the severe pustular form known as *acne varioloformis* is described in § 463.

VI. **Pustular Folliculitis** is a papulo-pustular condition specially affecting the hair follicles, due to a pyogenic infection (staphylococcus aureus, according to Sabouraud). It is independent of eczema, and affects only the hairy parts, especially the legs in men. It may be distinguished from a syphilide by the fact that each papule or pustule involves a single hair follicle.

VII. **Iodides** and **Bromides** sometimes produce pustular eruptions on a congested indurated base. Antimony, aconite, arsenic, sulphide of calcium, nitric and salicylic acids also produce pustular eruptions.

VIII. **Variola** (Small-pox).—The concluding stage of the eruption in this infectious fever is another illustration of pustules forming upon an indurated base (§ 355).

IX. The eruption of **Acute Glanders** when it has reached a pustular stage is so much like small-pox that it may very pardonably be mistaken for it (§ 362).

X. A pustular **Tuberculide** is described by some observers as a rare manifestation of tuberculosis.

c. Pustular Eruptions prone to become Furuncular, or Sloughing: viz., Boil, Carbuncle, and Kerion.

§ **488.** XI. **A Furuncle,** or boil, is an acute, circumscribed, suppurative inflammation in the skin, varying in size from a small pin's head to a bean. When the process fails to pierce the skin it is called a *blind* boil. As a rule, however, the inflammation involves the surface, which breaks and permits, in the course of a few days, of the discharge of the central necrosed portion, which is spoken of as the core. The cup-shaped cavity which is left heals by granulation, and a scar remains. The pain is considerable, especially in the early stages, and varies with the tension of the part. A specially severe form of boil is the " cadaveric boil," which is due to infection during a post-mortem examination. *Furunculosis* is the term applied to the condition in which boils are constantly recurring over a prolonged period at different parts of the body.

Treatment.—Protect the part from external irritation. Frequent ablution is necessary. Hot carbolic compresses relieve the pain and hasten recovery, and a weak tar ointment may be employed. Iron, strychnine, and sulphuric acid are useful, and the general nutrition should be improved. Vaccine treatment has been successful, and high-frequency currents abort boils.

XII. A **Carbuncle** may be regarded as a combination of several boils side by side, constituting an inflammatory area of considerable size spreading beneath the skin, with numerous openings in the skin through which the pus pours. A leathery slough forms as it were a sheet in the deeper layers of the derma. Its commonest position is the nucha or back, but it may occur on the sacral region from pressure, on the face (when a neurotic element can generally be traced), or elsewhere. The patient is nearly always advanced in years, and is often lowered in health in some way. The pain and constitutional disturbance are often very severe, and if the carbuncle be extensive general pyæmia may ensue, or death from exhaustion. The *Diagnosis* is never difficult, on account of the characteristic red, infiltrated, swollen, circumscribed area in one of the positions named, and honeycombed appearance with pus-discharging holes. The *Causes* are much the same as those of boils, though carbuncle is more often the result of debilitated states and diabetes, and is more often found in the aged suffering from cardio-vascular disease.

Treatment.—Warmth is in my experience most useful to check the extension, or, if this is impossible, to promote suppuration, and the separation of the slough. When by unmistakable fluctuation we know that suppuration has ensued, free crucial incisions should be made, the slough cut away as freely as possible, and frequent syringings every hour or so with a carbolic lotion, 1 in 100, adopted. Iron, arsenic, strychnine, and a liberal diet should be freely administered.

XIII. **Kerion** is a condition described by Celsus, and occurs chiefly on the heads of children suffering from neglected ringworm. Superficially it resembles a carbuncle, but without the same induration. It is a circular, raised, inflamed, boggy area of skin through which are a number of pus-discharging holes. It is commonest on the scalp and beard, and is due to the large spored ringworm (see § 498).

GROUP IV. MULTIFORM ERUPTIONS.

Multiform eruptions are sometimes found in the following conditions —syphilis, scabies, eczema, erythema multiforme, varicella, leprosy, and dermatitis herpetiformis.

§ **489. General Characters of Syphilitic Eruptions.**—Syphilitic eruptions have already been referred to under Syphilis (§ 404) and under papular and scaly eruptions. (1) They are of many different *kinds*, and several kinds may be present at one time (polymorphism). All kinds of elementary lesions may appear on the skin with the single exception of vesicles; eczema and other vesicular lesions are never found as a result of syphilis, a diagnostic feature of great importance. (2) The syphilitic *papule* may be regarded as a prototype of a syphilitic skin lesion. It is the starting-point of them all. (3) The *features common* to all syphilitic rashes are their reddish-brown colour, generalised or symmetrical distribution, grouping in segments of circles, preference for the forehead and flexor aspects, polymorphism and absence of itching. The later skin lesions in malignant cases (in which a so-called tertiary stage occurs) differ, however, in being asymmetrical, and with a marked tendency to ulceration.

The clinical features which distinguish syphilides are explained by three histological facts. (1) All syphilides are due to a deposit in the dermis or epidermis of a cellular *infiltration*. Hence the colour does not disappear on pressure, and is followed by staining. (2) The cells constituting this gummatous or granulomatous infiltration are of low vitality. They do not organise into connective tissue, but tend to undergo either *involution* by absorption on the one hand, or *suppuration* and pustulation on the other. Hence the depressed cup-shaped centre, and the great tendency to polymorphism. Hence also the absence of vesiculation or an eczematous form of eruption. (3) The infiltration *spreads centrifugally*. Hence the raised peripheral edge is the newest part, the shape most frequently assumed being that of a crescent, circle, or segment of a circle leaving a stained centre where the papule began. If these three principles be appreciated all the clinical features are explained.

Scabies (§ 465), as it occurs in children, is nearly always a multiform eruption, consisting of papules, vesicles, sometimes pustules, scratch-marks, and burrows. By the presence of the latter and the position of the eruption the diagnosis is arrived at.

GROUP V. NODULAR ERUPTIONS AND TUMOURS OF THE SKIN.

A nodule may be defined as a solid deposit in the skin, which is larger than a papule. The *commoner forms* are: I. Lupus Vulgaris; II. Syphilitic Gummata; III. Various Benign Tumours (*e.g.*, sebaceous cyst, lipoma, rheumatic nodules, vascular nævi, etc.); and IV. Epithelioma; while the *rarer forms* include: V. Leprosy; VI. Bazin's Disease; VII. Molluscum Contagiosum; VIII. Molluscum Fibrosum; IX. Sarcoma Cutis; X. Actinomycosis; XI. Yaws; XII. Mycosis Fungoides; and XIII. Blastomycosis.

§ **490.** I. **Lupus Vulgaris** may be summarily defined as a chronic disease of the skin, characterised by a collection of reddish-brown, semi-translucent ("apple-jelly") nodules embedded in the corium, which give rise rise to some general thickening and desquamation, and have a tendency to ulcerate and to result in cicatricial atrophy. Their favourite position is the face, in which postion the patches are rarely symmetrical, as in l. erythematosus. They occasionally affect the limbs—in 20 per cent. of the cases according to Kaposi. The disease almost invariably starts early in life—in childhood.

TABLE OF DIAGNOSIS.

Nodular Syphilide.	*Lupus Vulgaris.*	*Lupus Erythematosus.*
Nodular or diffuse infiltration with raised edges.	"Apple-jelly" nodules in derma. Sebaceous follicles not specially involved.	Superficial erythema. Sebaceous follicles plugged with hard sebum.
Destroys more in a month than lupus in a year. Stellate scarring.	Destroys slowly and usually leaves puckered scar.	Never ulcerates, though may leave a superficial scar.
Sometimes symmetrical.	Asymmetrical.	Bat's-wing distribution on face. Generally symmetrical.
Adults.	First appears in childhood.	First appears in middle life.
Amenable to Hg and KI.	Hg and KI do harm.	Hg and KI no good.

The *Prognosis* of lupus vulgaris turns principally on three things: (1) Its position, (2) its extent, and (3) the general condition of the patient. Untreated, the disease will spread for years.

Treatment.—General hygienic and tonic measures as for phthisis are useful—*e.g.*, good food, fresh air, malt, and cod-liver oil. Local treatment consists in (1) excision, which is only applicable to a very circumscribed area of disease; (2) curetting, followed by caustics or the cautery, the result of which is often very satisfactory in a localised patch, but unfortunately in most cases, requires periodic repetition; (3) Finsen lamp (photo-therapy), which is best suited to limited patches and yields excellent results; (4) X rays (radio-therapy); (5) high-frequency currents; (6) hypodermic injections of tuberculin have produced good effects, and are particularly suitable for lupus scattered widely over the body; (7) caustics and escharotics like acid nitrate of mercury carefully applied, carbolic and salicylic acid, etc., are extremely useful to arrest, and even, by prolonged treatment, to cure the

disease when other methods are not available. Prolonged exposure to bright sunlight when available is also curative.

II. **Syphilitic Gummata** occur in the skin for the most part in the later stages of the disease. They are met with as round or ovoid nodules in or beneath the skin. In the course of a few weeks they usually make their way to the surface in the form of an indolent abscess, which leaves a circumscribed punched out ulcer, sometimes of considerable depth. They may occur anywhere, but especially on the legs, brow, and sterno-clavicular region. They should not be lanced.

III. There are several other relatively common **Benign Tumours** or nodules originating in the subcutaneous tissue, which may involve the skin—*e.g.*, sebaceous cyst, fatty tumour, rheumatic nodules, fibro-neuroma, subcutaneous nævi, and lymphangiectasis. **Sebaceous Cyst** (Synonyms: Steatoma, Wen) is a tense, painless, cystic tumour due to the occlusion of a sebaceous follicle; sometimes associated with acne, and usually single. For its eradication the *capsule* must be entirely destroyed. **Fatty Tumours** are known by their doughy feel, lobulation, the puckered depressions seen on trying to lift up the skin over them. **Rheumatic Nodules** occur in successive crops, as small, hard, or elastic nodules, sometimes adherent to the skin, usually freely movable beneath, sometimes tender on pressure. Their favourite situation is over the fibrous tissue of the superficial bones—that is to say, chiefly around the joints and along the spine.

IV. **Epithelioma**, epithelial cancer, affects the skin in three forms, and the favourite seat of all three is the face. 1. In the *papular* form it is found as hard, glistening, pale, flat papules, which grow very slowly, become cracked, fissured, and ulcerated (*vide* Ulceration). 2. The *nodular* or deep-seated form is less frequently met with. It occurs as close-set, flat, or slightly raised, "very firm and somewhat translucent nodules. In the course of months or years it grows into a spherical or flat, hard tumour, whose surface is shining, waxy, or rosy, traversed by vessels, irregularly nodular. As the result of spontaneous retraction the centre is often drawn in like an umbilicus; the edges are steep and smooth."[1] Later, ulceration occurs. 3. Papillomatous or warty growths (*malignant papillomata*) are occasionally met with. All three may be found in the same individual, but the first is the most common and the slowest to grow. The favourite sites are the lower lip—at least 50 per cent.—the tongue, and external genitalia. The majority of cases occur in men. Lesions having these features occurring in a person past middle life should always be examined microscopically, and steps taken for their eradication.

§ **491.** Certain **rarer forms** of nodule and neoplasm also affect the skin.

V. **Leprosy** (Synonyms: Lepra, Elephantiasis Græcorum, Leontiasis Satyriasis) is a chronic constitutional disease, characterised by pigmentary, sensory, and nodular changes in the skin, due to a specific microbe affecting the skin and nerves. Leprosy used to be a widely prevalent disease, but only imported cases are now found in England. It is still endemic in Norway, Sweden, parts of Russia, Turkey, and the Turkish provinces, and in China, India, West Indies, etc. Sir Jonathan Hutchinson believes that the infection is conveyed by fish. It is communicable from man to man, though its infectivity is feeble, and probably only through an abrasion of the surface. The disease is met with in two clinical forms in its earlier stages. (*a*) *Maculo-anæsthetic* leprosy, which consists of patches of anæsthesia, sometimes of pigmentation or leucoderma, usually associated with thickening of the nerve trunk connected with the part, and a wide-spread eruption of reddish spots and patches over the body. (*b*) *Nodular* leprosy, in which are found small diffuse thickenings without much alteration of colour of the dermal tissue and

[1] Kaposi, "Diseases of the Skin," 1895.

mucous membranes. These increase to form bosses, and occurring on the face give to the patient a leonine aspect in course of time (facies leonis). The viscera are similarly involved, and wherever the granulomatous material is formed the characteristic bacillus is found, which closely resembles the bacillus of tubercle. *Mixed* forms of these two types are occasionally met with. The course of the disease is extremely prolonged, and generally fatal. Any age may be affected. It is endemic in certain countries, where want of cleanliness and hygienic principles lead to the transmission of the disease from person to person.

Treatment.—Choulmougra oil, 200 to 300 minims in capsules per diem, has certainly arrested the progress in some cases when given in gradually increasing doses.[1] Hypodermics of the same remedy or of mercury have controlled the disease in certain cases, and recently the injection of nastin has given good results.

VI. **Erythema Induratum Scrofulosorum** (Bazin's Disease) is a rare tuberculous condition, affecting chiefly young strumous women, and characterised by chronic subcutaneous nodules in the calves of the legs, which may ulcerate. They are sometimes difficult to distinguish from syphilitic gummata, but the latter are much more rapid in their progress. A *spurious form of Bazin's disease* is met with in young persons who have a feeble circulation. This leads to hypostasis of the legs, the skin of which is apt to be thickened and livid at certain spots, but the subcutaneous nodules of Bazin's disease are wanting.

VII. **Molluscum Contagiosum** consists of rounded, pearl-like elevations, varying in size from a pin's head to a pea, and semi-translucent appearance. A tiny depression is found in the centre through which the contents can be squeezed. If left alone inflammation and suppuration may occur, with spontaneous cure. The treatment consists either in snipping them off, or in squeezing out the contents, and touching the bases with silver nitrate or iodine.

VIII. **Molluscum Fibrosum** is a rare condition which consists in the formation of fibrous tissue in the deeper layers of the corium, slowly developing into tumours of varying size (up to 32 pounds), which may be sessile or pedunculated. Their favourite situation is the back. They should be removed by knife or ligature. One case under my care improved by painting with ethylate of sodium.

IX. **Sarcoma Cutis** may occur either as a primary affection or secondary to deposits elsewhere. It is met with in the form of purplish tumours of varying size of hard or spongy consistence, and a raised infiltrating margin. A small deposit with satellites around it is very characteristic. Sarcoma may develop on pigmented moles, a melanotic sarcoma being then reproduced elsewhere.

X. **Actinomycosis** is a rare chronic affection of the subcutaneous tissue, usually starting in the jaw, and spreading thence to the skin of the face and neck. It is due to the ray fungus or Actinomyces, which gives rise to a hard, slow-growing tumour, going on to ulceration, with a thin sero-purulent discharge, containing *yellow granules in which the ray fungus can be found* (§ 627). In other cases the disease attacks the lungs, the digestive tract, or the liver, causing much constitutional disturbance. The fungus enters through eating diseased grain improperly cooked. The prognosis is favourable if the disease is on the surface, where it can be dealt with surgically, or if taken early, when large doses of pot. iod. control the growth.

XI. **Framboesia** or **Yaws** is a chronic disease endemic in the tropics, rarely attacking the white population. The commonest form of eruption has a fungoid or raspberry-like character, whence its name is derived. Some observers consider the disease as identical with syphilis, modified by the climate, but it has been successfully inoculated into syphilitic patients.

XII. **Mycosis Fungoides** is a rare condition, characterised by the formation, after a long preliminary period, of reddish fungoid tumours. In the preliminary stages, which may last for months or years, there is an erythema or a scaly eczema attended by itching, followed by brownish-red papules, which leave pigmented

[1] See a case recorded by the author, Clin. Soc. Trans., about 1896.

and atrophied depressions, and are finally followed by smooth purple tumours, sessile or pedunculated, which ulcerate. The eruption usually appears on the trunk, and leads to emaciation and death. X rays have proved useful.

XIII. **Blastomycosis** is an extremely rare disease affecting chiefly the face and hands, characterised by papillomatous ulceration simulating tuberculosis cutis, and due to the fungus blastomyces.

GROUP VI. ULCERATIONS.

§ **492.** An ulcer is a loss of substance of the dermis and epidermis, exposing a granular surface, which secretes a sero-purulent fluid. Ulcers must not be confused with large vesicular or bullous lesions, such as occur in pemphigus foliaceus, in which the skin is only denuded of its cuticle. For clinical purposes ulcers may be divided into four groups :

(*a*) *Idiopathic* or *inflammatory ulcers*, caused by injury, hypostatic congestion, or varicose veins, and often aggravated by some blood dyscrasia, such as gout, anæmia, or scurvy.

In the *Treatment* of chronic ulcers the main point to remember is their absence of tendency to repair. (1) Many *local applications* have been tried. If the discharge is watery and excessive, and the granulations turgid, astringents are called for, such as zinc sulphate or lead lotion, or the painting on of nitrate of silver, 20 grains to the ounce, or the use of the solid stick, which acts also as an excellent stimulant. For the troublesome itching, carbolic lotion, 1 in 50, or 1 in 100, freely applied on lint or rag, often gives relief. Meat extracts and oxygen have been used as applications, and various protective dressings, such as strapping and starch dressings, are often successful. (2) Rest in the horizontal position is more efficacious than any other kind of treatment for ulcer of the leg, because the deficient return of the blood is one of the factors which prevent repair. With the same object a Martin's rubber bandage, a flannel roller, an elastic stocking, or a Scott's dressing is advantageous. (3) A liberal diet, and a moderate use of stimulants are often successful, combined with tonics, and among the internal remedies which I have found successful with aged persons is tr. opii, 2 to 5 minims, thrice daily. It acts by improving the tone of the cutaneous vessels. It may be combined with strychnine, which is one of the best tonics for the aged with which I am acquainted. (4) Incisions may be made through callous, indolent edges, either at right angles or parallel, to release the adhesions between them and the deep parts. Grafting by Thiersch's or some other method is, however, more successful.

(*b*) The *contagious ulcers* are hard chancre, soft chancre, the ulcer at the seat of inoculation of glanders, and certain tropical ulcers due to the poisonous stings of certain insects, and the inoculation of certain tropical diseases.

(*c*) *Neuropathic ulcers—e.g.*, perforating ulcer in tabes dorsalis.

(*d*) *Infiltrating* or *neoplastic ulcers* are due to the breaking down of some infiltration which has invaded the skin or subcutaneous tissue, and which can be detected in the tissue around—such as syphilis (rupial ulceration and breaking down of a gumma), lupus vulgaris, tuberculosis of the glands breaking down (strumous ulceration), epithelioma, rodent ulcer, and leprosy.

The differentiation of these several varieties depends largely on the history, the associated symptoms, the distribution and character of the ulcers, and these have been given under their respective titles.

An **infiltrating, ulcerating,** *and* **scarring** *eruption in a person of young or middle age is practically either* syphilis, lupus, *or* tuberculous ulceration. *If it occurs over forty or forty-five,* epithelioma *and* rodent ulcer *enter the category.*

I. **Syphilitic Ulceration**—other than the primary chancre—is of two kinds : (1) The breaking down of a large papular or lenticular syphilide *in* the skin gives rise to shallow irregular ulceration which may be covered with a scab which resembles the layers of an oyster shell (**rupia** of older authors). (2) The breaking down of a gummatous nodule which has started *beneath* the skin produces a deep punched-out ulcer. The three characteristic signs about all syphilitic ulcerations are—(1) the peripheral ring of infiltration, (2) the punched-out edge, and (3) the comparatively rapid march. The diagnosis from lupus and rodent ulceration is given in tabular form below.

TABLE OF DIAGNOSIS.

Syphilitic, Rupial, or Gummatous Ulceration.	*Ulcerating Lupus.*	*Rodent Ulcer.*
Anywhere.	Chiefly face.	Chiefly face.
Adult life.	Begins between ten and twenty years.	Over forty.
Progress rapid, destroying in weeks what others do in months.	Very slow.	Slow.
Sharp, clear-cut, punched-out, deep.	Edge rounded, sloping, and surrounded by apple-jelly nodules; superficial.	Edge rolled and *hard*; deep in later stages.
Discharge copious; offensive.	Scanty, inoffensive.	Scanty, and in later stages sanguineous.
KI and Hg efficacious.	No good.	No good.

NOTE.—A small piece may easily be removed for microscopic examination in any doubtful case.

II. **Lupus Vulgaris** may ulcerate, but only when near a mucous orifice, or subjected to injury. The nodules around are sufficiently characteristic (§ 490).

III. **Tuberculous** or **Scrofulous Ulceration** may occasionally take its origin from tuberculous deposits in the skin, but more frequently spreads from a caseating gland or strumous bone disease. The edges of the ulcer are dark purple, and undermined, never rounded and clean-cut as in syphilitic ulceration, and there are generally evidences of the strumous diathesis, or scars from past disease of the same kind. The patients are usually children, occasionally an old person (senile struma).

IV. **Rodent Ulcer** is clinically, to all intents and purposes, a variety of epithelioma in which the loss of tissue is more pronounced than the new growth; is found chiefly on the upper half of the face after forty years of age, and in the course of years will destroy the eyelids, eyeballs, and a great part of the face. It appears as a very shallow, roundish ulcer, with a flat base, scanty viscid secretion, and a very hard " rolled " edge matted to the subjacent structures. It often cicatrises in the middle, so that the ulcer becomes crescentic. It is more frequent in men. Its progress may extend over many years, but it may suddenly take on activity at any

time, and it may, as the author has seen, take on the clinical and histological characters of epithelioma in the evolution of the case.

V. **Epithelioma** is apt in course of time to undergo ulceration, but the diagnosis rests upon the characters of the initial growth, which is always found in considerable quantity around when the stage of ulceration is reached. If a small piece can be examined under the microscope the typical "bird-nest" growths of epithelioma (which are absent in rodent ulcer) can be seen.

The treatment of *neoplastic ulcers* is of four kinds: (1) Constitutional treatment appropriate to the nature of the lesion; (2) caustics and escharotics applied regularly and freely, so as to destroy the neoplasm around; (3) various operative procedures—removal, scarification, or cauterisation; and (4) the use of the X rays or cataphoresis with zinc ions. Excellent results have been obtained by the X rays in the treatment of rodent ulcer and superficial epithelioma.

GROUP VII. WARTS AND EXCRESCENCES.

§ **493.**—This group, which does not include neoplastic formations referred to in the last two groups, consists of verruca (wart), condyloma, corns, papilloma lineare, acanthosis nigricans, porokeratosis, and angiokeratoma.

Verruca, wart, or papilloma cutis, is an excrescence consisting of thickened epidermis containing elongated papillæ. Warts may occur singly, or they may be multiple. They are most frequently met with on the hands. More rarely they occur on the head, face, or genital organs. The *Cause* of warts is obscure, but they are undoubtedly in some cases contagious (Dr. J. F. Payne), and in that way spread over the hands and other parts of the body.

Varieties.—1. *V. vulgaris* occurs on the hands, and forms a horny growth the size of a small pea. 2. *V. plana*, a flat, dark-brown elevation found on the face or back of old people. 3. *V. acuminata* is moist, sessile, or pedunculated, usually quite small, but may grow to be as large as the fist, occurs chiefly on the genital organs, or where opposed surfaces are in contact, and resembles condylomata. 4. *V. seborrhœica* is an oily, fawn-coloured, slightly elevated and rounded body, in which the papillæ are mixed with inspissated sebum. 5. *V. necrogenica*, or "post-mortem wart," is a tuberculous infection of the skin which appears on the hands of doctors, post-mortem porters, leather-dressers, cooks, butchers, etc. It starts as a crimson, flat, indurated papule, which spreads, and sometimes becomes pustular, the pus drying and forming into a scab. A white and pinkish stellate cicatrix may be left behind as the disease progresses (Fig. 136). Warts are best dealt with by applications of glacial acetic acid, potassa fusa, or other caustics, or salicylic acid plaster. Small doses of mag. sulph. t.i.d. have cured some cases. *V. necrogenica* is treated like lupus.

Syphilitic Condyloma is really a papular syphilide occurring (1) on the mucous membranes; (2) near the junction of mucous membrane and skin; or (3) where opposed skin surfaces are in contact. They

very commonly occur at the angles of the mouth, and between the buttocks or labia. They are slightly raised discs of various sizes, covered with greyish epithelial or soddened epidermal flakes, and exuding a highly contagious fluid.

Corns are localised thickenings of the epidermis consequent on localised pressure. The side of the toe is a common position. They may be cured by painting with salicylic acid (20 per cent.) and collodion every night for a week ; soon afterwards the corn will flake off. *Soft corns* arise between the toes, due to hard corns becoming soddened with perspiration. Treatment consists in keeping them dry with dusting powder, such as zinc oxide and starch, keeping the toes separate with small pads of cotton wool, and by relieving pressure.

Papilloma Lineare has been described under Ichthyosis, § 476.

Acanthosis Nigricans is a rare condition characterised by progressive pigmentation of the skin, with papillary growths, terminating fatally in a few years. The

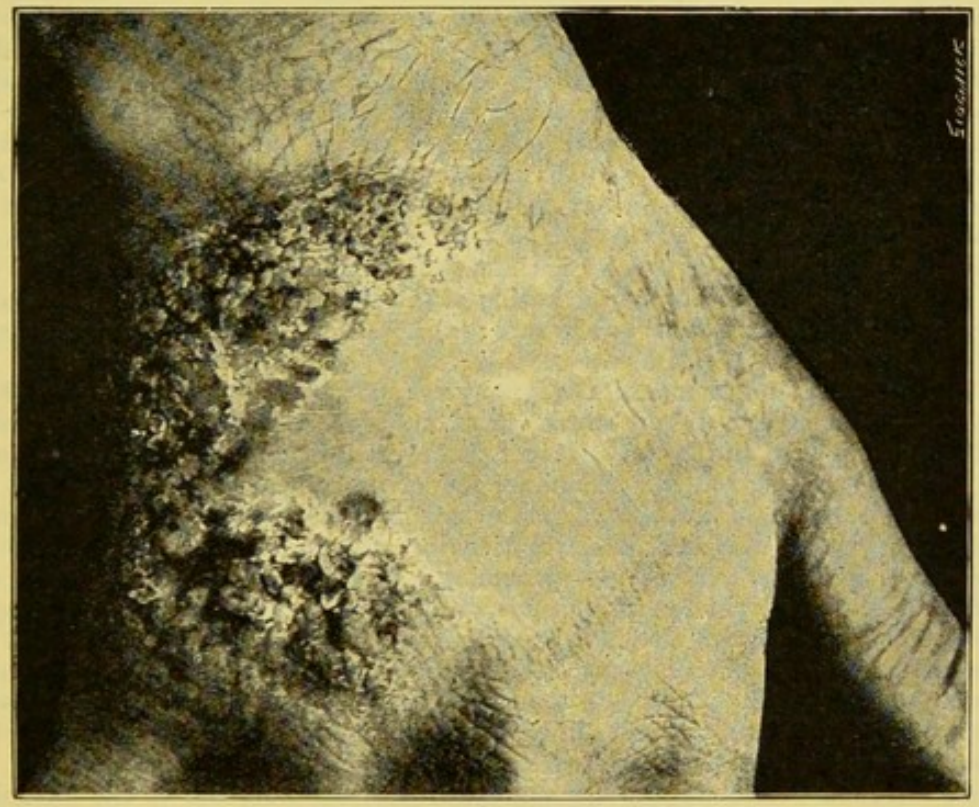

Fig. 136.—Verruca Necrogenica on the hand of a gamekeeper, aged thirty-five.

colour of the skin varies from a sallow hue to bronze and dirty brown. It is generalised, but more pronounced in the flexures. The disease may occur at any age after childhood. In most of the recorded cases it has been associated with abdominal cancer, but in others no cause has been found.

Porokeratosis, a very rare disease, occurring chiefly on the backs of the hands, is characterised by patches of atrophic skin, surrounded by a thin horny ridge or " wall " immediately inside which are seen tiny grey papules, which can be picked out. It is said to be a hyperkeratosis of the mouths of the sweat glands with destruction of glands and hair follicles.

Angiokeratoma is a rare condition consisting of telangiectases, which develop into warty growths, occurring usually after chilblains, on the backs of the fingers, toes, hands, and feet. *Treatment* consists in employing warmth and electrolysis.

GROUP VIII. ATROPHIES AND SCARS.

§ **494.** Scars, scleroderma, and atrophy of the skin may be considered together, because they not only resemble each other clinically, but fibrosis of some of the cutaneous tissues and atrophy of others occur in varying degrees in all three conditions. The disorders met with in this group are :

I. Scars. II. Atrophoderma. III. Scleroderma. IV. Keloid and its congeners Rhinoscleroma, Kraurosis Vulvæ, and Ainhum.

I. **Scars** may result from burns, wounds, or infiltrating or suppurating eruptions in which there has been a loss of substance. If much deformity or loss of mobility results, plastic operations are called for ; but it is wonderful how much can be done in young patients by means of persevering massage with oleaginous substances, and especially, in my experience, cod-liver oil. Scars are liable occasionally to be affected by keloid (see below).

II. **Atrophoderma** (Atrophy of the Skin) occurs as : (*a*) Atrophy of the entire cutaneous covering is common in old age ; (*b*) Lineæ albicantes is a term applied to the atrophic streaks found on the abdomen and breasts after pregnancy, over the hips and other parts when the patient has been getting rapidly stouter. A case of linear atrophoderma due to neuritis after enteric fever is recorded by Sir Dyce Duckworth.[1] The author and others have also observed cases which could be traced to a neuropathic cause. (*c*) *Unilateral atrophy* of the skin is met with in the condition known as Hemiatrophy Facialis (§ 617), which is of nerve origin.

III. **Scleroderma**, or fibrous thickening of the skin, is met with in three clinical forms, all of which are more or less rare : (*a*) Localised (or morphœa) ; (*b*) diffuse ; (*c*) S. neonatorum.

(*a*) Localised Scleroderma (Synonyms : Morphœa of Erasmus Wilson, Addison's Keloid) is a disease consisting of one or more localised ivory patches of sclerosed skin with, in the earlier stages, a congested lilac border. The patch may be atrophic and pigmented. There are few or no subjective sensations, but the tactile sensation is diminished. Some cases undergo spontaneous resolution in course of years. The favourite situations are the face, neck, and beneath the breast. There is a tendency to symmetry. It is thought that the shape and distribution of some patches corresponds with the distribution of a nerve, the supra-orbital being a common site, but it seems more probable to the author that the lesions are associated in some way with Head's sensory and visceral areas. Females are more prone to be affected in the proportion of three to one, and the disease appears mostly in the first half of life. Beyond the disfigurement and contraction the patient suffers but little inconvenience from this variety of the malady.

(*b*) Diffuse Scleroderma is a somewhat different affection to the foregoing, and consists of a parchment-like thickening and contraction of the skin, starting and predominating in the extremities. It progressively increases until the parts become completely hidebound and immobile. The face in such cases wears a smooth, expressionless aspect. This disease is one of much gravity. By degrees fissures and ulcers form, and some of the fingers may become gangrenous, and death from some intercurrent malady occurs. Many degrees of severity are met with, and in some the condition only produces a constant liability to cold and to various superadded skin lesions.

(*c*) Scleroderma (Synonym : Sclerema) Neonatorum is a different disease. It appears congenitally, and is generally fatal in the course of a few weeks. The affected skin is bound down to the parts beneath in livid, tense, shining patches, which tend to become universal.

The *Treatment* of the three conditions is not satisfactory. Local massage may be recommended. Several slighter degrees of generalised scleroderma under my care

[1] *Brit. Journ. Dermat.*, No. 62, vol. v.

improved under thyroid. The constant current and fibrolysin have done good in the localised form.

IV. **Keloid** consists of a fibromatous deposit in the skin occurring primarily in unaffected skin, or secondly in old cicatrices. The lesion appears as a small firm nodule, of a crimson or pinkish colour, which slowly enlarges by means of tentacle-like processes. At first it is raised above the skin level. Such growths if excised immediately recur, and in that sense are malignant. The negative pole of a mild constant current has been attended with favourable results. Fibrolysin injections are useful.

Acne Keloid (Synonyms: Dermatitis Papillaris Capillitii, Sycosis Capillitii) is a rare disease which occurs on the nape of the neck, a slow pustular affection, resulting in keloid formation.

Rhinoscleroma is a chronic inflammatory affection characterised by the development of hard, circumscribed, nodular growths in the skin and mucous membrane, most commonly of the nose and naso-pharynx, due, it is believed, to inoculation with a specific bacillus. The bones and cartilage may be involved.

Kraurosis Vuvlæ is an atrophy and contraction of the mucous membrane of the external genitals and adjacent perinæum of women. The tissues are atrophied, with thickened patches.

Ainhum consists of a slow strangulation and amputation of one or more toes by the growth of a constricting band of hypertrophied skin. It is not seen in this country.

GROUP IX. PIGMENTARY AND VASCULAR ALTERATIONS.

§ **495.** Alterations of colour depend mainly upon the condition of the vessels and the amount of pigment in the skin. A *diminution* of pigment is not frequent, and occurs only in two conditions: (1) Albinism, a congenital condition in which there is deficient pigment in the skin and its appendages, and in the iris and choroid; and (2) Leucoderma (Synonym: Vitiligo), a condition in which there is an absence of normal pigment in areas which are surrounded by darker-coloured skin. The transition from the pale to the dark area is abrupt, and it is the dark-coloured margins which attract the notice of the patient. It may be congenital or acquired, and is probably neurotrophic in its origin.

a. A *localised* increase of pigment occurs in:

I. Chloasma.
II. Lentigo.
III. Pityriasis versicolor.
IV. Pigmentary and vascular moles.
V. Purpura.
VI. Urticaria pigmentosa.
VII. Xeroderma pigmentosa.
VIII. Xanthoma.
IX. Morphœa alba and nigra.
X. Leprosy.

b. A *generalised* increase of pigment occurs in (1) arsenical and silver pigmentation; (2) Addison's disease; (3) abdominal cancer; (4) cardio-vascular disease; (5) bronzed diabetes; (6) constipation; (7) melanotic sarcoma; and (8) acanthosis nigricans; but in these the pigmentation is subordinate to other symptoms.

I. **Chloasma** occurs in single or multiple patches of diffuse discoloration on various parts of the body, varying in shade from a light yellow to a deep brown. Several varieties may be referred to: 1. *Chloasma symptomatica* is met with most frequently in pregnancy or uterine disease, and its most usual position is on the face and round the nipples. 2. *Chloasma cachecticorum* occurs in association with

malaria, cancer, senile atrophy, rheumatoid arthritis, abdominal tubercle, or cancer, and exophthalmic goitre. 3. *Chloasma traumatica* is the pigmentation beneath the garters, or around the waist in tight-lacing women, in pediculosis or scratching, and after sinapisms, blisters, etc. In this category may be included the pigmentation which follows chronic eczema, syphilis, psoriasis, or any other long-continued afflux in the skin capillaries. 4. *Chloasma caloricum* is the pigmentation due to sun and wind, or to heat, as on the shins of women who sit over the fire.

II. **Lentigo** (Synonyms: Freckles, Ephelides).—Freckles are multiple, circumscribed pigment spots on the portions of the body exposed to light. In advanced age they may occur anywhere, and are apt to become malignant by taking on an epitheliomatous growth.

III. **Tinea Versicolor** (Synonym: Pityriasis Versicolor) is a vegetable parasitic affection of the skin, which appears as variously sized, irregularly shaped, dry, highly furfuraceous patches, yellowish in colour, found generally upon the trunk, and especially in the hollow of the breast bone, due to a specific parasitic fungus, the microsporon furfur (Fig. 137).

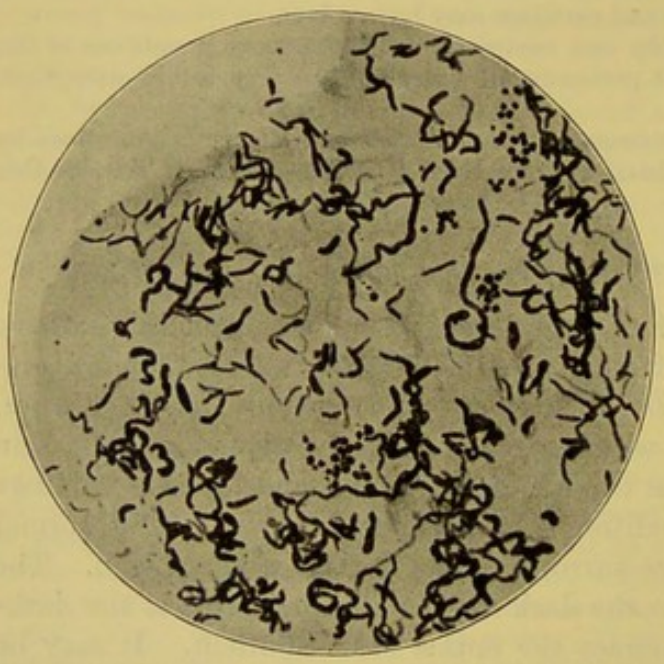

Fig. 137.—MICROSPORON FURFUR, the fungus of PITYRIASIS VERSICOLOR, × about 50.—Shows the branching irregular mycelium and the constellations of spores. Stained by Gram's method.

IV. **Pigmented and Vascular Moles** are distinguished from other pigmented conditions by their being congenital, and being more or less raised. Nævus spilus is a smooth discoloured spot of otherwise healthy skin. Nævus verrucosus is rough, and sometimes bristling with hairs (nævus pilosus). *Vascular moles* are purple spots of increased vascularity, usually a little raised above the surface, varying widely in size and thickness. They are sometimes stationary, but more often gradually increase in size. *Telangiectasis* is a localised dilatation of the vessels of the skin, and is a frequent accompaniment of acne rosacea. Small spots are met with independently of any skin affection on the face and various parts of the body in healthy persons, particularly as life advances, or when the peripheral circulation is feeble. *Port-wine mark* is a venous and capillary dilatation over an area of skin.

Treatment.—Leucoderma, lentigo, and chloasma are best treated with strong mercurial lotions (½ to 1 per cent.), but they are difficult to cure. For pityriasis versicolor thorough cleansing with a hard brush and soap is the essential treatment, together with ung. sulph. or a lotion of sod. hyposulph. (1 drachm to the ounce). Electrolysis, escharotics, or excision give the best results in pigmented and vascular nævi.

§ **496.** V. **Purpura** consists of dark, abrupt-edged purple spots due to extravasations of blood into the skin. The eruption does not fade on pressure. It is sometimes accompanied by similar extravasations into the mucous membranes and internal organs, and hæmorrhages from the mucous surfaces. The constitutional symptoms vary considerably, and may be absent. Pyrexia, usually slight, occurs in about half the cases.

The *Causes* of purpura are but little understood, but may be grouped under three

headings : 1. *Local* and *mechanical* causes, such as *heart disease* and *old age*. The most frequent illustration of this is met with in eczema of the leg (indeed, most eruptions on the legs of old people), which assumes a purpuric character on account of the hypostasis of the blood. Mitral disease is associated not infrequently with purpuric eruptions on the legs. 2. Purpura is associated with certain *nervous* conditions, such as cerebro-spinal meningitis (almost always), tabes, myelitis, hysteria, and, according to some, neuralgia. 3. *Changes in the blood* produce purpura in (i.) various *fevers*, especially typhus (typhus has been called purpura contagiosa) and cerebro-spinal fever, in which a purpuric eruption is constant ; measles sometimes ; malignant endocarditis, and pyæmia occasionally ; the initial stages of small-pox, and in the *malignant type* of that and most of the other acute specific fevers ; (ii.) certain *drugs* occasionally, especially mercury and iodide, quinine, copaiba, belladonna, ergot, salicylic acid, and the mineral acids ; (iii.) *autotoxic* conditions, such as Bright's disease, hepatic disease, including cirrhosis, acute atrophy, carcinoma, and any aggravated jaundice ; bad food also seems in some cases to account for the onset of purpura ; and (iv.) *constitutional* diseases, such as leukæmia, scurvy, lymphadenoma, and great debility from any cause.

Four special *varieties* of purpura are recognised. (*a*) *Purpura Simplex* (morbus maculosus of Werlhof) is the name given to a mild attack of purpura for which no cause can be assigned. It is usually met with in young persons, and runs a benign course. (*b*) *P. Hæmorrhagica* is a severer variety, which may start as P. Simplex, or independently. The spots are larger, and sometimes raised or œdematous. Hæmorrhage occurs from the mucous membranes, and pyrexia is more marked. (*c*) *P. Rheumatica* (Peliosis Rheumatica, Schönlein's disease), and *Henoch's Purpura*, both of which are described elsewhere. (*d*) Purpura and erythema are related, the first being due to an exudation of all the constituents of the blood, the second to an exudation of serum. Consequently it is not surprising to find that many intervening stages of the two are met with, and are known as *purpuric erythema*.

The *Diagnosis* of purpura is easy, but difficulty lies in ascertaining its cause. The differentiation of *scurvy* has been given (§ 410). To ascertain the cause carefully consider the previous history, and thoroughly examine every organ. If no cause can be found, the disease is called P. idiopathica, simplex, or hæmorrhagica.

The *Prognosis* is extremely grave when associated with the specific fevers, or with a high temperature. P. simplex usually results in recovery in a few weeks ; p. rheumatica is rarely fatal, though it may last for months or years, and may recur ; Henoch's purpura is favourable as regards recovery, but is apt to recur.

The *Treatment* is unsatisfactory ; arsenic, iron, ergot and ol. terebinth deserve trial.

VI. **Urticaria Pigmentosa** (described by Nettleship in 1869, and so named subsequently by Sangster) is a chronic or recurrent urticaria, in which each crop of wheals leaves behind spots of brown pigmentation of considerable persistency. The disease starts in early childhood ; it may cease spontaneously about puberty, but it more frequently lasts for many years. The *Treatment* is like that of urticaria, plus calcium chloride and bromides.

VII. **Xeroderma Pigmentosa** (Synonym : Kaposi's Disease) is a very rare disease of a chronic progressive character starting in early childhood, often in members of the same family, and marked by small dark freckles, with atrophy and contraction of the skin between them, and the occurrence of telangiectases. There is also a distinct tendency to a malignant new growth, both in the skin and the internal organs in the shape of a malignant sarcomatous or cancerous infiltration. The distribution is universal, and the contraction gives rise to eversion of the eyelids and other orifices. It usually terminates in death before the age of twenty-two.

VIII. **Xanthoma** (Synonym : Xanthelasma) is a rare condition most commonly occurring in the face in cases of diabetes and chronic jaundice. It consists of yellowish nodular deposits in and beneath the cutis, varying in size from a millet-seed to a bean, or larger.

IX. **Morphœa Nigra** and **Morphœa Alba** are names employed by Erasmus Wilson for the disease which we now describe as localised scleroderma (§ 494), when attended by excess or deficiency of pigment.

X. **Leprosy** (§ 491).—Patches of pigment and white spots may occur in the early stage of anæsthetic leprosy, and dark spots occur, especially on the face, in the early stage of nodular leprosy.

GROUP X. DISORDERS OF THE SWEAT.

§ **497.** Four disorders of sweat are met with: Anidrosis, hyperidrosis, bromidrosis, and chromidrosis.

I. **Anidrosis** is not common apart from the conditions mentioned in Group VIII.

II. **Hyperidrosis** is the term applied to an excessive secretion of the perspiration, and may be general or localised. When general it may be due to a lowered neuro-vascular tone, excitement, corpulency, or the use of stimulating foods and drinks. It also occurs in fevers at the crisis, in ague, acute rheumatism, chronic tuberculosis. The *localised* form affects most often the feet, axillæ, and palms. The sweat mixed with sebaceous secretion decomposes on the clothing, and gives rise to a pungent and disagreeable odour. The feet are apt to become tender, and eczema may supervene.

The *Treatment* of hyperidrosis consists of the application locally of a mixture of tr. belladonna and water equal parts, or sponging with vinegar and water are useful, and internally hypodermics of atropin, or the administration of arsenic, and the mineral acids or tonics. *Local* hyperidrosis, especially of the feet, is troublesome to get rid of. The stockings should be changed several times a day, and put into a saturated solution of boracic acid before being used again. Dusting powders relieve the slighter forms. Applications of tannic acid, chromic acid, salicylic acid, and of diachylon plaster should be tried in graver cases.

III. **Bromidrosis** is the term applied to a disturbance in the function of the sweat glands in which the perspiration has an offensive odour. Mere excess of perspiration in the feet or axillæ may render the person disagreeable to his companions. The subject is therefore interwoven with hyperidrosis, to which reference should be made for the treatment.

IV. **Chromidrosis** is a rare disturbance in the function of the sweat glands in which the perspiration is coloured.

GROUP XI. DISEASES OF THE SCALP AND HAIR.

§ **498.** The diseases special to the scalp and hair are:

I. Ringworm.
II. Favus.
III. Alopecia.
IV. Seborrhœa.
V. Canities.
VI. Pediculi Capitis.
VII. Hirsuties.
VIII. Trichoptylosis.
IX. Trichorrexis Nodosa.
X. Leptothrix.
XI. Tinea Imbricata.

I. **Ringworm** (Synonyms: Trichophytosis Capitis, Tinea Tonsurans) starts as an insignificant, semi-bald, pink patch, usually overlooked, and when first seen by the physician is a white, powdery, circular patch on the scalp of children, with *broken hairs*. The spot varies from the size of a threepenny piece to one denuding half the scalp. *Kerion* is a name given to a boggy suppurating patch on the head caused by one variety of ringworm.

Diagnosis.—The broken hair stumps are quite characteristic. Dabbing chloroform over the part will reveal the diseased hairs, which then look whitened like hoar frost. The diagnosis should be clinched by placing the hair on a slide with a drop of liquor potassæ, and examining under the microscope. The varieties of the fungus, however, can only be distinguished after staining by Gram's method (§ 627). A bald form of ringworm occasionally occurs resembling *alopecia areata*, but some broken stumps of hairs can be found at the margins of the patches. *Seborrhœa* has no broken hairs nor ringworm fungus.

Etiology.—The disease is rare in children over fourteen, but common under ten. Fair-haired children are more susceptible. Adults are practically immune. Animals, especially domestic animals, may contract ringworm and give it to children. The disease spreads rapidly in families and schools, and is due to a specific fungus. Two varieties

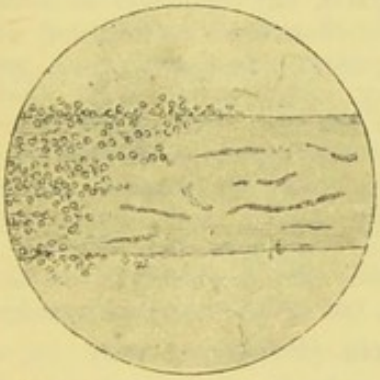

Fig. 138.—Small Spored Ringworm (Microsporon Audouïni) under a ⅛-inch objective.—Spores forming a thick-set sheath outside hair; mycelium within the hair branching and irregular, not parallel.

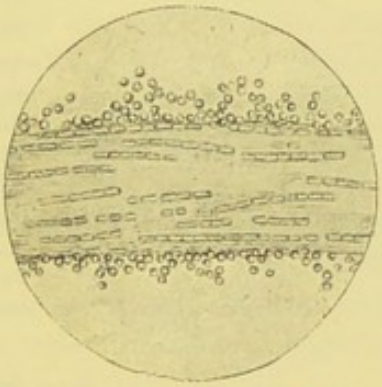

Fig. 139.—Large Spored Ringworm (Megalosporon).—Mycelium within the hair parallel and breaking into equal segments with truncated ends.

of the fungus cause the disease—small spore (Microsporon Audouïni) and large spore (Megalosporon) (see Figs. 138 and 139).

Prognosis.—The disease lasts an indefinite time, but tends to disappear spontaneously about puberty. Much depends upon the stage at which the disease is first seen, the diligence of the treatment, and the variety of spore present, the small spore being much more intractable. Fair-haired children are more difficult to cure. The average duration is two or three years.

Treatment.—Ringworm is a most difficult malady to cure, and great perseverance is required. The head should be shaved every ten days, and a linen cap worn which can be renewed every two or three days, the old one being burned. Patches seen very early may be aborted with strong parasiticides—*e.g.*, pure carbolic acid, iodine, or croton-oil. Every day (1) cleanse and remove all débris with A.C.E. mixture; (2) apply parasiticides. Air and water should, as far as possible, be

excluded. Amongst the numerous remedies may be mentioned salicylic acid (10 grains to the ounce of collodion), chrysarobin, carbolic, mercury, and oil of cade in varying strengths; and it is well to change the remedies at times.

It is often very difficult to decide when a child is free from infection. My own method is to leave the case which appears cured without treatment and untouched for ten days. If at the end of that time the surface is free from scaliness, the hairs are growing normally, and the microscope gives negative results, I recommend further treatment for a week, and then, after a second interval of ten days, if the same tests answer, I pronounce the case as probably cured. When the services of a dermatologist skilled in the use of the X rays can be obtained, the quickest and best method of curing ringworm is to epilate the hair by the Sabouraud method of X-ray administration.

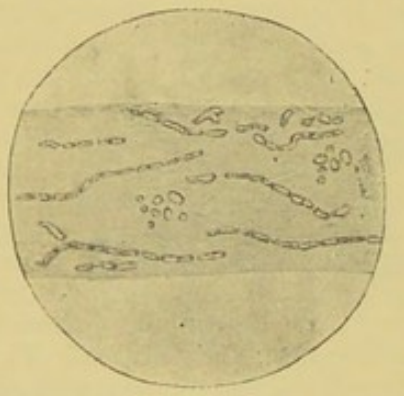

Fig. 140.—Favus Fungus (tinea favosa or achorion Schönleinii) magnified about 300.—Groups of irregular-sized spores within the hair; mycelium irregular in thickness and arrangement.

II. **Favus** occurs on the head and the body. It is rare in England, but commoner in Scotland. It forms such characteristic irregular yellow crusts, with yellow, cup-shaped tops, and is accompanied by such a mousy smell, that the diagnosis is not difficult. The microscope reveals the spores and the mycelium of the achorion Schönleinii (Fig. 140). It develops slowly, is accompanied by itching, and leaves atrophic scars. It is less contagious than ringworm, but more intractable. The treatment is the same as in that disease, but epilation is more necessary.

III. **Alopecia** (Baldness) may be congenital or acquired, partial or complete, diffuse or in patches. The acquired condition may arise from (1) *premature senility*, which usually begins on the vertex; (2) *general malnutrition*, when the hairfall is diffuse, as in tuberculosis, acute fevers, anæmia, and nervous exhaustion; (3) *syphilis*, in which the baldness is disseminated or patchy in the early stages, or localised in the later stages of the disease (being then due to syphilitic lesions of the skin); (4) *favus and ringworm*, which affect the hairs and lead usually only to temporary and localised baldness; (5) *lupus erythematosus*, in which the bald patches are permanent; (6) impetigo, eczema, X rays, etc., with temporary loss of hair; (7) *seborrhœa capitis*, which is probably the commonest cause of both diffuse and patchy baldness; (8) two forms of cicatricial alopecia are met with rarely—pseudo-pelade of Brocq, and folliculitis decalvans.

Alopecia Areata is a special form of baldness occurring in circular patches which are smooth and white. Each patch slowly increases peripherally, and at the margin short diseased hairs may be seen, which have so characteristic an appearance as to enable us at once to identify the disease. The free end is of a normal thickness, but presents a ragged

fracture where the hair has been broken off ; from this point the shaft gradually becomes thinner towards the root, which is extremely atrophied. Thus it somewhat resembles a note of exclamation (!). Any part of the body may be affected. The disease runs a very protracted course, lasting, if untreated, for years. In course of time a few downy hairs begin to grow, white at first, but gradually becoming coloured. It is undecided whether the disease is of parasitic or neurotic origin.

In the *Treatment* of baldness attend to the general health, and apply local remedies in order to stimulate the vascularity of the part by means of lotions or ointments, commencing with ammonia, turpentine, and cantharides. If seborrhœa is present, it must be corrected (see below). The treatment of the bald scars left by lupus erythematosus and other scarring eruptions of the scalp is hopeless, for the hair-bulbs are destroyed. In alopecia areata the patient should be assured that nothing but a prolonged course of treatment is necessary. Galvanism, high-frequency currents, and massage are useful.

IV. **Seborrhœa Capitis Sicca** is the name usually given to cases of **pityriasis Sicca** (dandruff) occurring in localised patches or generalised over the scalp. It is due to the bottle bacillus. It may be accompanied by a degree of inflammation due to the staphylococcus griseus, and is then known as seborrhœic dermatitis (Sabouraud). It is diagnosed from eczema by the greater inflammation and exudation in the latter ; and from psoriasis by the larger number and size of scales in psoriasis. An abnormally greasy scalp is found with seborrhœa oleosa, which is due to, or at least associated with, the microbacillus of Sabouraud. Both pityriasis and seborrhœa oleosa lead to diffuse hairfall.

The *Treatment* consists in washing the head once or twice a week with equal parts of soft soap and spirits of wine, and in rubbing in every night a lotion or ointment containing mercury, tar, or sulphur.

V. **Canities,** or whiteness of the hair, is, as the name implies, most usually an evidence of advancing years, or of overwork, sudden or prolonged grief, or neuralgia. The *Treatment* is unsatisfactory apart from the improvement of the general health by tonics. The head should be examined for seborrhœa, pityriasis, or any other local disease, which, though it does not cause, may expedite the loss of colour.

VI. **Pediculosis Capitis** presents the following features: (1) The pediculi (Fig. 141); (2) white specks on the hairs (the eggs or " nits "), which cannot be pulled off, by which they are distinguished from dandruff (Fig. 142) ; and (3) irritation and inflammation. If the condition is untreated there results pustulation with formation of thick crusts, matting of the hair, and enlargement of the occipital glands. Kill the living parasite and also the nits. This may be done by soaking the hair with methylated spirit or kerosene for one or two nights, and combing the hair thoroughly with a small-toothed comb. Ungt. hyd. amm. may be rubbed in. The nits may be dissolved by washing the hair with strong vinegar.

VII. **Hypertrichosis** (Synonym : Hirsuties) is a growth of hair either abnormal in amount or in position, and the most troublesome of these is the moustache or beard found on the faces of some women. *Treatment* consists of the removal

either by the razor or electrolysis. Depilatory pastes of barium or calcium do not prevent regrowth. The X rays in skilled hands are useful.

VIII. **Trichoptylosis,** or splitting of the ends of the hairs, is met with sometimes in women. It often occurs in association with deranged general health.

IX. **Trichorrexis Nodosa** is a very rare disease in which a series of spindle-shaped swellings appear upon the hair, which break transversely, and leave a brush-like extremity.

X. **Leptothrix** (Synonyms : Mycosis Axillaris, Trichomycosis Nodosa) is a disease affecting the hairs of the axillæ and scrotum. The hairs are dry and knotty, due to adherent small concretions, which may affect the whole length of the hair, but not the follicle, or may occur as separate nodules on a hair. Bacilli are found in these concretions, and the hair may be split longitudinally (Fig. 143).

XI. **Tinea Imbricata** is a contagious disorder of the tropics due to a fungus affecting chiefly the axillæ.

§ **499. General Remarks on the Treatment of Skin Diseases.**—There are three principles upon which we must depend for success: (1) If we except mercury and iodides in syphilis, and perhaps arsenic in lichen planus, pemphigus, and dermatitis herpetiformis, there are no such things as specific remedies in skin diseases. It is therefore not sufficient for purposes of treatment to diagnose a case as eczema,

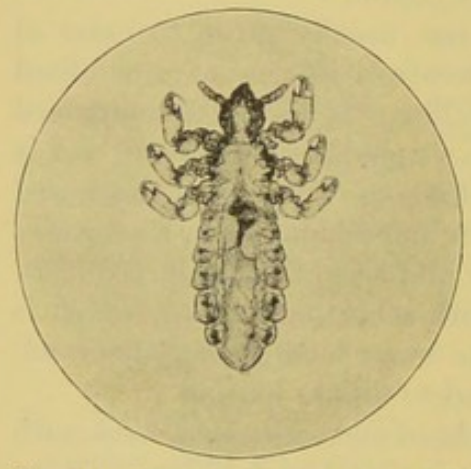

Fig. 141.—Pediculus Capitis × 10.—It differs from the pediculus corporis only in being shorter, and in its thorax and abdomen being more nearly equal in size (see p. 672).

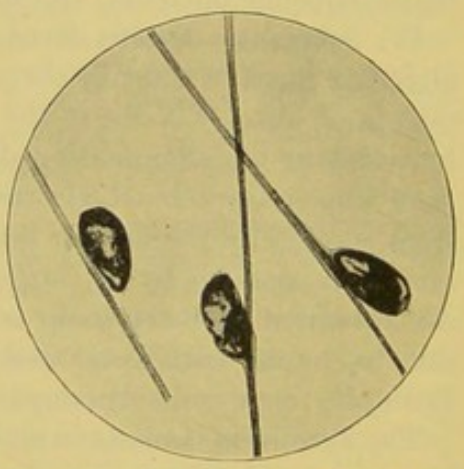

Fig. 142.—Nits (eggs) of Pediculus Capitis on hairs, slightly magnified.

psoriasis, lupus, etc. We must recognise *the stage of the disease* and the *precise pathological process* before us. An ointment which would cure a chronic eczema would greatly aggravate an acute weeping one. It follows, therefore, that it depends not so much upon the name which we decide to give to an eruption, as upon the amount of congestion, swelling, scaling, thickening, discharge, itching, etc., which is present. We have, in a word, to treat the symptoms, the sum of which constitutes the disease at that particular moment. This requires very considerable experience, and herein lies one of the justifications of the speciality of dermatology. (2) The *method* of application of a remedy is of quite as much importance as the composition of it. (3) The *idiosyncrasy* of a patient and the susceptibility of his skin to various remedies must always be remembered. This is especially true of the face, for what will benefit one person's skin will irritate another's.

Bearing in mind these three important considerations, it may be convenient to divide external remedies into three groups, the first being chiefly indicated for acute, congestive, or moist conditions, the second for dry, scaly, hard, and chronic conditions, and the third group (caustics) having a corrosive action for the removal of diseased structures.

(*a*) Sedatives and Astringents are used chiefly in acute conditions to reduce hyperæmia, to check exudation, and to allay pain. In order of increasing strength the principal ones are zinc, lead, bismuth, mercurous, and, in a less degree, mercuric

and silver salts. Zinc and bismuth are inert on the unbroken skin. These remedies are generally applied in the form of ointments,as a powder, or, still better, suspended *in lotions* for acute eczema and similar moist conditions. Zinc oleate is also a bland preparation, and zinc gelatine may be used when there is little discharge. Ung. diachyli is also soothing. Mercurial preparations are more efficacious in chronic conditions. The perchloride is irritating, and if used strong comes under the heading of caustics. Vegetable astringents are not much used, though the glycerine of tannic acid may be used for congestive conditions around the mucous orifices.

(*b*) Stimulating Applications, Antiseptics, Tars, and Aromatics are useful to stimulate chronic conditions to more healthy action. They include wood tar or creosote, and coal tar or creolin, carbolic acid, resorcin, thymol, β-naphthol, benzoic and salicylic acids—salicylic acid especially being a very useful preparation to reduce hyperplasia of the epidermis—ichthyol, sulphur, and chrysarobin. The earlier named of these are less irritating than the later ones ; indeed, weak preparations of tar may act as a sedative.

(*c*) Caustics have a definite corrosive action upon the skin. Thus liquor potassæ may be used to remove the superfluous epidermal scales of psoriasis prior

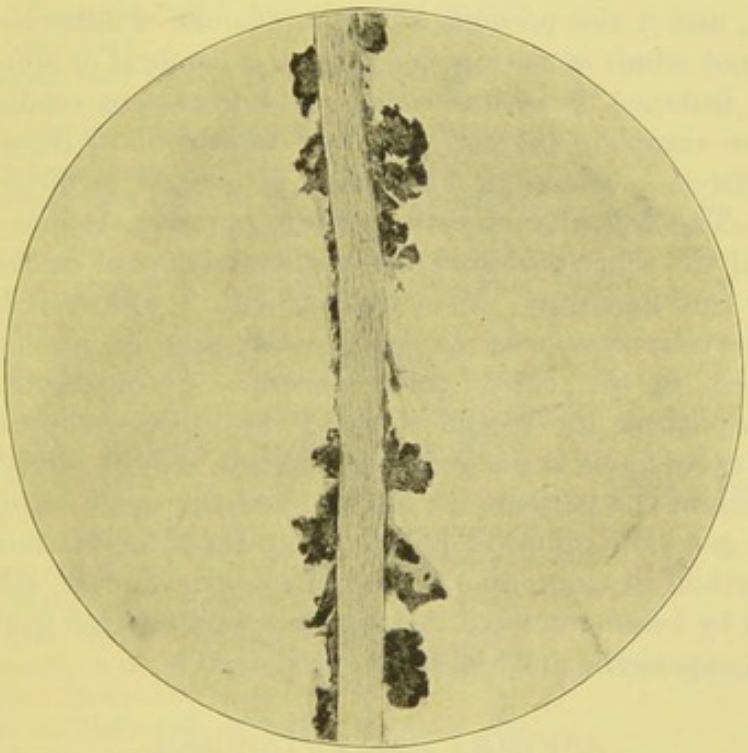

Fig. 143.—Leptothrix, slightly magnified.

to the application of an ointment or plaster. Others are mercuric chloride, acid nitrate of mercury, nitric acid, pure carbolic acid, etc. Soft soap is the mildest of such preparations, and may be usefully added to an ointment for the treatment of inveterate psoriasis.

(*d*) Protective Measures.—The old-fashioned paste—*i.e.*, an ointment made up with a large proportion of some powder (*e.g.*, F. 75)—comes under this heading. Pastes are applied by laying on in a thick layer. One of the most useful forms of protection is zinc gelatine. Painted on to the diseased skin this fulfils four indications : (i.) Gentle compression and support, as in varicose eczema or hypostatic congestion ; (ii.) protection from the action of the air or friction while allowing natural evaporation and healthy action to go on beneath ; (iii.) it allays itching ; (iv.) is an excellent dressing and means of applying remedial agents constantly. Another protective agent is the plaster mull introduced by Unna (Hamburg), which consists of reagents mixed with gutta-percha and some basis, and spread upon a piece of muslin.

CHAPTER XIX

THE NERVOUS SYSTEM

THE nervous system is the governing and controlling power of the body, and permeates every other system and structure. As a consequence the investigation of its diseases necessarily requires a wide knowledge, and it also presents a certain amount of difficulty. Neurology does not admit of narrow specialism; a cerebral or spinal hæmorrhage, for instance, is in its etiology and treatment cardio-vascular, and for the complete investigation and treatment of diseases of the nervous system a thorough knowledge of general medicine is indispensable. The difficulty of investigation, however, is more apparent than real if the scheme of studying medicine adopted in this work be followed. Our first duty, when dealing with a widespread structure like the nervous system is to *localise* the seat of mischief: Is it, for instance, in the brain, cord, nerves, or sympathetic system? This accomplished, the second stage of the process, the diagnosis of the *nature of the lesion* is not generally difficult, for the position it occupies, the age of the patient, his history, and the mode of onset of the illness will generally afford us fairly certain bases for decision. A convenient method of examining a case of nervous disease will be given below, and by bearing in mind the two steps just referred to the student should not experience much difficulty in diagnosis.

ANATOMY AND PHYSIOLOGY.

It follows from what has just been said as to the necessity of identifying the *locality* of a nervous disease before we can diagnose its *nature*, that an accurate knowledge of the anatomy and physiology of the nervous system is essential. Anatomically the nervous system consists of Encephalon (Cerebrum, Cerebellum, Pons Varolii, and Medulla), Spinal Cord, Peripheral Nerves, and Sympathetic System. But such a division into localities is purely artificial, because the cells and their processes connect these parts together histologically, physiologically, and pathologically by an endless series of intercommunicating cells and fibres.

§ **500. The Neuron.**—Histologically, physiologically, and pathologically the nervous system consists of a collection of cells which with their processes are called *neurons*. The nerve-cells are called the *neuron-bodies*, and the cell processes, which

when elongated form the nerve fibres (or nerve fibrils), are sometimes spoken of as *dendrons*, their ultimate branching processes being called *dendrites* (Fig. 144). They are supported in connective tissue known as neuroglia. One of the dendrons is longer and larger than the others, and receives a special covering in the shape of a medullary sheath after leaving the neuron-body or cell ; this process is called the axis-cylinder process or *neuraxon*. The neuron-bodies or cells are found entirely in the *grey matter* of the brain and cord, or in the various ganglia, such as those of the posterior roots or sympathetic system. The neuraxons in passing to their destinations are often of considerable length ; they pass through and constitute the fibres or *white matter* in the brain and cord, where they form the nerve fibres, and finally traverse the *nerve trunks* to their destination in the muscles, skin, special senses, or elsewhere. The words " ganglion cell," " multipolar cell," " neuron cell," and " neuron-body " are also synonymous.

The different cells at one time were thought to be connected with each other by means of the branching ends (dendrites) of their processes. Doubt has recently been thrown on this, but whether there is organic connection between the different neurons or not, their dendrites interlace and lie side by side, and a nerve impulse can pass from one to another, either directly or after the manner of an electrical inductive process.

Ascending and Descending Degeneration. — A nerve *cell* depends for its nutrition on the quality of the surrounding matrix, just as the nutrition of a tree is modified by the soil surrounding its roots. A *nerve-fibre* appears to depend mainly on its originating nerve-cell for its nutrition. When a nerve-fibre is divided (or injured) the part on the side farthest from the cell of origin rapidly degenerates (Wallerian degeneration). It is evident, therefore, that the cell body has a powerful control over the nutrition of the nerve-fibre to which it gives origin. This nutritional dependence of the nerve-fibres on their cells of origin is one of the leading facts in neuropathology, and it accounts for those *descending* (motor) and *ascending* (sensory) degenerations which we shall meet with so frequently (Figs. 149 and 150). Moreover, by means of the artificial division of nerve-tracts histologists have been enabled to trace the course of nerve-fibres in the brain and cord which would have been impossible of dissection.

Waldeyer's neuron scheme of the nervous system as above depicted is not regarded by everyone as the most scientific, but for clinical purposes it is the most lucid and practical existing at the present time.

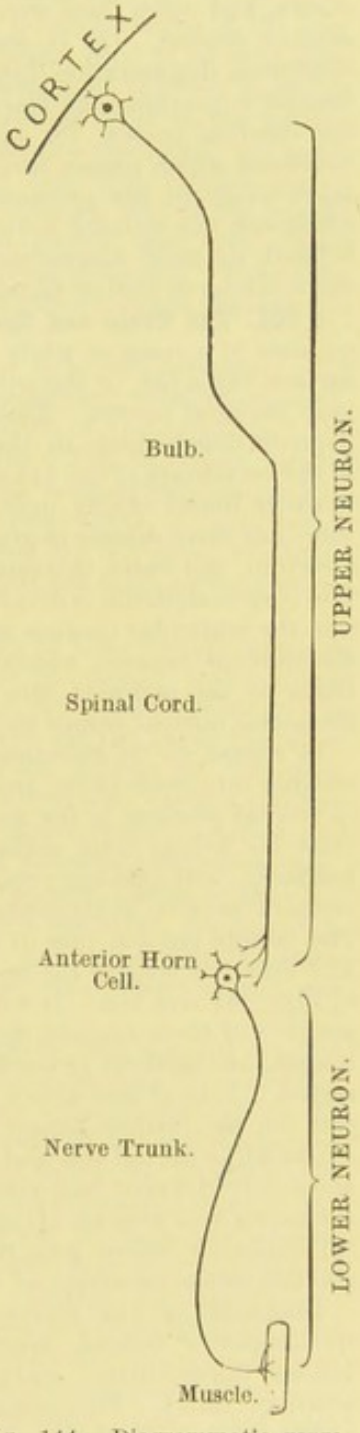

Fig. 144.—Diagrammatic representation of a MOTOR NEURON, the upper level (UPPER MOTOR NEURON) being from brain to spinal cord, the lower level (LOWER MOTOR NEURON) being from anterior horn to muscle. Compare Figs. 149 and 163—*i.e.*, Motor Tract and Path of Tabes.

The functions of the intimate structure of the different parts of the nervous system and the paths or tracts along which the impulses are transmitted, have been learned in three ways : (1) By laborious dissection relatively little information was

gained, though by microscopic examination, aided by differential staining, important paths were revealed. (2) By experiments on animals, Hitzig, Ferrier, Horsley, and others discovered the functions of important parts of the cerebral cortex, and other facts were discovered, upon which the brilliant results of craniectomy depend. (3) It was, however, by the application of the principle of Wallerian degeneration just enunciated that the greatest information has been learned concerning the tracts in the cord and elsewhere. When a motor cell in the cerebral cortex, for instance, is severed by injury or disease from the long neuraxon which passes from it down the spinal cord, a descending degeneration takes place in the pyramidal tracts of the spinal cord (Fig. 150). Conversely whenever the sensory nerve-cells in a posterior root-ganglion, for instance, are injured, or their connections severed, an ascending degeneration takes place up the columns of Goll or Gowers (see below).

§ 501. The Brain and Spinal Cord.—Looked at in its simplest form, the **brain** consists of a mass of white fibres (the corona radiata) spreading out towards the surface like a fan, or the petal of a tiger lily, the edges of which are covered by the grey cerebral cortex. This surface or cortex is amplified by means of foldings (convolutions) which, in the genus homo are more numerous than in any other of the vertebrata (Figs. 145 and 146). Unlike the spinal cord, the grey matter of the brain is found chiefly upon its surface. But in the interior, at its lower part, there are three masses of grey matter which, from before backwards, are the corpus striatum, the optic thalamus, and the corpora quadrigemina (two on each side). The corpus striatum is divided into two grey masses (the caudate nucleus internally and the lenticular nucleus externally) by a most important band of white matter, the *internal capsule*, which carries the conducting strands from one side of the brain to the opposite side of the body and limbs (Fig. 149). Internal to the lenticular nucleus comes the anterior end of the thalamus.

In regard to its *Functions* (Fig. 147) the cortex of the brain may be divided roughly into three parts, anterior, posterior, and middle third. The anterior third, or frontal portion, is the seat of the intellect. Gross lesions in this position may exist for a long time without any symptoms other than dulness and stupidity, headache, and perhaps vertigo and nausea.[1] The posterior third, or roughly the occipital portion, is the seat of vision and several other important sense perceptions. The middle portion just in front of the fissure of Rolando forms the motor area. The different centres for movements of the limbs on the opposite side are shown in Figs. 147 and 148. It will be observed (and this will enable us to remember the position of these centres) that the most complex movements, those of the lips and tongue, are farthest *forward* and lowest down, nearest the frontal or intellectual region. Behind and above this in order come the centres for the face, arm, leg, and (on the median aspect of the hemisphere) the trunk. This order, it will be observed, is that of less and less complexity of movement. These centres are not absolutely defined, but overlap each other. This, the Rolandic, region is called the motor area because (1) electrical stimulation in animals gives rise to movements, (2) irritative lesions give rise to convulsions, and (3) destructive lesions in this position cause paralysis, of the respective limbs.

Diseases of the Brain may be classed under circulatory lesions, tumours, inflammatory lesions, degenerative lesions, and functional diseases. Vascular lesions (hæmorrhage, embolism, thrombosis) are of sudden, tumours of more gradual, onset. Functional diseases include hysteria, epilepsy, insanity, and neurasthenia. Syphilis may affect the brain in four ways—endarteritis (leading to thrombosis), meningeal affections, gummata, and degeneration, as in paralytic dementia. Gummata constitute one of the most frequent forms of cerebral tumours.

[1] Apathy and somnolence were absolutely the only symptoms in a case shown by Dr. Ferrier at the Neurological Society in 1892, and in two cases under my own care at the Paddington Infirmary, in 1892, verified by autopsy. These symptoms occur in monkeys deprived of their frontal lobes, but they sometimes recover completely, and regain their faculty of attention.

Medulla and **Spinal Cord.**—Those parts of the bulb and the pons varolii which form the floor of the fourth ventricle and the iter a tertio ad quartum ventriculum, contain a series of grey nuclei which give origin to the cranial nerves. These form, as it were, a continuation upwards of the anterior and posterior horns of the grey matter of the spinal cord. Indeed, if we imagine the spinal cord to be split from behind, and as it is traced upwards, to be opened outwards, so that the posterior grey cornua come to be external, and the anterior columns come to the surface beside the middle line, we should find, what is actually the case, that the *motor* nuclei of the twelfth, eleventh, seventh, sixth, and the fifth cranial nerves (motor nuclei corresponding to the anterior horns) lie on each side of the middle line, and that the sensory nuclei of the tenth, ninth, eighth, and fifth nerves (corresponding to the posterior horns) lie in a more external situation. The nuclei of the other motor nerves (fourth and third) lie much farther forward beneath the aqueduct of Sylvius.

Functions.—The bulb contains important reflex and automatic centres. The *reflex centres* are for the closure of the eyelids, sneezing, coughing, sucking, mastication, secretion of saliva, swallowing, vomiting, dilatation of the pupil, and the vaso-motor regulation (general dominant reflex centre). The *automatic* centres are respiratory, cardio-inhibitory, vaso-motor, sweating, and convulsion centres. The convulsion centre is situated just where the medulla joins the pons ; stimulation of it causes general spasms. The centre may be excited by a venous condition of the blood, as in asphyxia, anæmia (as when the carotids are tied), or congestion, as in compression of the veins coming from the head.

The **spinal cord** in the adult extends from the margin of the foramen magnum to a point opposite the first lumbar spine, and measures 16 inches. In the infant it extends to the bottom of the spinal canal. There are enlargements in the cervical and lumbar regions, containing the ganglion cells for the nerve supply of the upper and lower limbs respectively. In the *white* matter of the cord, which is concerned in conducting impulses upwards and downwards, certain columns have been mapped out by means of developmental and experimental inquiries, and by studying the position of secondary descending and ascending degenerations ; these are shown in Fig. 150. The best way to regard the *grey* matter of the cord is as a series of segments or discs, superimposed one upon the other, thirty-two in number, corresponding to the pairs of the spinal nerve roots, each segment being concerned in the three functions of the cord—viz., conduction, reflex action, and the regulation of certain neuro-muscular phenomena.

The Motor Tract (Fig. 149).—It is important to remember that a motor impulse passing from the cerebral cortex to the periphery must pass through two neurons : (1) The *upper motor neuron*, connecting the cortex with a multipolar cell in the medulla or cord ; (2) the *lower motor neuron*, which consists of the multipolar cell in the anterior horns of the cord, and the nerve fibre from this to the muscle fibres. A motor impulse starting in the Rolandic area in the cortex passes through the corona radiata in the central white matter of the brain, through the internal capsule—in which situation all the motor fibres are collected into one small bundle occupying the anterior two-thirds of the hinder limb—through the middle two-fifths of the crus cerebri of the same side, through the pons in a band lying between the superficial and deep transverse fibres, and through the anterior pyramids of the medulla. Here the bulk of the motor fibres cross to the opposite side to form the *crossed pyramidal tract* in the lateral columns of the spinal cord. This tract diminishes in size from above downwards as the terminal dendrites come into relation with those of the anterior multipolar cells (the dark band in Fig. 149, horizontal section in Fig. 150). At the point of decussation in the medulla a few of the motor neuraxons, instead of crossing over, pass down the same side of the cord in the anterior column close beside the anterior fissure, forming the *direct pyramidal tract* which terminates about the middle of the dorsal region. The lower motor neurons (the spino-muscular level) start in the multipolar cells of the anterior horns (or their analogues, the motor cranial nuclei), and pass out through the anterior nerve roots (or the cranial nerves) into the peripheral nerve trunks,

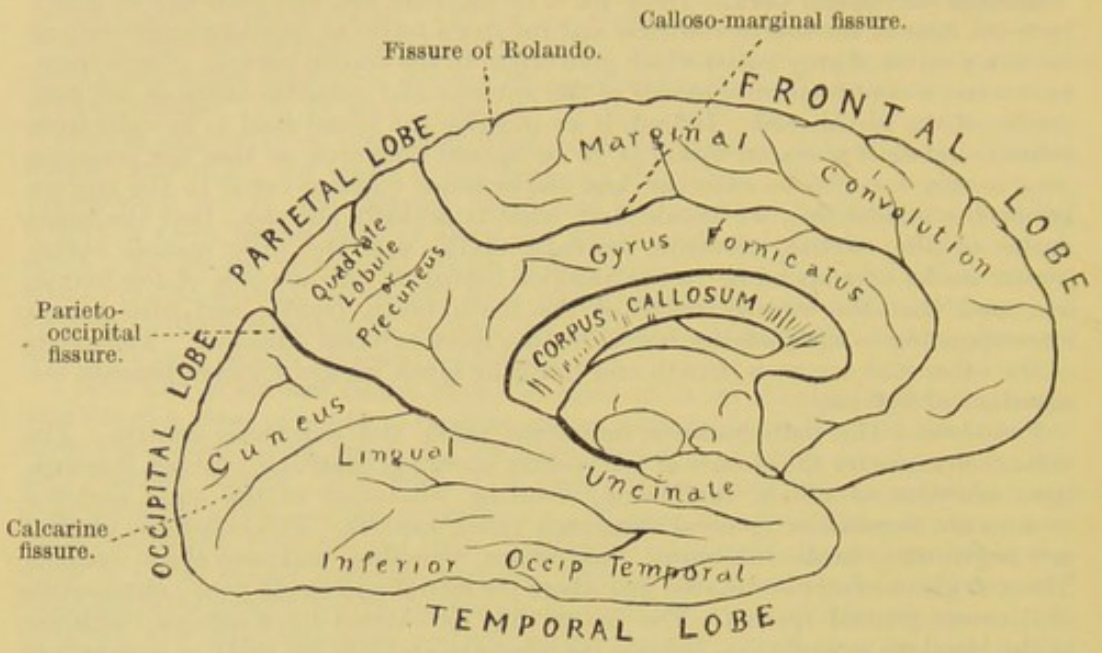

FIG. 145.

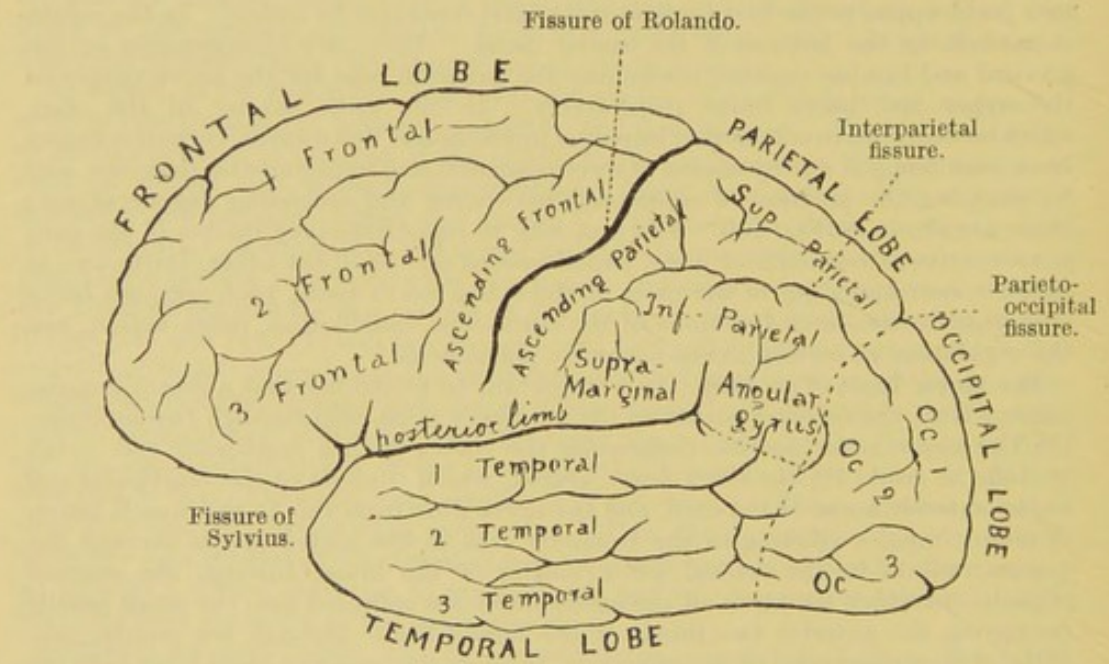

FIG. 146.

Figs. 145 and 146.—CONVOLUTIONS AND FISSURES OF THE BRAIN, external surface (lower figure) and median aspect (upper figure) of the left cerebral hemisphere. The uncinate lobule is sometimes called the gyrus hippocampus. This with the gyrus fornicatus together form the falciform lobule. The paracentral lobule is the posterior part of the marginal convolution.

and terminate in the muscles. The total number of fibres (neuraxons) of the lower level passing out through the anterior roots is far greater than the number of neuraxons in the upper level; it follows therefore that one neuraxon of the cerebro-spinal series must control several associated neurons of the spino-muscular level.

Taking a *horizontal section of the internal capsule* (Fig. 156, p. 800), the motor

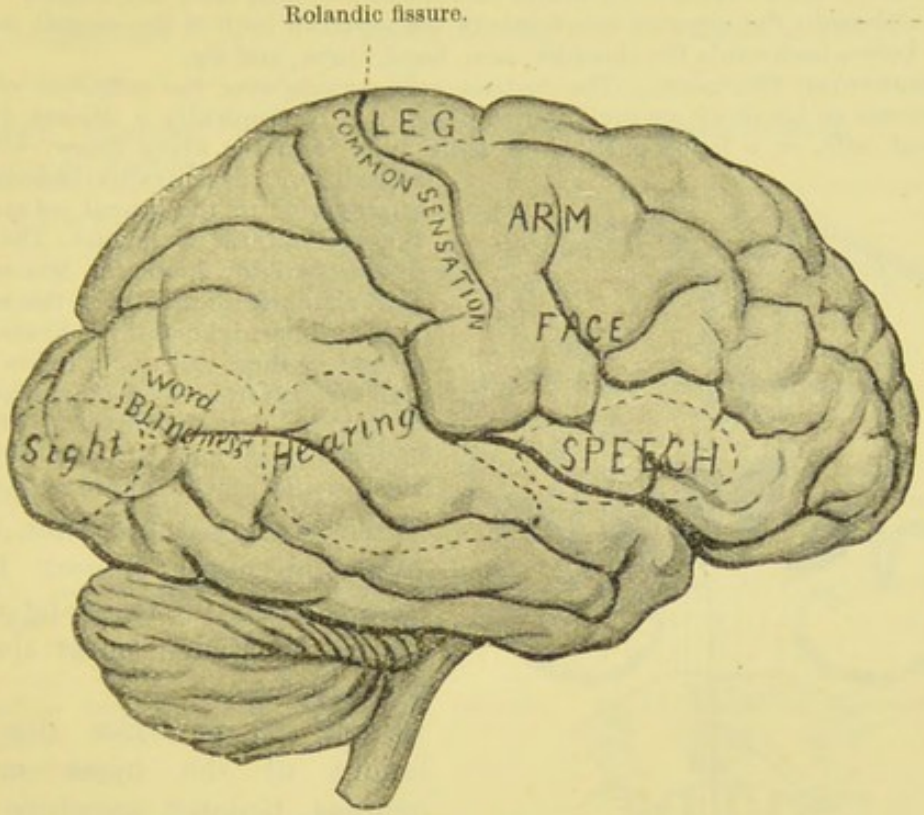

FIG. 147.

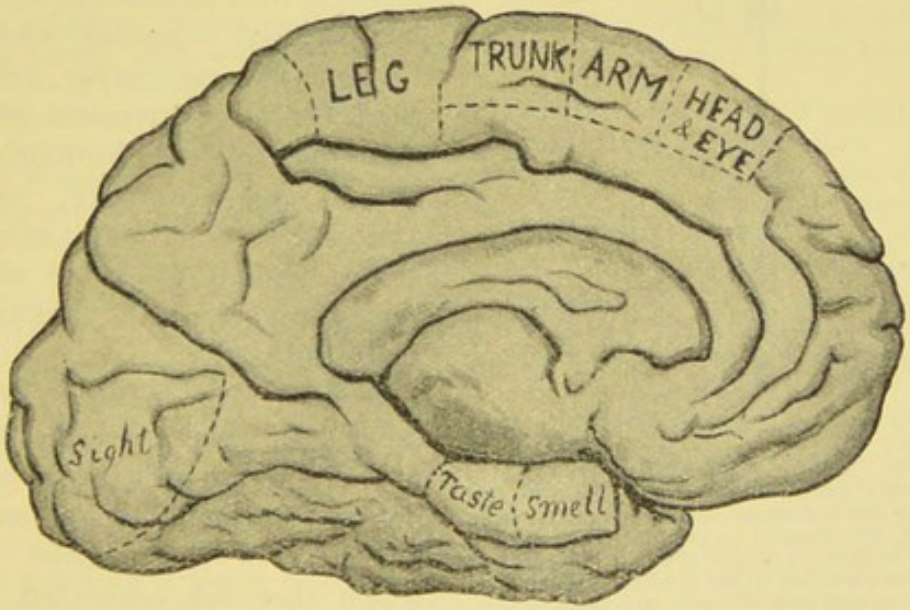

FIG. 148.

Figs. 147 and 148.—LOCALISATION OF THE CHIEF FUNCTIONS on the cerebral cortex of the RIGHT HEMISPHERE, outer surface above, median aspect below.—The *motor area* was formerly located both in front and behind the fissure of Rolando, but Sherrington and Grünbaum's researches (Trans. Path. Soc. Lond., 1902), confirmed by Alfred Campbell and others, have shown that it lies wholly in front of this fissure. Moreover, *Common Sensation* is now located behind the fissure of Rolando (see p. 718). NOTE.—The *Speech Centre* occupies the posterior half of the third **left** frontal convolution. It is indicated on this, the right. hemisphere, only for completeness.

fibres in the genu supply from before backwards the eyes, face, and tongue. The motor fibres in the anterior two-thirds of the posterior limb of the capsule supply from before backwards the shoulder, arm, hand, trunk, and leg.

Descending Sclerosis.—The cortical cells preside over the nutrition of the neuraxons as far down as the anterior horns, and consequently a disease of the cortical cells, or a lesion cutting off these cells from the fibres below, will be attended by a degeneration (**descending sclerosis**) down the lateral column to the ends of these neuraxons. The cells in the anterior horns of the spinal cord similarly preside over the nutrition of the peripheral nerve fibres, and disease of these cells, or a lesion cutting them off from the neuraxon below, is followed by degeneration of the motor nerves and atrophy of the muscles with which they are connected.

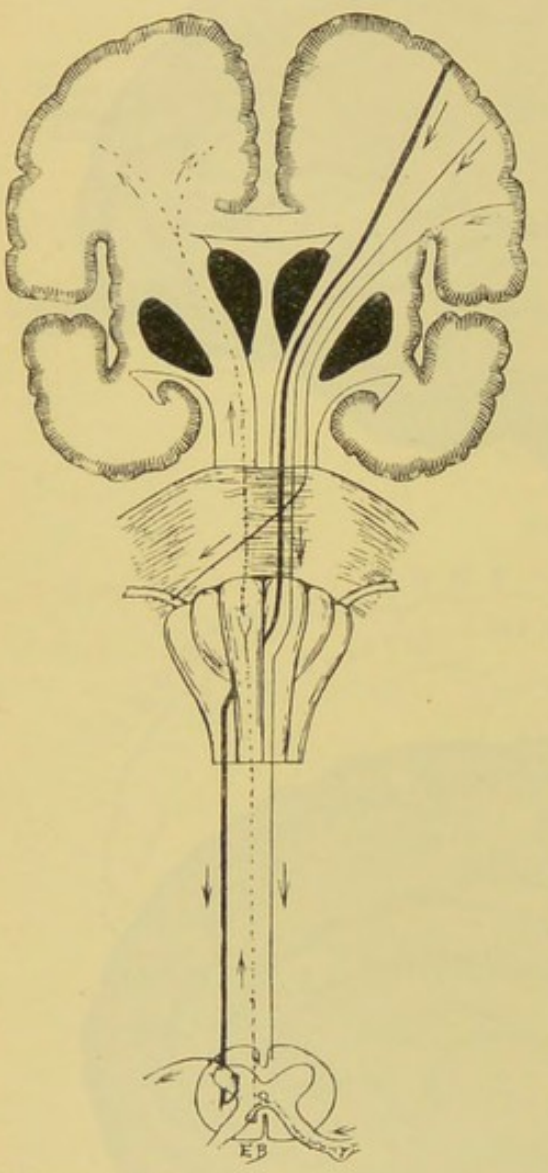

Fig. 149.—Motor (descending arrows) and Sensory (ascending arrows) Tracts.—The motor tract showing the course of the facial fibres, the crossed pyramidal tract (thick), and the direct pyramidal tract. The sensory tract shows the crossing, at the spinal level, of the sensations of touch and pain.

We shall see hereafter that two important clinical facts follow from the foregoing data which the student must always remember.

First, all paralyses due to lesions of the **upper motor neurons,** situated anywhere between the cortex and the anterior horns, are attended by muscular rigidity, with increase of reflexes, but without muscular wasting, because the multipolar cell of the lower neuron remains intact. Consequently, all hemiplegic lesions and paraplegic lesions attended by lateral sclerosis are recognised by being RIGID, NON-ATROPHIC, with INCREASED DEEP REFLEXES.

Secondly, the anterior multipolar cells which start the lower neuron, preside over the nutrition not only of the peripheral nerves, but also of the muscles with which they are connected, and consequently **lower motor neuron** lesions, on the other hand, are characterised by being FLACCID, ATROPHIC, with LOSS OF DEEP REFLEXES.

Tract or **System Lesions of the Cord** are degenerative lesions limited to one or other *vertical* tract (Fig. p. 718). The symptoms of these diseases differ from lesions, such as transverse myelitis, which involve a *horizontal segment* of the cord. (Table on opposite page.)

Symptoms.	*Tracts Affected.*	*Types of Disease.*
Rigidity, increased reflexes, no wasting.	Crossed pyramidal tract.	Lateral sclerosis.
Ataxy, loss of reflexes.	Posterior columns.	Tabes dorsalis.
Flaccidity, loss of reflexes, wasting, and R. D.	Anterior horns.	Anterior poliomyelitis, acute and chronic.
Ataxy, rigidity, increased reflexes, no wasting.	Posterior columns, direct cerebellar and crossed pyramidal tracts.	Ataxic paraplegia.
Rigidity, increased reflexes, some wasting.	Anterior horns and crossed pyramidal tract.	Amyotrophic lateral sclerosis.
Ataxy, tremor, and loss of knee jerk.	A combined disease of posterior and lateral tracts.	Friedreich's hereditary ataxy.
Intention tremor, nystagmus, and other symptoms.	Scattered patches of sclerosis.	Disseminated sclerosis of Charcot.

§ **502. Sensation and the Sensory Tract.**—The sensory or centripetal tract is not so clearly known as the motor, chiefly because of the difficulty of accurately testing it in man and animals.

The *peripheral afferent nerves* may, from the researches of Head, Rivers, and Sherren (*Brain*, 1906, and elsewhere), be *differentiated into three systems according to the kinds of sensation* they transmit:

1. The Epicritic System of fibres run in the cutaneous nerves. They transmit **tactile** sensation and the recognition of *slight* differences of **temperature.** Interruption of these abolishes: (*a*) Recognition of light touch (*e.g.*, by cotton-wool); discrimination of size of object and compass points; (*b*) cutaneous localisation; and (*c*) discrimination of temperature between about 25° and 40° C.

2. The Protopathic System of fibres also run in the cutaneous nerves, but by fibres independent of the preceding. They transmit **painful** cutaneous sensations and *extremes* of **temperature**. Interruption abolishes: (*a*) Cutaneous pain (*e.g.*, by pricking or strong Faradic currents); (*b*) cutaneous temperature sense below about 20° C., and above about 45° C.

3. The Deep Sensibility System of fibres run chiefly in the nerves to the muscles. They transmit deep **pressure** sensations and **muscular** sensations. These are not destroyed by dividing all the cutaneous sensory nerves. Loss of deep sensibility abolishes: (*a*) Sense of movements (extent and direction) in joints, tendons, and muscles (muscle sense and position of a limb), as tested by distinguishing the difference of weight of objects of similar size, or by ability to place one limb in the same position as the other with eyes closed (compare Muscle Sense, § 503 below); (*b*) sense of pressure (position and degree) in the deep parts.

The leading fact to remember about common sensation (tactile, painful, and thermal) is that it enters the cord by the posterior roots (Fig. 149), and crosses to the opposite side soon after entering, so that a unilateral lesion of the cord produces paralysis of the same, anæsthesia of the opposite, side (Brown-Séquard's paralysis). After entering the cord from the periphery the sensory neuraxons take several different routes. (1) One set of fibres enters the postero-external (Burdach's) column, or into the postero-median (Goll's) column. (2) Other fibres end in the grey

matter of the cord round cells whose axis-cylinders cross and ascend the cord in the antero-lateral (Gowers') tract. (3) The direct cerebellar tract, passing on to the cerebellum. (4) Another set of fibres passes into the grey matter of the cord and ends round the anterior horn cells, thus forming the path of the reflex arc. *The first three tracts pass up to the medulla.* Goll's and Burdach's columns form the funiculi in the medulla, and fibres from these cross in the medulla and pass up together with Gowers' column and the fifth cranial nerve (which joins the sensory tract at that part) in the lemniscus or fillet which occupies *the posterior part of the pons*, through the *tegmentum of the crus* cerebri, to the *basal ganglia*, where some of the fibres end. The sensory tract in the brain then passes up through the posterior third of the hinder limb of the *internal capsule* (sensory crossway), coming, be it observed, from the opposite limbs. In this position it receives the visual and auditory fibres from the optic and auditory nerves, and probably also from the nerves of smell and taste, all from the opposite side. The sensory tract continues upwards through the corona radiata. Some doubt has hitherto existed as to the part of the cortex

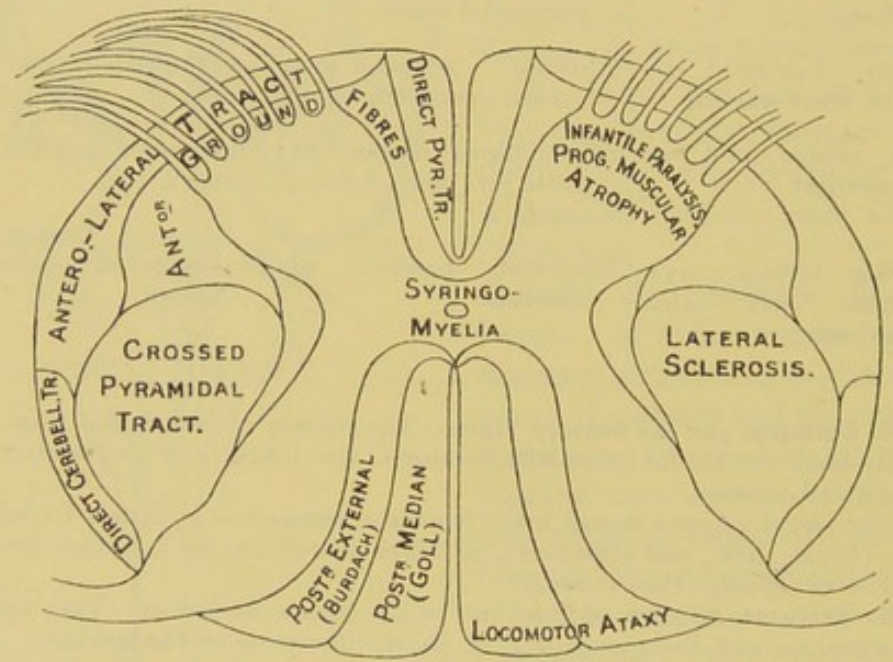

Fig. 150.—TRANSVERSE SECTION OF SPINAL CORD in the cervico-dorsal region.—DESCENDING DEGENERATIONS take place chiefly in the crossed pyramidal tract and direct pyramidal tract: chief ASCENDING DEGENERATIONS in the columns of Goll and Burdach, Gowers' tract, direct cerebellar tract.

subservient to sensation. Sir Victor Horsley originally located common sensation in the gyrus fornicatus, and the author recorded a case[1] where a localised lesion in this situation was attended by hemianæsthesia. But the most recent researches of Sherrington and Grünbaum in animals, Horsley,[2] and Cushing[3] in man, seem to indicate the ascending parietal convolution (post-central gyrus) as the more probable position (Fig. 147). The last named observer stimulated the post-central gyrus of a conscious patient by unipolar faradisation.

ASCENDING DEGENERATION, secondary to lesions in the cord lower down, is found in the columns of Goll and Burdach, the antero-lateral tracts, the direct cerebellar tracts, and sometimes in the comma-shaped tract.

§ 503. The Cerebellum, Co-ordination, and Equilibrium.—In order that our balance in the erect posture may be preserved and our different muscular movements effective, the contractions of the various muscles must be co-ordinated or correlated to one another. The centre for this co-ordination appears to lie in the cerebellum, and especially in its middle lobe. The cerebellum consists of two hemispheres and a middle lobe. It is connected above with the cerebrum by the superior peduncles, below with the medulla by the inferior peduncles, and in front

[1] *Brain*, 1891, p. 270. [2] *Brit. Med. Journ.*, July 17, 1909. [3] *Brain*, part i., 1909.

with the pons varolii by the middle peduncles—cerebro-afferent fibres. In order to co-ordinate the various movements and to preserve equilibrium of the body, the cerebellum must be kept correctly informed of the relation we bear to surrounding objects, and also as to the state of contraction of the various muscles. This is accomplished by four different sets of afferent or in-going impulses. (1) The *sensation* of touch of surrounding objects is transmitted through the paths of common sensation. (2) The *sight* of surrounding objects informs us of our relation to them, and therefore it is assumed centripetal fibres pass from the centres for movement of the eyeballs and sight to the middle lobe of the cerebellum, to assist in regulating the attitude of the body so far as it relates to the maintenance of our proper distance from the objects around us. It is possibly in this way that nystagmus comes to be a symptom of cerebellar tumour.

(3) *Orientation*, or the position in which our body lies at any given moment in relation to vertical and horizontal planes, is recognised by impulses coming from the semi-circular canals, transmitted through the vestibular portion of the auditory nerve to the cerebellum. Disease of the semi-circular canals or of this nerve produces a feeling of giddiness or unsteadiness, and there may be an inability to stand (*e.g.*, in Ménière's disease).

(4) *Muscle-Sense.*—A knowledge of the state of contraction of our muscles is recognised as muscle-sense, and it is necessary for the preservation of equilibrium and co-ordination. This muscle-sense tract starts, we now know, in the muscle spindles.[1] It passes through the nerves into the spinal cord by the posterior root, up the columns of Goll (postero-median columns), and the direct cerebellar tracts to the middle lobe of the cerebellum. The columns of Goll transmit muscle-sense impressions from the lower extremities ; the direct cerebellar tracts those from the upper dorsal and cervical regions chiefly. Interruption of these muscle-sense tracts is the principal factor in the production of tabes dorsalis (§ 578, Fig. 163).

Destruction of any one of these four tracts disturbs equilibrium more or less. For instance, in tabes dorsalis there is generally anæsthesia of the soles of the feet (1 above is destroyed), and the muscle-sense (4) is also disturbed, so that when the eyes are shut and path 2 is interrupted, the patient tends naturally to fall (*Romberg's sign*).

Cerebello-Efferent Tracts.—The cerebellum only regulates, it does not initiate, muscular contractions. These are started by the cerebrum, and are merely controlled by the subordinate centre in the cerebellum. The connection of the cerebellum with the cerebral hemispheres is mainly a crossed one, fibres passing from the cerebellar hemisphere of one side to the cerebral hemisphere of the opposite. *Lesions of the cerebellum* produce (1) a staggering or reeling gait, and a deficiency in equilibrium, but they do not produce absolute paralysis. Lesions in the upper part of the middle lobe produce a tendency to fall forwards ; those of the lower part to fall backwards ; and if of the lateral lobe, the patient may show a tendency to fall *towards* the affected side, or there may be forced rotary movements towards the affected side. Tumour of the lateral lobe may produce inco-ordination and paresis of the limbs on the same side, from pressure on the motor tract or from affection of the contra-lateral cerebral centre.

§ 504. The Membranes of the Brain and Spinal Cord should be mentioned together, as they are identical in structure, continuous with one another, and subject to the same diseases.

The membranes of the brain and cord are subject to a great many *lesions*. (1) Meningitis is inflammation of the membranes ; several different forms are recognised. (i.) External or pachymeningitis begins and predominates in the dura. (ii.) Internal or lepto-meningitis is inflammation which begins and predominates in the pia and arachnoid. (iii.) A specific epidemic form of meningitis is recognised, which consists of a lepto-meningitis in which the pneumococcus of Fraenkel has been found. (iv.) A septicæmic form of meningitis is recognised, which is apt to complicate scarlatina and other acute diseases. Meningitis has also been traced to syphilis and bacterial processes. (2) Tubercle is one of the commonest diseases

[1] F. E. Batten, *Brain*, 1897, part xx., p. 138.

affecting the meninges in children. It involves the pia mater and arachnoid, and almost invariably starts and predominates in the transverse fissure and fissure of Sylvius. (3) Syphilitic gumma of the meninges is the commonest meningeal disease in adults. Syphilis also produces simple thickening of the meninges and disease of its arteries. (4) Cancer chiefly affects the dura mater secondarily to cancer in other parts of the body. (5) Fibrous thickening of the dura is found as a chronic form of pachymeningitis. (6) Bony plates may be found as the result of a chronic meningitis. I have generally found them in the arachnoid, and chiefly in old people. (7) Hæmorrhage may take place in both the meninges of the brain and cord. In the former position it is known as hæmorrhagic pachymeningitis, and in the latter hæmatorrhachis. The blood may be effused in three positions; it may be extradural, subdural, or subarachnoid. (8) Finally, the meninges of either brain or cord may be the seat of injury.

In all these disorders the disease may start or predominate either in the cranium or the spine, but it is very apt to spread to the other. It must also be remembered that the surface of the brain and spinal cord derive their nourishment almost entirely from the pia and arachnoid which invest them, and therefore diseases of the membranes impair their nutrition considerably. And since the cortex of the brain is the most important part functionally, grave consequences may ensue from meningeal diseases.

§ 505. The Cerebral Circulation.—The ARTERIES of the brain are derived from two internal carotids and the vertebrals, which form the circle of Willis (Fig. in § 552). The middle cerebral is the most important artery of the brain, and it is a continuation onwards of the internal carotid. The internal carotid (which arises from the highest part of the aorta) and left middle cerebral are in a direct line for emboli from the valves of the heart. There are two series of branches of the middle cerebral, the external branches and the internal branches. The left sylvian artery, which constitutes the chief supply of the internal capsule, is also in the direct line from the heart, and as it feels the full ventricular shock it is frequently the seat of hæmorrhage. For the same reason also it is frequently the seat of embolism in younger persons, and this accounts for the greater frequency of right hemiplegia in circulatory lesions. Figs. 151 and 152 show the vascular areas supplied by the cerebral arteries.

VEINS AND SINUSES.—The venules collect the blood into veins of the brain (which have no valves), which enter the cerebral sinuses backwards. Most of the blood leaves the cranial cavity through the internal jugular veins, which are continuations of the lateral sinuses. Should they be blocked, there are a few collateral communications through which the intracranial circulation communicates with the veins outside the skull. Thus (1) the interior end of the superior longitudinal sinus communicates with the veins of the nose; (2) the ophthalmic veins communicate through the orbit with the facial veins; (3) the lateral sinus communicates with the occipital veins through the mastoid cells; (4) the superior longitudinal sinus communicates through the calvarium with the veins of the scalp; and (5) the inferior petrosal sinus communicates with the deep cervical veins. The veins of Gâlen collect the blood from the collateral plexuses in the lateral ventricles, and empty themselves into the straight sinus, and thence into the torcular Herophili and through the lateral sinus into the internal jugular vein. Pressure on or thrombosis of these veins produces distension of the lateral ventricles with fluid (hydrocephalus) if the foramen of Majendie be occluded.

The PECULIARITIES OF THE CEREBRAL CIRCULATION are as follows: (1) There are no anastomoses between the arteries of the cortex and those of the interior of the brain; there are no anastomoses between the several branches in the interior, and only very few between the several cortical branches, each of which supplies its own area. (2) There are no communications between the several branches of the vertebral, basilar, and cerebellar arteries, which supply the pons and bulb; but the superior, middle, and inferior cerebellar arteries communicate freely, hence the greater frequency of vascular lesions in the former as compared with the latter positions. (3) The cranium being a rigid box the quantity of blood in it is always

the same, but the velocity varies considerably, and the velocity depends on the blood-pressure in the carotids and vertebrals. The blood-pressure in these varies inversely as the dilatation of the splanchnic area, and the rapidity of the cerebral circulation, it seems quite clear, is regulated mainly by the great reservoir in the splanchnic area. When this is dilated the general blood-pressure is low, and the circulation in the brain is slow; conversely, when the splanchnic area is contracted, the general blood-pressure is raised, and the circulation of the brain becomes more

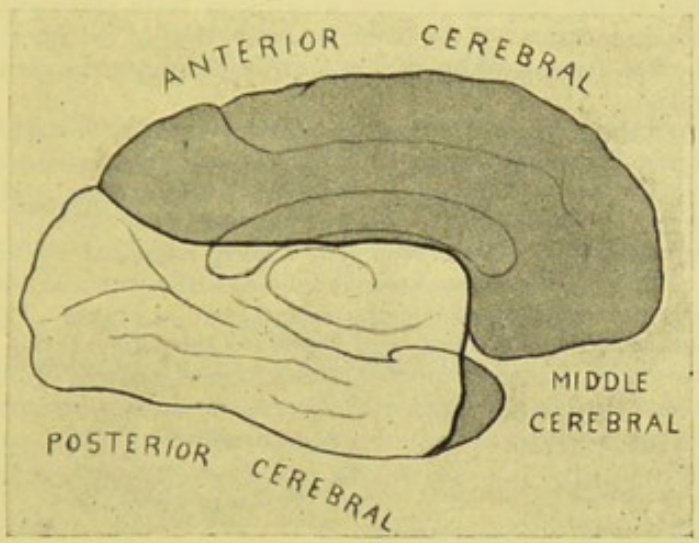

FIG. 151.

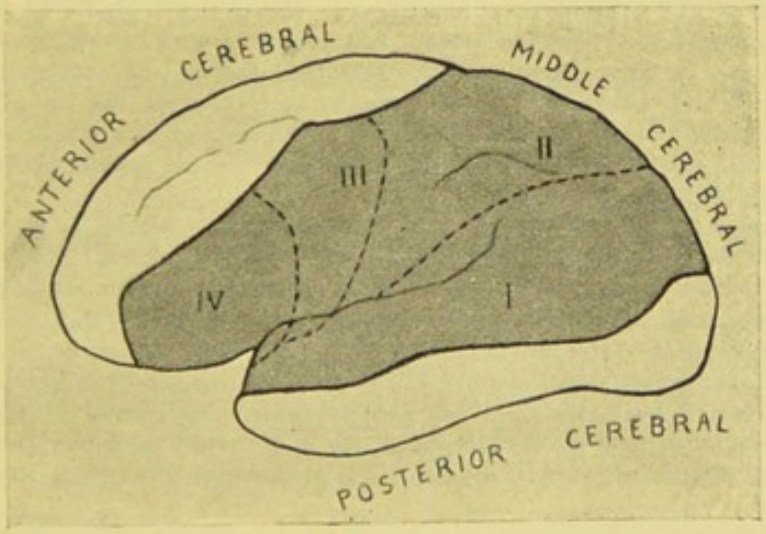

FIG. 152.

Figs. 151 and 152 show the areas of distribution of the CEREBRAL ARTERIES—median surface of the brain above, external aspect of the brain below.

rapid. Dr. Leonard Hill maintains that there is no vaso-motor regulation of the cerebral arteries, a fact which is hard to believe in view of the amount of involuntary muscular tissue present in these vessels. Professor Sherrington maintains that they have the same nerve-supply as the other vessels. At any rate, high blood-pressure in the carotids, and therefore in the cerebral circulation, is attended with rapid circulation in the brain (wakefulness), and conversely low blood-pressure is attended with slow cerebral circulation, as in sleep.

§ **506. The Sympathetic Nervous System** is almost as extensive and elaborate as the cerebro-spinal nervous system, with which it is intimately connected. The sympathetic system consists in the main of a double series of ganglia arranged along the ventral aspect of the vertebral column. In the abdomen these ganglia are massed and matted together into the cœliac plexuses and semilunar ganglia, which evidently constitute the chief centres, the " brain," as it were, of the sympathetic system. The sympathetic ganglia are connected by non-medullated fibres with one another, with the cranial nerves, with the anterior and posterior roots of the spinal cord, with all the arteries (around which they form plexuses), and with all the unstriped muscular tissue of the body—namely, that of the arteries and that of intestines and other viscera. The only named branches are the greater and lesser splanchnic nerves.

One function of the sympathetic system is to regulate the flow of blood through the arteries by means of their tunica media or muscular coat, and thus to control and co-ordinate the various vascular areas one with another, and regulate the nutrition going to the tissues and viscera. It has been shown (by Dr. Leonard Hill, for example) that the cerebral circulation is mainly regulated by the contraction and dilatation of the vascular area (the splanchnic) within the abdomen. Stimulation of the sympathetic fibres to the heart accelerates that organ. This system also regulates the intestinal movements, and therefore the alimentation of the body. It also regulates the secretion of various glands either directly or indirectly through the vessels which supply them. The pathological effects are most definitely recognised in the case of paralysis of the cervical sympathetic (§ 612). In the author's view the sympathetic system is also largely responsible for most of the phenomena of hysteria, many of those ascribed to neurasthenia, and some of the other so-called functional diseases of the nervous system.

§ **507. General Principles in Neuro-pathology.**—(1) Whenever the nutrition of a neuron is impaired, *the peripheral endings of* its processes (dendrites) are the first to show degeneration. An illustration of this is seen in tabes, where the peripheral terminations of the muscle-sense neuraxon in the muscle spindle is the first to undergo degeneration. This principle, which has a widespread application in neuropathology, is the outcome of the fact that the cell or neuron-body is the most essential and vital part of the neuron, and controls the nutrition of the neuraxon and dendrites. Consequently, when the neuron as a whole suffers from want of nutrition or other cause of degeneration, the parts *farthest from the cell* first show the degenerative change.

(2) *Prolonged forced functioning of any nerve structure results in its atrophy; it also results in the predominance and overpowering of that structure by the surrounding tissues, and a final degeneration of the fatigued structure.* This is sometimes known as Edinger's theory, but it is a principle with which neurologists have long been familiar. A typical instance of it is seen in the tremor, spasm, and atrophic paralysis which accompany various occupation neuroses. It explains also the predominance of tabes in the male sex, who use their muscle-sense neurons so much more than females, and it also explains the fact why tabes supervenes during the prime of manhood, at a time when the muscle-sense neurons are exercised most.

(3) *Sclerosis is a consequence, not a cause, of the atrophy of the parenchymatous tissues* (nerve structures proper). This is a general pathological principle, which is applicable also to the nervous system. It was formerly thought that the occurrence of fibrous tissue in cirrhosis of the liver, or fibrosis of the kidney (contracted granular kidney) was the cause, by pressure, of the atrophy of the glandular cells, but modern pathology teaches the converse of this, and shows how true was the statement of Cohnheim, made about the year 1860, that " the mutual resistance of tissues to each other's encroachment, limits tissue growth normally. If you remove one tissue element—by degeneration or otherwise—the surrounding tissues (deprived of this opposition), tend to take on increased activity, growth, and proliferation." An application of this principle is seen also in tabes, where, as a consequence of the degeneration and atrophy of the muscle-sense neurons, secondary

sclerosis occurs of the posterior columns, in which the muscle-sense neuraxons lie. It is seen in many other scleroses of the spinal cord.

(4) *Functions which are last acquired in the evolution of the nervous system are the first to succumb to disease, and vice versa.* One illustration of this principle is seen in mental disorders, in which the latest acquired faculties of the mind are those which most readily become disordered. Again, in aphasia (loss of memory for the signs of thought), the memory for printed or written signs is lost more often and sooner than the memory for spoken words.

PART A. SYMPTOMATOLOGY.

The most constant and CARDINAL SYMPTOMS of diseases of the nervous system may be *subjective* or *objective.* Among the objective symptoms **defects in muscular power** are the most obvious, because the muscular system is entirely dependent on the nervous system; and these will be considered seriatim under Paralysis, Inco-ordination and Disordered Gait, Muscular Rigidity, Tremor, and Amyotrophy, in Parts B. and C. **Defects of sensation** and the **special senses** will also be there considered.

The *subjective symptoms* met with in disorders of the nervous system are very numerous, but they can practically all be brought under one of six headings—**defects of the mental powers, nervousness, pain** (including **headache**), **disordered sleep, vertigo,** and **disordered sensations.**

The **mental powers** are disturbed in greater or less degree in most diseases of the nervous system, particularly in those of the brain. Generally there is inaptitude for mental work; sometimes there are transient disturbances of thought; at other times there is stupor, mental apathy, or coma. Sometimes there is loss of speech or of memory, or some other faculty of the mind. Sometimes there is restlessness, excitement, or delirium; at others there is a more chronic perversion of the mind, which amounts to insanity. A simple epitome of these various symptoms and their causes occupies many pages (§§ 537 to 542).

In the mental symptoms which arise in certain inflammatory and other diseases of the brain it is useful to remember that delirium, convulsions, and headache are evidences of *cerebral irritation,* whereas mental dulness, stupor, paralysis, and coma are evidences of *cerebral compression* or intense toxæmia.

§ **508. Nervousness** is a symptom which frequently guides us to disorders of the nervous system. In its colloquial sense it generally means "easily agitated." The patient comes to us, for instance, because the least noise startles him or the least worry upsets him. (1) It is the leading and most constant symptom in *neurasthenia,* an asthenic state of the nervous system (§ 523) arising from gastro-intestinal or other toxæmia, malnutrition, fatigue, accident, or some emotional strain.[1] (2) *Hysteria* is the next most common cause of nervousness. But here we have an inherent emotional and vaso-

[1] "Clin. Lects. on Neurasthenia," fourth edition, Glaisher and Co., London, 1909.

motor instability, which is manifested by "hysterics" and various other forms of attack.[1] (3) In a great many *structural diseases* of the nervous system the patient comes to us for what he calls nervousness, particularly in those which will be mentioned under the symptom tremor. (4) Chronic alcoholism, morphinism, and their effects, are manifested by nervousness; and (5) convalescence from severe illness and many other debilitated conditions are also attended by nervousness.

§ **509. Pain and Neuralgia.**—Pain is a subjective symptom of considerable frequency in all diseases, but not more in those of the nervous system than other systems unless the peripheral nerves are involved. We know but little about the psychology and pathology of pain, but a careful clinical observer may derive considerable help by investigating the four important qualities which I have many times mentioned: (1) Its position; (2) its character—whether throbbing, pricking, shooting, knife-like, dull, aching, etc.; (3) its degree; and (4) its constancy—*i.e.*, whether persistent or intermittent, or a combination of the two (paroxysmal or exacerbating). We should never allow ourselves, for the sake of time or trouble, or by pandering to the ignorance or whim of our patients, to treat pain simply as pain, by the administration of soporifics, hypnotics, and the like. Pain is a sure indication of abnormal structure or function, and it is our bounden duty to endeavour to trace out its *causal agency*, however difficult the task may be. The best method of investigating the cause of a neuralgia or pain in any given case is, first, to examine the nerve apparently involved in the pain, and the *locality* around, for direct causes of irritation, and particularly any bony orifice through which the nerve passes; secondly, to seek for any *reflex cause* of irritation in disease of organs more or less distant, such as the teeth or the uterus; and, thirdly, to search for any general or *constitutional derangement* which may act as a predisposing or exciting cause, such as anæmia, rheumatism, gout, tubercle, or syphilis.

Pain and Neuralgia in different parts of the body and limbs are discussed fully under Neuralgia (§ 604).

§ **509*a*. Headache** (Cephalalgia) is often met with in diseases of the nervous system, but it quite as often attends morbid conditions of some other, and particularly the vascular, system. Its generalised distribution on both sides (a feature which distinguishes it from neuralgia of the fifth nerve) suggests that it is due in most cases to disturbances of the circulation within or outside the cranium. Frontal, vertical, and occipital headache were believed by Dr. Hughlings Jackson[2] to indicate an abdominal, cerebral, and circulatory origin respectively, but the position of the pain is not, in my experience, much guide to its cause.

[1] "Lectures on Hysteria," Glaisher, London, 1909.

[2] Quoted by Dr. J. S. Bristowe, "Prin. and Pract. of Med.," fourth edition, London, 1882.

The *Causes* of headache are numerous, and do not admit of pathological classification, but inquiries should be directed to possible Local, General, and Reflex causes, as in the case of pain and neuralgia.

(*a*) Among the following Local Conditions, the first five cause continuous pain of some duration. (1) *Syphilitic disease of the cranium* is a frequent cause of continuous headache, and a marked feature of this headache is its nocturnal exacerbation and its associated tenderness. (2) Various *meningeal conditions*, acute or chronic, cause pain, and here syphilis again may play a leading part. In children tuberculous meningitis should always be suspected, and the temperature carefully taken. (3) *Intracranial tumours*, especially when affecting the meninges or cortex, may be known by the association of vertigo, occasional vomiting (especially when the headache is worst), and optic neuritis; localised tenderness over the seat of the lesion is sometimes observed. (4) *Ear disease* causes headache, and in such cases pressure over the mastoid cells often reveals tenderness. (5) It must also be remembered that disease of the *frontal sinuses*, which is usually secondary to nasal or post-nasal catarrh, gives rise to dull, continuous headache. (6) The *supra-orbital branch* of the fifth nerve supplies the forehead, and neuralgia of this nerve necessarily produces frontal headache of a shooting and paroxysmal character. (7) *Excessive brain work* is frequently followed by a feeling of dull, heavy weight on the vertex. (8) The wearing of hard and heavy hats.

(*b*) Among the Constitutional or General Conditions the first seven causes mentioned below are due to toxic or hæmic conditions, and perhaps eight and nine also. All of them are apt to have periodic exacerbations. (1) *Chronic interstitial nephritis* should always be suspected in the aged, a suspicion which is confirmed when the patient frequently rises at night and passes large quantities of urine of low specific gravity. (2) *High arterial tension* is probably in operation in the foregoing, but from whatever cause arising is a very frequent source of headache. Indeed, it is generally the earliest and most pronounced symptom which attracts the patient's attention. (3) *Hepatic derangement* and the condition known as *lithæmia* give rise to what is sometimes called bilious headache. (4) *Chronic alcoholism*, *gout*, *rheumatism*, *syphilis*, and *plumbism* are also attended by headache, but it has no special characters. (5) *Ague*, though rarely seen now in this country, is attended by a severe frontal headache; hence the term "brow ague." (6) *Pyrexia*, due to any cause, is usually accompanied by headache. (7) *Chlorotic patients* suffer a good deal from headache, but it has no special features beyond the concurrent signs of anæmia. (8) Closely allied with the preceding is the headache of *exhaustion* or inanition. (9) *Hysteria* and *neurasthenia* are frequently attended by headache. The first is often likened to a nail being driven into the skull at one spot (hence the term "clavus" applied to

this headache); the second very frequently takes the form of a feeling of constriction around the head ("casque neurasthenique" of Charcot). (10) *Migraine*, which is a special paroxysmal form of headache, is described in § 605.

(*c*) Reflex Causes.—(1) *Asthenopia* or eye-strain is an extremely frequent cause of headache in modern times, and is generally associated with some uncorrected error of refraction or astigmatism. It may arise without any error in those who read small print too much. This kind of headache has, in my experience, fairly constant characters—viz., it is worst on rising in the morning, improves a little, and then again gets worse after the day's work. (2) *Constipation*. (3) *Dyspepsia* and derangement of the stomach are certainly amongst the most frequent causes of headache in everyday life. It is said by some to be situated in the frontal region, but in my belief it has no special characters beyond the associated symptoms of these maladies. (4) In *diseases of other viscera*, especially of the heart, lungs, uterus, and liver, a pain referred to the head is often present. The headache of heart disease is often known as congestive headache, and is certainly best relieved by cardiac depressants or bleeding.

The palliative *Treatment* of headache as a symptom will be found under Neuralgia and Migraine, §§ 604 and 605.

§ **510. Disordered Sleep** is not exclusively connected with nervous disorders. We have to rely very much upon a patient's own account, and some care is required in accepting his statement in this matter. Persons differ considerably in the amount of sleep they require. The aged will do with half the sleep of adolescence, and the middle-aged with half the sleep of babyhood, the respective quantities being approximately five and ten hours, eight and sixteen hours. Brain workers and town dwellers require more sleep than labourers and country folk, though as a rule they get less. The popular belief that "six hours for a man, seven for a woman, and eight for a fool" is sufficient sleep is highly fallacious. Sir William Jones' adaptation in the eighteenth century of an old Persian saying—

> "Seven hours to work, to soothing slumber seven,
> Ten to the world allot, and all to Heaven,"

comes much nearer the truth, and my own belief is that a town life, with brain work, requires at least seven or eight hours of quiet and uninterrupted sleep.

Sleep may be (*a*) deficient in quantity (insomnia), or (*b*) defective in quality (restlessness, dreaming, etc.); these generally co-exist, and their causes are interchangeable. (*c*) It may be excessive in quantity. Normal sleep is attended by anæmia of the brain. The causes of (*a*) and (*b*) depend either on inherent irritability of the brain or an excess or defect in quality (toxæmia) of its blood supply.

(*a*) Insomnia, wakefulness, or deficient sleep may arise under the following conditions: (1) All *painful affections* are apt to be attended by sleeplessness. (2) *Defective hygienic conditions* or mode of life —*e.g.*, late hours, late suppers to those unaccustomed to them, indigestible food, overwork of mind or body, unaccustomed surroundings and the like. (3) In neurasthenia, hysteria, and other functional *disorders of the nervous system*, mental excitement, all acute and many chronic forms of mental derangement, sleeplessness may be one symptom of the ailment. (4) In the absence of any of the foregoing causes, search should be made for some *general constitutional condition*. Sleeplessness, like headache, in the aged should always make one suspect chronic interstitial nephritis. Such patients often complain of "cat-sleeps"—*i.e.*, dropping off for a few minutes at a time. (5) Among *local disorders*, cardiac valvular disease may be mentioned, in which the patient starts up as soon as he falls off to sleep with a feeling of suffocation.

(*b*) Defective Sleep.—(1) *Dream-disturbed sleep*, in my experience, generally indicates a toxæmia of some kind. *Nightmares* and *dreams* in the young are often due to an undigested meal taken late. They are also a characteristic symptom of neurasthenia, anæmia, deficient oxygenation, and various other toxic conditions of the blood. (2) *Night terrors* in children are sometimes induced by worms or late suppers, or other dietetic or gastro-intestinal defects—especially in nervous children. Among other causes may be mentioned nasal or pharyngeal obstruction (*e.g.*, adenoids). If continual, they should make us suspect petit mal, especially when combined with nocturnal incontinence. There is a residue of cases in which no cause is apparent excepting the neurotic diathesis, which subsequently becomes manifest by the development of hysteria, epilepsy, chorea, or mental degeneracy.[1] (3) *Sleep-walking* and sleep-talking are curious phenomena in which certain functions of the brain are not only awake, but sometimes in an exalted condition. They are allied to hysteria, and the same treatment applies. Here again petit mal may be in operation. (4) *Twitching of the limbs* as a person drops off to sleep may be an early symptom of peripheral neuritis or of neurasthenia. It is a common symptom of some toxæmias (particularly those of intestinal origin) on the one hand, or hypersensitiveness of the nervous system (such as occurs after morphinism, § 527) on the other. In all of these the twitchings may pass on to intolerable restlessness.

(*c*) Excessive Sleep.—Drowsiness is a symptom met with in the intense venous congestion of *heart disease*; it is also an early symptom of *acute uræmia*. In some other *toxic states*, and some *organic brain* affections (especially tumour in the region of the third ventricle),

[1] See also a clinical lecture by Dr. Leonard Guthrie, *Clinical Journal*, June 7, 1899.

sleepiness is met with, and in all of these it tends to pass into stupor (§ 526). But sometimes it is met with in apparent health. I was once consulted by a curate who went to sleep almost as soon as he sat down in church during certain parts of the service, or when his rector was preaching—a most inconvenient circumstance. In that case it was apparently due to *lithæmia* and *disordered liver*, which being remedied, the sleepiness disappeared. Dyspepsia (when the somnolence follows meals) and anæmia may act in the same way. The *trypanosoma*, when affecting the cerebro-spinal fluid, produces "sleeping sickness," in which the patient sleeps for days, and then generally dies. Persistent and even fatal sleep has been known to follow *influenza*.

The *Treatment* of insomnia must be directed to the cause. It is in cases of sleeplessness with or without pain that hypnotism is sometimes of value. Among the simpler remedies, I have often found useful a cup of warm milk or gruel, or a hot bath or hot-pack, last thing at night. Sometimes an evening walk is useful, or Swedish and deep-breathing exercises, or some means which take the mind away from the occupations of the day. Massage or cold compresses over the legs or abdomen may promote sleep. Sod. hypophosphite, 20 grains, in warm milk or milk and water at bed-time is a simple yet excellent remedy for the sleeplessness of mental fatigue, or bromide of ammonium, 20 grains, thrice daily, or an alkaline draught of 30 grains of bicarbonate of soda in a tumbler of hot water. A weak galvanic current through the brain I have sometimes found good. When these simpler measures are unavailing, recourse may be had to hypnotics and sedatives—care being taken not to engender a habit—such as the following: Alcohol, chloral hydrate, camphor, cannabis indica, codeina, hyoscyamine, lupulin, narceine, paraldehyde, bromidia, chloralamide, chloralimide, chlorobrom, hypnal, sulphonal, tetronal, trional, urethane, chloralose, scopolaminæ hydrobromide, chloretone.

§ **511. Vertigo** is a subjective sensation experienced by a patient that either he or the objects round him are rotating, accompanied by a momentary loss of equilibrium. The latter may be so marked as to lead to a reeling in the gait, or actual falling down. The symptom is sometimes difficult to elicit without putting a leading question, but it is best to ask the patient if he has "any sensation" in the head. People describe it sometimes as a "giddiness," a "dizziness," or "swimming in the head." The rotation may appear to be horizontal, vertical, or oblique. The sensation is often attended by nausea, and intense giddiness, from no matter what origin, may be sufficient to cause vomiting.

The *Causes* of vertigo may be mentioned under six headings: (*a*) Diseases of the ear, (*b*) diseases of the nervous system, (*c*) diseases of the eye, (*d*) diseases of the circulatory organs, (*e*) affections of the stomach (gastric vertigo), and (*f*) laryngeal vertigo. The first thing to do in any given case of vertigo is to decide whether it be accompanied by any defect of hearing or not, as being the commonest cause.

(*a*) Aural Vertigo.—Any disease of the external meatus, Eustachian tube, or middle ear, which is accompanied by alteration of pressure on the fenestra ovale, is usually accompanied by giddiness, and almost invariably by defective hearing. The deafness, which may perhaps be slight, may be either *obstructive deafness*, such as arises in chronic middle ear catarrh, or *nerve deafness*, such as that in

labyrinthine disease. These conditions are mentioned as the commonest in the two classes named, but there are many other less common forms of obstructive deafness and nerve deafness which are attended by vertigo (§ 618*a*).

(*b*) Diseases of the Nervous System.—(1) In *epilepsy*, vertigo often constitutes the aura or warning of convulsive attacks (grand mal) and the whole phenomenon in slight attacks of minor epilepsy (petit mal). It may be known by being invariably accompanied by a temporary interruption of consciousness, however slight the attack, and by a history of similar attacks dating from about fourteen to twenty years of age. (2) *Hysteria*, *neurasthenia*, or any exhausted or depressed condition of the nervous system, such as that produced by drugs, alcohol, or mental strain, may be accompanied by giddiness. Hysterical vertigo generally comes on only when the patient is startled or frightened. Neurasthenic vertigo is usually apt to come on when the patient goes out of doors. (3) *Various intracranial lesions* in different situations may give rise to giddiness. Intracranial tumours in general, but especially those affecting the cortex, cause vertigo, and are usually also accompanied by headache, vomiting, optic neuritis, and perhaps convulsions, together with paralysis of some of the cranial nerves. Vertigo is a very marked feature in lesions of the *cerebellum* or its peduncles, and here giddiness and a reeling gait are often the leading and sometimes the only symptoms. (4) *Disseminated sclerosis* is attended in three-fourths of the cases (Charcot) by vertigo. A disease has been described under the name *endemic paralytic vertigo*, occurring in Switzerland and Japan, characterised by paroxysmal vertigo and paralysis.

(*c*) In regard to Circulatory Disorders (1) giddiness constitutes the first stage of syncope, which may stop short at vertigo. In this way any condition of debility, or poverty of blood, anæmia, convalescence, or exhausted states of the system, may give rise to giddiness. Anæmia is perhaps the commonest cause of vertigo in the young. (2) Any *cardiac weakness* or degeneration, or disease of the coronary arteries, may give rise to vertigo, especially among the aged. Excessive smoking, by acting as a cardiac depressant, may act in the same way. (3) *Arterial disease* is attended by vertigo, which even more than the preceding is characterised by coming on chiefly when the patient rises from a sitting or stooping posture. This Senile Vertigo, or as I have elsewhere described it,[1] *postural vertigo*, very often occurs on rising at night to pass water, or on first getting up in the morning. (4) Vertigo in the aged may also be the only indication at the time of their occurrence of the *minute hæmorrhages* or *softenings* so often found in their brains after death. (5) *High arterial tension* arising from various permanent or temporary causes, as, for example, excess of uric acid in the blood, is not infrequently attended by slight giddiness. Apart from the actual occurrence of hæmorrhage, vertigo in the aged may always be taken as a warning of some kind of cardiac or vascular failure, and the circulatory system should be carefully investigated.

(*d*) Ocular Vertigo is usually due to weakness of one or other of the ocular muscles. It is not uncommon in cases of myopia, in which the constant strain of the internal recti leads to weakness of these muscles. It may also be produced by other ocular conditions, especially in diplopia from any cause. Here the vertigo is characterised by being relieved by shutting one or both eyes. So also is the dizziness felt on climbing a height, or in a wide open space, as in the agoraphobia of neurasthenia.

(*e*) Gastric Vertigo is a variety which has been described by several observers (*e.g.*, by Trousseau), but for my own part I believe many of such cases are of circulatory origin. The fact of the close relation between the taking of food and the giddiness, to which some refer, is sufficiently explained by the effect of a full stomach upon the abdominal sympathetic or mechanical impediment to the heart. Disorder of the liver is also credited with producing giddiness.

(*f*) A Laryngeal Vertigo has been described in which the patient gets giddy, and may fall on attempting to cough.

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[1] *British Medical Journal*, January 23, 1897, and Transactions of the Pathological Society of London, 1904.

The *Treatment* of vertigo must be directed to the cause, but as a palliative measure the bromides are of great value.

Disordered Subjective Sensations of many kinds may be complained of in diseases of the nervous system.

a. Subjective sensations referable to the *extremities*, such as numbness, tingling, etc., which will be mentioned under peripheral neuritis and disorders of sensation.

b. Subjective sensations referable to the *special senses*, smell, sight, hearing, and taste, which will be mentioned under diseases of the cranial nerves.

c. Subjective sensations referable to the *mind*, hallucinations, delusions, etc., which are dealt with in mental disorders.

d. Subjective sensations which affect the *equilibrium* are mostly included under the term vertigo or giddiness (*vide supra*).

PART B. CLINICAL INVESTIGATION.

§ **512.** The **Method of Examining** a nerve case differs somewhat from that in other departments of medicine, partly on account of the inaccessibility of the nervous system to direct examination, and partly owing to the widespread effects of its diseases. It is, however, not difficult provided the beginner adopts a fixed order of examination. It requires more time, but if the reader studies carefully what follows, he should find no more difficulty in diagnosing diseases of the nervous system than those of the heart or stomach, especially if he remembers that there are *two problems* in the diagnosis of *nervous cases*, which should be solved in this order : first, to LOCALISE the lesion (is it generalised, or in the brain, the cord, or the nerves ?) ; and, secondly, to ascertain what is the NATURE of the lesion. The following table represents a scheme which may be adopted for a systematic and complete investigation, taking first that nervous function to which the patient's symptoms are mainly referable :

First, examine the leading symptom or sign.

Secondly, the history of present illness, the previous and family histories.

The **History** of the case is of considerable importance in diseases of the nervous system, and must be patiently and thoroughly investigated.

The *history of the present illness* needs most careful inquiry. Many nervous symptoms are vague, or what the patient regards as unimportant. Sometimes the onset is acute, but far more frequently it is most insidious and chronic. Paraplegia and many other diseases run a very prolonged and changing course, and the physician who sees a case three or four years after its onset may be wholly without reliable data on which to found a diagnosis as to the nature of the lesion, other than those which the history reveals. The exact dates (1) of ceasing work, and (2) taking to bed are most important.

The *previous history* may reveal lead or other metallic poisoning, gout, tuberculosis, syphilis, or other predisposing causes of nervous affections. Alcohol and syphilis play a prominent part in the etiology of nearly all diseases of the nervous system. The toxins of diphtheria and influenza among the infective fevers seem to be the most inimical of the nervous system. Traumatism, mental and emotional strains often play a part in the etiology. The influence of sexual abuses is considerable, but is often, in my belief, exaggerated. Some diseases, like hysteria and migraine, are recurrent throughout life, and the history of previous attacks is a most important aid to their diagnosis.

In the *family history* a neuropathic diathesis may be revealed by ancestors and relatives having suffered from some nervous disease. The more important inherited diseases are epilepsy, hysteria, mental disease, migraine, and other vasomotor conditions. Consanguineous marriages intensify this diathesis.

Thirdly, proceed to the examination of the—

I. General symptoms and mental attitude ; the temperature in certain cases ; pain ; any defect in the skull.
II. Muscular system—weakness, walk, spasm, tremor, atrophy.
III. The deep and superficial reflexes.
IV. Electrical reactions.
V. Special senses and cranial nerves.
VI. Cutaneous sensation.
VII. Organic reflexes ; trophoneuroses ; the sympathetic system and the angioneuroses.

§ **513. I. General Symptoms.**—The majority of cases met with in private practice and out-patient work belong to the **generalised neuroses,** where the symptoms consist of vague pains or bodily discomfort, nervousness, restlessness, insomnia, etc. In many of such cases an *examination of all the organs and functions of the body* will frequently reveal some defect of these organs or functions to which the nervous condition is secondary. The neurologist needs to be a skilled general physician.

The **mind** of the patient plays a very important part in all disorders of the nervous system. We have to judge how much of the malady exists in the patient's mind, how far we can rely on his description of his symptoms, even apart from any mental or moral alienation which may exist. Careful observation during our investigation of his medical history may teach us much concerning a patient's mental condition, and some of the chief points to investigate are sleep, dreams, memory, intelligence, reasoning power, decision, attention, moral and ethical standards, delusions, hallucinations. In judging a person's mental condition or ability, one must be a well-read man of the world first and a physician afterwards. This was what Sydenham meant when he replied to a gentleman who asked what books his son should read as a preparation for the medical profession—" Let him read ' Don Quixote '; that's a very good book." Common sense and tact, as well as ability and experience, are indispensable. It is, moreover, necessary to enter sympathetically into the feelings and thoughts, mode of life, and mental attitude of your patient before he will favour you with his entire confidence.

Pyrexia is absent in chronic diseases of the nervous system. It ushers in Infantile Paralysis, and attends Meningitis. In cerebral abscess pyrexia may be absent, excepting just at the outset. Practically, persistent fever in a case of nervous disease indicates some inflammation of the cerebro-spinal meninges.

Pain and its method of investigation are mentioned under Neuralgia (§ 604), and in various other parts of this work.

The investigation of the **skull** is considered in §§ 13 and 624.

§ **514.** The **Muscular System** gives some of the most reliable evidences of disease of the nervous system. The muscles may be affected in four ways—

(*a*) WEAKNESS or paralysis.
(*b*) Alteration of GAIT, or INCO-ORDINATION.
(*c*) Muscular SPASM, tonic or clonic, or TREMOR.
(*d*) ATROPHY.

There are two other points which are specially related to muscular defects—the condition of the DEEP REFLEXES, and the ELECTRICAL REACTIONS of the muscles involved.

(*a*) Is there any **loss of power**? Can the patient walk? Can he sit up in bed? Can he move each of his limbs as a whole? Is the motor weakness localised to a few muscles? These are all points to investigate. The *degree* and exact *position* of the weakness (which enables us to localise the lesion in the nervous system) should then be investigated. Paralysis is a total, paresis a partial, loss of power. In the case of the handgrip the degree of weakness can be measured by a dynamometer, but in other instances we can only roughly estimate the degree of weakness of a muscle or group of muscles by the strength of passive resistance on the part of the operator required to prevent a certain movement.

HEMIPLEGIA is paralysis of one side of the body; PARAPLEGIA, paralysis of both legs; MONOPLEGIA, paralysis of one limb (crural of one leg, brachial of one arm); DIPLEGIA, paralysis of both sides of the body; BRACHIAL DIPLEGIA, paralysis of both arms; DIPLEGIA FACIALIS, of both sides of the face. In cases of apoplexy or coma it is often difficult to test the presence of paralysis, but its existence on one side may be indicated by a greater limpness on that side—the arm when raised and allowed to drop will fall inertly—or it can only with difficulty be raised because of the paralytic rigidity on the paralysed side. Sometimes an *individual muscle* or a *group of muscles* is affected, and a knowledge of the action of muscles enables us to decide which is involved. Go patiently through the movements of a joint, or those performed by individual muscles, *offering passive resistance* to each movement in turn; this reveals the position and the degree of the defect. The big joints are capable of six movements—flexion, extension, adduction, abduction, rotation, and circumduction; the smaller joints only the first two or the first four. The action and nerve supply of the various muscles are given under plexus and single nerve paralysis (§ 569).

(*b*) Is there any **alteration of gait** or **inco-ordination**? Note should always be made whether a patient can or cannot walk, and a patient should, if possible, be made to walk before us. The *gait* in locomotor ataxy, paralysis agitans, spastic paraplegia, and many other affections, is very characterstic (§ 577).

INCO-ORDINATION is a defective co-operation of the different muscles involved in a particular movement of a limb, unaccompanied, it may be, by any loss of muscular power. In the *legs* inco-ordination is apparent by an exaggeration of the normal movements (as in locomotor ataxy), or an inability to balance while walking (as in cerebellar tumour). The patient may be asked to walk along the edge of the carpet. If the patient is in bed ask him to follow your finger round in a circle

with his big toe, or to touch, with his eyes closed, the dorsum of one foot with the big toe of the other. Another test is to ask him to stand with heels together and the eyes shut, and notice if he stands steadily or sways about (ROMBERG'S SIGN). A very delicate test of the same kind is to ask him to balance himself on tiptoe, with knees bent and eyes closed. To test the *upper extremities* ask him to thread a needle, or (with eyes shut) bring his two forefingers tip to tip in front of him, or to touch the tip of his nose. Co-ordinated muscular movement depends upon the integrity of the muscle sense, the vision, and the cerebellar control. When the eyes are closed or bandaged, vision is eliminated.

Muscle Sense is the power by which the patient appreciates or judges the state of contraction of a muscle. It is an extremely delicate sense, for it is mainly by the muscle sense of the ocular muscles that we judge distance. In tabes dorsalis this sense is defective. It is tested in two ways : (i.) The *sense of judging weight* is tested in the arm by placing objects of the same size and configuration, but of different weights, in the patient's hand. A match-box with coins, and one with cotton wool inside, or balls of the same size but different weights may be used, or objects can be placed in a handkerchief and slung on to the hand or foot. Normally a healthy person can detect a difference of one-seventeenth between two weights. (ii.) The *sense of position* of a limb is tested by bending a joint about in various positions, and finally asking the patient in what position you have left the fingers or limb—bent or straight—or asking him to put the other hand or leg into the same position. The operator must cease to touch the skin, and the finger or toe must not be in contact with any other member or object, otherwise the patient's tactile sense comes into play. Another rough test is to tell the patient with his eyes closed to touch the tip of his nose with the tip of his first finger.

(*c*) Is there any **spasm, tonic** or **clonic,** or **tremor** ; if so, note its kind, degree, and distribution. Tonic spasm or rigidity is a continuous muscular contraction ; clonic spasm or tremor is an intermittent muscular contraction. Smaller, more rapid or vibratory movements are known as *tremors.*

A tonic rigidity is obvious on attempting to bend the limb ; it may be generalised, as in the case of tetanus, or localised to the paralysed limbs in hemiplegia and paraplegia (owing to descending sclerosis). Early and late rigidity are referred to under hemiplegia.

Clonic spasms and *tremors* can generally be rendered more obvious by the patient holding up the affected member. Some tremors are only present when the affected muscles are in action (intention tremor)—*e.g.*, those of disseminated sclerosis.

Athetosis is a peculiar condition of slow mobile spasm intermediate between tonic and clonic spasm.

Convulsions are violent clonic spasms. It is important to ascertain first the point at which the convulsions started or predominated, or whether they were generalised ; secondly, whether the patient was unconscious or not ; thirdly, whether the fit was preceded by a warning ; fourthly, what was his condition after the fit ; fifthly, whether there was any involuntary evacuation of motions or urine, or biting of the tongue ; sixthly, whether the patient has had any previous attacks of the same or a different kind.

Hypertonia or hypertonicity is increase in the tone of all the muscles hardly amounting to tonic spasm ; *hypotonia* is a diminution of the muscular tone. *Kernig's sign*, which is found in 85 per cent. of cases of acute cerebro-spinal meningitis, is an evidence of hypertonicity. It consists of a strong contraction of the hamstring muscles after they have been stretched. To elicit it with the patient in the recumbent posture, bring the thigh to a right angle with the abdomen ; then on trying to extend the leg on the thigh strong contraction of the hamstrings prevents full extension.

(*d*) Is there any **muscular atrophy** ? Atrophy may be elicited roughly by pinching the muscles and finding them flabby and wasted, or by

the measuring-tape. Finer defects of muscular nutrition can only be obtained by electrical examination (§ 516).

Muscular atrophy arises from (1) *disuse;* (2) diseases of the *spinal cord* which involve the anterior horns; (3) diseases of the *peripheral nerves;* (4) diseases of the *muscles* (myopathies); and (5) diseases of the joints.

§ **515.** III. There are three kinds of **Reflexes** to be investigated—**deep, superficial,** and **organic.**

(*a*) Are the **deep reflexes** altered? When the tendon of a muscle that has been put on the stretch is struck, the muscle immediately contracts and produces a jerk of the limb. This is known as the deep or "tendon reflex." To elicit the KNEE-JERK OR PATELLAR TENDON REFLEX get the patient, if possible, to sit on the edge of a bed, table, or chair, with the legs *hanging freely,* or cross one leg over the other and let it hang as though it did not belong to him. These positions slightly stretch the quadriceps extensor and reflexly increase its tone. Now strike the patellar tendon sharply with the tip of the fingers, or the edge of a rubber shod hammer or stethoscope, and the leg will immediately jerk forwards from the sudden contraction of the quadriceps. The patient's attention may be engaged by conversation or by hooking the fingers of the two hands tightly together, and trying to pull them apart; by some observers this is called the "reinforcement" of the knee-jerk. An increase or diminution in the knee-jerk may be conveniently indicated by k.j. + or — 1, 2, or 3. The strength of the knee-jerk varies in health; it is less marked in the young and in the old. It is exaggerated in all UPPER MOTOR NEURON lesions; it may also be increased in hysterical and other functional neuro-muscular irritability. It is diminished or lost in LOWER MOTOR NEURON lesions or whenever the reflex arc is interrupted by disease, as in locomotor ataxy.

The *knee-jerk is increased* (k.j. +) (1) when the lateral columns are affected by sclerosis, after cerebral or spinal lesions (sooner or later *all upper neuron* lesions are so attended). (2) When there is increased irritability of some part of the reflex arc, as in (i.) tetanus and strychnine poisoning, or (ii.) spinal meningitis. (3) When there is defective inhibitory control from the higher centres, as in hysteria (strychninism of Charcot), or in toxæmic states, such as phthisis, typhoid, or some cases of neurasthenia.

The *knee-jerk is diminished* (k.j.—) *or absent* (1) in all lesions of the *lower motor neuron,* such as infantile paralysis and other diseases of the anterior horns, and peripheral neuritis (alcoholic, diphtheritic, etc.). (2) In certain chronic spinal lesions, notably tabes dorsalis, and in some cases of disseminated sclerosis when the grey matter is involved. (3) In certain acute lesions of the cord other than infantile paralysis; thus in myelitis affecting the lumbar enlargement, and in transverse myelitis or other complete transverse lesions of the spinal cord, the knee-jerk at first is generally, but not always, absent. (4) In primitive myopathies (idiopathic myopathy and pseudo-hypertrophic paralysis), in proportion to the loss of muscular power. (5) In cerebellar tumour, especially in tumours of the lateral lobe. (6) During coma, and also directly after the convulsive stage of epilepsy.

Most of the *superficial tendons* can be tested in the same way, though not with the same facility, the points being (1) to get the muscle to be tested relaxed by the patient, (2) gently stretch the muscle, and (3) strike its tendon. The tendo Achillis or *ankle-jerk* may be tested while the patient is kneeling on a chair with the

calf relaxed, the foot being bent forwards, and the tendon struck by the operator. The *triceps* or *elbow-jerk* is elicited by hanging the patient's elbow over your wrist, and striking the triceps tendon ; the *supinator jerk* by tapping the tendon just above the styloid process ; and the *wrist-jerk* by striking the extensor tendons when the hand is hanging loosely. The *jaw-jerk* is not present in health ; when present it can be elicited by placing one of your fingers firmly on the front of the chin, and tapping it with the other, as in percussion.

Ankle-clonus, or the clonus imparted to the calf muscles by stretching the tendo Achillis, is elicited by supporting the patient's knee with one hand and suddenly dorsiflexing the foot with the other hand, gently maintaining the pressure of your hand on the ball of the foot all the time. It is present *with organic diseases* in the same circumstances as increased knee-jerk and ankle-jerk—*i.e.*, in *upper neuron lesions.* Under the same conditions knee-clonus may be obtained by placing one finger above the patella, the patient being in a recumbent position, and percussing the finger with the other hand.

The tendon reflexes are of use not only to detect which of those two important groups of lesions is present—upper or lower motor-neuron lesions—but also to ascertain approximately the level of the cord which is involved, as may be seen from the table below.

Table showing the Spinal Segment involved in the Deep Reflexes.

Reflex.	*Spinal Segment Involved.*	*Opposite Vertebral Spines of—*
K.-J.	Second and third L.	Tenth and eleventh D. spines.
Ankle-clonus.	Third, fourth, and fifth sacral	First L.
Elbow-jerk.	Seventh cervical.	Fourth C. spine.
Supinator-jerk.	Fifth cervical.	Third C. spine.
Wrist-jerk.	Sixth cervical.	Fourth C. spine.
Jaw-jerk.	Motor nucleus of the fifth cranial nerve.	——

Myotatic Irritability consists of the too-ready contraction of a muscle when its muscular substance is struck. This is frequently seen in advanced phthisis, and other exhaustive diseases—*e.g.*, in percussing the chest. In tetany the condition is very marked, the facial muscles being thrown into contraction when struck or scratched.

(*b*) **The Superficial Reflexes.**—On stimulation of certain parts of the skin or mucous membrane with a blunt pin or the top of a penholder, a contraction of certain associated muscles takes place, as shown in table in § 558. It is a true reflex action, and can only be obtained when the afferent and efferent paths and the corresponding grey matter in the cord or brain are intact. The chief use of the superficial reflexes in medicine is to determine the locality of a disease in the spinal cord. The mode of eliciting and situation of the ganglionic centre for the plantar, gluteal, cremasteric, epigastric, abdominal, and interscapular reflexes are given in

the table just referred to. The conjunctival reflex, obtained by touching the conjunctiva, causes contraction of the orbicularis palpebrarum, and its ganglionic centres are situated in the fifth nucleus (sensory) and the seventh nucleus (motor). The palate reflex, obtained by touching the soft palate, leads to its elevation by the levator palati ; the afferent nerve is the ninth, the efferent the accessory part of the eleventh (through the vagus). The cutaneous reflexes vary considerably in different individuals. They are normally more prompt in children than adults, and in women than men, and are difficult to obtain when the skin is harsh and insensitive, as it usually is in the aged, or when there is much subcutaneous fat.

Babinski's Reflex is a modification of the plantar superficial reflex. To elicit it the patient should be in the recumbent posture, with the lower limb slightly flexed, and the sole of the foot warm and dry. The sole is gently stroked upwards by the finger-nail or a blunt pin. In health the big toe and the other toes will become flexed upon the sole ; this is the normal reaction. In Babinski's reflex there is *extension of the great toe* followed by flexion of the other toes. The stroking should not be hard enough to evoke dorsiflexion of the foot, as this obscures the big toe reflex. This reaction is only met with in *organic disease* involving the lateral column. Babinski found it was elicited best by stroking the outer side of the sole. In *functional* cases the plantar reflex, if it can be elicited, gives a *flexor* response as in health (in functional cases a diminution of the plantar reflexes with exaggerated knee-jerks is a characteristic combination), and the same obtains in peripheral neuritis, poliomyelitis, tabes, and even in intracranial tumours, provided the pyramidal tracts are not involved. In infancy, before the age of walking, an extensor (not a flexor) response is normal.

§ **516. Electrical Examination of Muscles** and the nerves which supply them is an important aid in distinguishing *lower* from *upper* motor neuron lesions, and in detecting the degree of disease or degeneration in a nerve or muscle.

The apparatus required is not complex. The chief requisite is a *faradic coil*, because the first and most important point is to ascertain the degree of faradic contraction. A simple Ruhmkorff coil, of which the secondary coil slides on to the primary, is needed. It is better if the wire of the former is not too thin, as it produces unnecessary pain. A couple of moderate sized Leclanché cells will drive it. Two wires and three electrodes are required, one large (measuring about 6 by 4 inches), a round medium-sized one, and one small (about 1 inch in diameter). The two smaller ones should be fitted with an interrupting handle.

A suitable *galvanic battery* is more expensive, for it requires at least twenty-four small Leclanché cells (capable of giving about 35 to 40 volts), a collecting board and reverser, with electrodes as before, and, to be complete, a galvanometer that has been properly tested (Fig. 153). The constant current can be used from the main with a suitable switchboard.

To *test the faradic reaction* place the large electrode in the patient's hand, on the back of the neck, or some other indifferent position, and another electrode, connected with the interrupting handle, over the motor point of the nerve or muscle to be tested. If, as frequently happens, the current is too strong for the finer degrees of difference, the operator should take the electrode in one or other of his hands, and apply his well-wetted finger to the well-wetted skin of the patient. A knowledge of the motor points of nerve and muscles is not indispensable, for a great deal may be learned by rubbing the medium-sized electrode with plenty of water well over the limb. The motor point of a muscle is near the point of entry of its nerve ; that of a nerve is generally near its most superficial part. The electrodes and the skin should be very *thoroughly* wetted with plain warm water. Some prefer salt and water, but this damages the instruments more, and does not increase the conductivity, provided the electrodes are kept clean with a little soap occasionally. The patient should be so placed in a good light that both sides of the body can be seen equally. Having thoroughly *wetted the skin on the two sides* you are about to compare, ascertain first (by gradually sliding up the secondary coil) what is the *minimum* current necessary to produce a minimum contraction of the muscle or muscles on the healthy side. Then, secondly, test the side suspected of disease

with the same amount of current, to see if the same degree of contraction is produced, or if not what strength of current is requisite.

The faradic contraction of a muscle can only be obtained through its nerve—*i.e.*, when a healthy nerve is connected with it, however healthy or unhealthy the muscle itself may be, and therefore the faradic current is an accurate test for the condition of the nerve. If the nerve is damaged or destroyed there will be a proportionate diminution or total loss of the faradic muscular reaction. This fact was discovered in 1859 by Baierlacher, who experimented on the facial nerve. *A diminution of faradic contraction signifies a lesion of some part of the lower motor neuron.*

To *test the galvanic reaction* the electrodes are placed in the same position as before. For (*a*) *quantitative* alterations, compare the two sides as before, noting

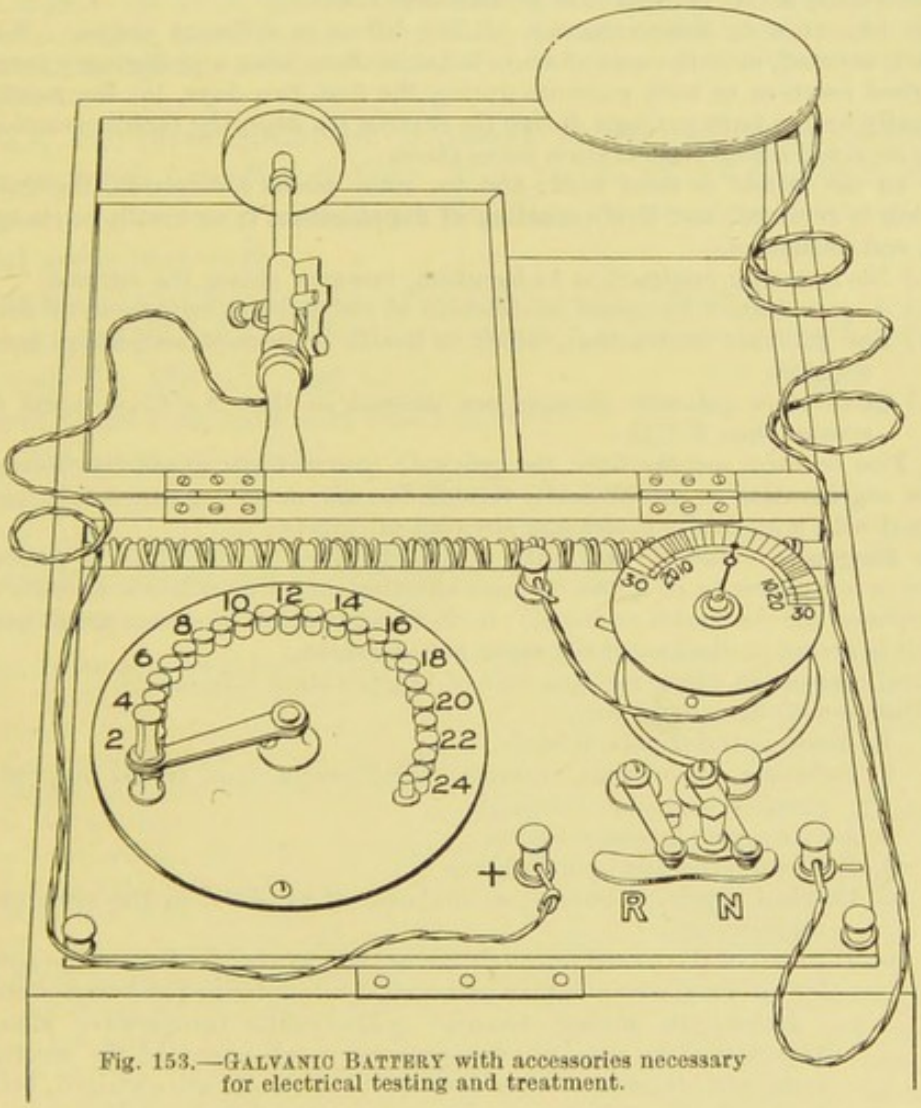

Fig. 153.—Galvanic Battery with accessories necessary for electrical testing and treatment.

what amount of current (as indicated by the number of cells used, or the number of m.a. registered by the galvanometer), is required to produce a minimal contraction on both sides when both sides have been wetted equally. (*b*) The *qualitative* alterations are a trifle more intricate. Begin with the kathode (negative pole) placed on or about the motor point under investigation. Close the current by means of the interrupting handle, and the contraction obtained is known as the Kathodal Closing Contraction (K.C.C.). The kathode or negative pole is the one connected with the zinc element in the cell. If you cannot tell which is the kathode, place the two wires in a glass of water, and a lively production of hydrogen gas comes off at the kathode ; or place both wires on a piece of wetted litmus paper,

which becomes reddened around the positive pole (anode) from the liberation of oxygen. Next convert the electrode on the patient into the anode or positive pole by means of your reverser, and repeat the process of closing the current. The resulting contraction is called the Anodal Closing Contraction (A.C.C.). These are the two important data to obtain, for normally with the same strength of current and the same degree of wetting of the skin K.C.C. is greater than A.C.C. (K.C.C. > A.C.C.), or what would amount to the same thing, a greater strength of current is required to produce A.C.C. than K.C.C.

Muscular contraction with a galvanic current is only produced at its closing or opening. Sometimes the opening contractions are tested, the normal order being **K.C.C.** > **A.C.C.** > A.O.C. > K.O.C.

Abnormally **A**.C.C. is equal to or greater than K.C.C.

The REACTION OF DEGENERATION (R.D.) differs in different stages. When a nerve is severed, or is the seat of acute inflammation, after a preliminary increased electrical reaction to both currents during the first two days, (*a*) the reaction is gradually lost to both currents *during the ensuing ten days*, the faradic reaction not being regained unless regeneration takes place.

(*b*) In the *second or third week*, and for some weeks afterwards, the galvanic reaction is restored, and Erb's **reaction of degeneration** is met with in its typical form, and consists of—

(i.) No muscular contraction to faradism, however strong the current.
(ii.) A quantitative increased contraction to the galvanic current in all forms.
(iii.) The galvanic contraction, which in health is prompt and sharp, becomes sluggish.
(iv.) Qualitative galvanic changes are present in that A.C.C. is equal to or greater than K.C.C.

(*c*.) *Two or three months later* the galvanic contractility gradually disappears (unless regeneration is established), though for one or two years A.C.C. can be obatined with a gradually increasing strength of current.

The **diagnostic** value of electricity is considerable, for the R.D. is present in greater or less degree in all severe diseases affecting the anterior horns, anterior roots or peripheral nerves (motor or mixed) ; in short, any part of the **lower motor neuron,** while it is absent in diseases of the **upper motor neuron.**

List of diseases in which the Reaction of Degeneration is found :

I. Diseases of the *anterior horns.*
   1. Acute anterior poliomyelitis.
   2. Subacute and chronic anterior poliomyelitis (*e.g.*, spinal form of progressive muscular atrophy).
   3. Amyotrophic lateral sclerosis.
   4. Syringomyelia and spinal gliosis.
   5. Cervical myelitis, transverse lumbosacral myelitis, in the arms or legs respectively.
   6. Diseases of the motor nuclei in the medulla corresponding to the anterior horns (progressive bulbar paralysis, acute inferior polio-encephalitis).

      NOTE.—In chronic anterior poliomyelitis (progressive muscular atrophy) there is a slow progressive involvement of the multipolar cells, and, therefore, irregular electrical results are obtained, because one part of a muscle may be more involved than another. In idiopathic myopathy irregular results are also obtained.

II. Diseases of the *anterior roots :*
   1. Compression from tumours and thickened membranes (*e.g.*, in syphilis, and hypertrophic cervical pachymeningitis).
   2. Compression in the intervertebral foramina in vertebral diseases (caries, tumours, fracture, luxation).

III. Severe diseases of the *peripheral nerves :*
   1. Injury or pressure from tumours, persistent thickenings, bone disease, operations, etc.
   2. Rheumatic (*e.g.*, in facial paralysis), and other toxic and infectious causes —*e.g.*, lead, alcohol, arsenic, diphtheria, and other infections.

The diagnosis of Myasthenia Gravis is facilitated by finding a characteristic faradic reaction of exhaustion. The muscles at first contract normally to the faradic current, but after a few contractions they get " tired," and will not contract even with the strongest current. There is no alteration to galvanism.

In regard to **prognosis,** whenever faradic reaction is retained, even if only slight with a very strong current, restoration of a injured or diseased nerve is possible ; if it is quite lost for a few weeks restoration is still possible ; but it if remains totally lost after several months there is but little prospect. Whenever an incomplete R.D. is present the prospect of recovery may be considered even after notable atrophy. Slight voluntary movement sometimes returns *before* the return of the electrical reactions.

The slighter the lesion the less the alteration in electrical excitability. Slight compression of a nerve enough to produce paralysis may produce but little alteration, though in nearly all cases the faradic excitability is somewhat diminished.

§ **517.** The investigation of the **Special Senses** and **Cranial Nerves** is given in detail hereafter (§ 608). The points to investigate in order summarily given are as follows (the Roman numeral refers to the cranial nerve involved) :

Symptoms referable to the nose—smell (I.) ; discharge.

Symptoms referable to the eye—vision, ocular movements, pupils, and fundi (II., III., IV., and VI.).

Taste, facial sensation, and mastication (V.).

Facial movements (VII.).

Hearing, tinnitus, or vertigo (VIII.).

Pharyngeal sensation, deglutition, muscles of palate and larynx ; also sterno-mastoid and trapezius (IX., X., XI.).

Muscular power of tongue (XII.).

§ **518. Common Sensation.**—Four kinds of common sensation are now recognised by authors—touch, pain, thermal sense, and pressure sense—and the two chief points about each of these are whether it is increased or diminished, and what the boundaries are of such alteration. To test a person's capacity to feel we must first obtain his intelligent co-operation ; honesty and good will are large factors in the investigation. His eyes should be covered, and he should be instructed to say simply " yes " immediately he perceives any sensation. Corresponding points on the opposite sides of the body should be tested if possible, and a negative test should be applied from time to time. The sensibility differs in different persons and in different parts of the body. The student should first study § 502 and the remarks on Epicritic, Protopathic, and Deep Sensibility.

(1) Cutaneous sensibility for TOUCH may be tested by the smooth head of a lady's hat-pin, the finger, the corner of the handkerchief, or if desired to altogether eliminate the sense of pressure, a small pad of cotton wool. For delicate investigations the points of compasses are sometimes used. Ascertain whether (i.) sensation is *lost* (anæsthesia) or *increased* (hyperæsthesia), and (ii.) what are the *boundaries* of such loss or increase (*e.g.*, hemi-anæsthesia or hemi-hyperæsthesia). Hysteria and locomotor ataxy are frequent causes of altered sensation. In the former, spots of an an- or hyper-æsthesia may be present, and pressure on the tender spots (hysterogenic zones) may produce fits. (iii.) Tactile sensation may be *delayed*, as in peripheral neuritis, or (iv.) *misplaced* (allocheiria) as in tabes. *Astereognosis* is a want of recognition of shapes of objects. *Atopognosis* is failure to locate a sensation properly. Both may arise from gross lesions in the post-central gyrus, and therefore may be attended by hemiparesis most marked in the facial and arm regions.

(2) Cutaneous sensibility to PAIN may be tested by the sharp end of the hat-pin

or by a faradic current. Occasionally, without loss of tactile sense, cutaneous sensibility to pain is lost (analgesia), or the reverse (hyperalgesia).

(3) Cutaneous sensibility to TEMPERATURE (thermal sense) is tested by test tubes of hot and cold water, or hot and cold silver spoons, the patient being asked to identify them. Loss of the thermal sense is a constant feature of syringomyelia.

(4) DEEP SENSIBILITY to PRESSURE and the torsion of JOINTS is a sense apart from the foregoing (see § 502). Both are increased in peripheral neuritis. Arthritic sensibility to torsion and sensibility of the mucous membranes are lost in some cases of hysteria.

MUSCLE-SENSE is one of the functions of co-ordination (see §§ 502, 503, and 578). It is tested by asking the patient, with his eyes covered, to say whether a certain joint is bent or extended, or to say which of two objects, like a sovereign and a shilling, is the heavier.

Paræsthesia (perverted sensation) and various subjective sensations are described in the disorders of sensation. Aura is a term applied to a morbid sensation or " warning " which precedes epileptic fits.

§ **519. Organic Reflexes.**—Defæcation, micturition, deglutition, and respiration are sometimes spoken of as the organic reflexes, because they consist of the contraction of involuntary muscular tissue, resulting from the automatic stimulation of a mucous membrane. As regards *defæcation* and *micturition*, the patient should be questioned as to the existence of retention or incontinence, particularly in cases of paraplegia. The presence of such symptoms indicates direct or indirect involvement of the lumbar enlargement, and frequently occurs in myelitis and certain system lesions, such as tabes dorsalis.

§ **520. Trophic Changes.**—The nervous system controls the nutrition, not only of the muscles, but of all the organs and tissues of the body. The trophic change which results may be revealed in a variety of ways.

The *skin* may become " glossy "—*i.e.*, smooth, thin, and atrophied. Erythema, ulceration (as in the perforating ulcer of tabes) or sloughing (*e.g.*, bedsores) may appear, vesicles may develop when an irritative lesion of a ganglion (*e.g.*, of the fifth cranial nerve) is present, and painless whitlows on the fingers, as in syringomyelia.

The *bones* may undergo absorption or arrested development (as in poliomyelitis).

The *joints* may be the seat of painless effusion and absorption of the bone ends (as in tabetic arthropathy).

§ **521.** Disorders of the **Vaso-motor** and **Sympathetic Systems** have hitherto been but little studied. The former gives rise to angio-neurotic œdema, and various disorders of circulation and nutrition. The sympathetic system controls all the involuntary muscular tissue in the body—intestinal, pulmonary, and cardio-vascular—and therefore effects of its disease (unrecognised perhaps) may occur in any organ or tissue of the body. The clearest recognised instance is paralysis of the cervical sympathetic ganglion (§ 612). In the author's view hysteria is mainly a disorder of the sympathetic system.

PART C. DISEASES OF THE NERVOUS SYSTEM : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **522. Routine Procedure and Classification.**—*First*, as on previous occasions, we ascertain and investigate the patient's LEADING SYMPTOM, probably one of those mentioned in Parts A or B.

Secondly, interrogate the patient as to the HISTORY OF THE ILLNESS, his PREVIOUS and FAMILY HISTORIES, all of which require special care in nervous diseases (as mentioned in § 512).

Thirdly, the PHYSICAL EXAMINATION has been dealt with systematically in Part B, but for a **routine examination** in ordinary cases it is

sometimes convenient to adopt a regional method, beginning at the head and proceeding downwards.

(*a*) THE HEAD.—Inquire as to intelligence, sleep, pain, "attacks," or head sensations of any kind.

Eyes—vision, abnormality of pupils, squint, ptosis, nystagmus, ophthalmoscopic examination.

Face—notice any defect of speech, tremor of the lips, or immobility. Test the muscles by such directions as "Show me your teeth," "Screw up your eyes," "Put out your tongue."

Hearing and other *cranial nerves*, as may be necessary, in numerical order.

(*b*) UPPER LIMBS.—Examine the state of the muscles, and compare the force of the grasp of the two sides.

Elicit any *tremor* or involuntary movement by extending the hands and fingers; and direct the patient to touch the tip of his nose with his forefinger, repeating the performance with eyes closed (ataxy).

Test the supinator and triceps *reflexes*.

(*c*) LOWER LIMBS.—Examine the muscles for paralysis, rigidity, flaccidity, or wasting.

Walking—Notice any peculiarity in the attitude or the *gait*; can the patient stand with heels together and eyes closed (Romberg's test)?

Examine the *knee-jerks*, test for *ankle-clonus*, etc.

(*d*) Test for abnormalities of COMMON SENSATION (touch, pain, and temperature).

Inquire as to state of the SPHINCTERS.

Remember, once more, that there are two steps in the diagnosis of diseases of the nervous system, which, in order, are—first, **localisation**, namely, what part of the nervous system is the seat of disease? secondly, what is the **nature** of the disease in that locality?

If the symptoms point to some **generalised neurosis**, turn first to Group I. (below).
If to defect of **consciousness** or the **mind** (Group II.) . . . . § 529
If there is definite **pyrexia** (Group III.) . . . . . § 547
If the symptoms relate to the **muscular system** (Group IV.)—
Paralysis . . . . . § 552
Inco-ordination or defect of gait . . . . . § 577
Rigidity . . . . . § 582
Tremor . . . . . § 589
Convulsions . . . . . § 597
Muscular atrophy . . . . . § 600
If to some **sensory** or **painful** disorder (Group V.) . . . . . § 604
If to the **special senses** or cranial nerves (Group VI.) . . . . § 608
If there is some deformity of the **skull** (Group VII.) . . . . § 624

GROUP I. GENERALISED NEUROSES.

The word "neurosis" connotes a functional disorder of the nervous system, and by generalised neuroses are meant those which present generalised symptoms. The symptoms presented in this group of disorders are widespread and manifold, but nine-tenths of *the patients complain of nervousness*. The remarkable resemblance of chronic alcoholism, morphinism, and toxic neurasthenia in their clinical features to the other members of this group, suggests that many of the cases here met with may be dependent on some disorder of the blood.

I. Neurasthenia.
II. Hysteria.
III. Alcoholism.
IV. Morphinism and other drug habits.
V. Hypochondriasis.
VI. Collapse.

§ **523. Neurasthenia** is an irritable weakness of the nervous system, which may arise from a great variety of causes, and may result in many and various symptoms of nervous, mental, and bodily inefficiency.

The *Symptoms* are of a subjective order. The patient may come for many different reasons: (1) A feeling of "weakness and nervousness" is one of the most usual complaints, or he may state that he is easily tired, easily startled, easily upset. The physical debility or disability is sometimes less marked than the mental, but it is often severe, and may be sufficient to confine the patient to bed. Slight anæmia and loss of weight may also be present. There are no physical signs unless the occasional presence of exaggerated knee-jerks, retraction of the fields of vision, or dilatation of the pupils may be so considered. It is convenient to describe a cerebral, cerebro-spinal, and spinal type, according to the prevailing symptoms. (2) Cerebral or mental symptoms are always present and generally predominate. Everything the patient has to do is a trouble to him, and a source of worry, and sometimes the simplest mental work, such as adding up a column of figures, is impossible. The sleep is disturbed by dreams, or there is insomnia, or "startings" in the sleep. All the special senses are easily tired, and sometimes the patient is quite unable to read. The pupils are usually dilated, and reaction to light and accommodation is sluggish. The memory and the power of concentrating the attention are defective. Some patients are *irritable*, egotistical, and exacting; others *gloomy* and melancholic, constantly on the grumble. Anxiety and morbid dreads form a prominent feature, sometimes indefinite—the fear of some unknown evil—sometimes definite, such as agarophobia (fright when being in an open space), claustrophobia (fear of going into churches or other buildings), monophobia (fear of being alone), antheropophobia (dread of society or of people). Sometimes the dreads take the form of paroxysmal panics or terrors, which pass

off as suddenly as they come, and I have seen several of such cases pass on to melancholia and dementia.[1] There is no doubt that the miseries they suffer are very real, and not a few patients, particularly cases of the gloomy type—those who go wearily on with their work, saying but little of what they feel, who have probably never actually threatened suicide—seek relief by terminating their lives. Other patients drift into a chronic and incurable hypochondriasis. (3) Spinal and musculo-sensory symptoms are also present in greater or less degree—restlessness and jerking of the limbs, weariness on the least exertion, vague pains in the back and limbs, generalised tenderness or a hypersensitive condition of the whole body, neuralgic pains and tender spots. Very generally there is fine muscular tremor, but never localised paralysis or hemi-anæsthesia, as in hysteria, and never absent knee-jerks, as in tabes dorsalis. Absence of knee-jerks excludes the diagnosis of neurasthenia. Spermatorrhœa, nocturnal emissions, or the discharge of glairy fluid at the stool, and sexual disability are met with. (4) There are generally symptoms which may be aptly attributed to disturbance of the vaso-motor sympathetic system—*e.g.*, long-drawn sighs, causeless palpitation, attacks of flushing, followed by shivering, a sensation of " pins and needles " in the limbs, cold hands and feet. Palpitation of the heart and various " throbbings," giddiness, and faint feelings are also met with. It is in the presence of these sympathetic symptoms and attacks that neurasthenia overlaps hysteria. (5) According to some authors, gastric symptoms form an essential part of the symptoms of neurasthenia, and this is often, but not always, true. In a great many cases gastro-intestinal disorder is the cause and accompaniment of neurasthenia, and in others the asthenia of the nervous system has given rise to asthenia of the stomach (gastric myasthenia).

In the *Diagnosis* the very vagueness of the symptoms is an aid. Neurasthenia is certainly not the same disease as *hysteria* (see table), nor is neurasthenia a new name for hysteria, as some suppose, though they overlap in some respects. Hysterical phenomena are paroxysmal and recurrent, with intervals of health, throughout life ; in neurasthenia the symptoms are more or less continuous, occur almost equally in either sex, and are liable to come on for the first time at any age.

The diagnosis from early *Tabes Dorsalis* and *General Paralysis of the Insane* is sometimes extremely difficult. In neurasthenia the knee-jerks are never, in my experience, absent, and the typical Argyll-Robertson pupil never present. For the diagnosis of general paralysis of the insane, chief reliance has to be placed on the typical articulation and the tremor of the lips and tongue.

Prognosis.—Neurasthenia is essentially a chronic disease, and leads to a great deal of misery, but is never fatal, excepting by suicide or

[1] "Clinical Lectures on Neurasthenia," 4th edition, J. H. Glaisher and Co., London, 1908.

complications. Some hold that neurasthenia is incurable. The author, however, believes it to be mostly curable, provided a careful investigation be made, leading to the discovery of the cause or causes in operation. It may run on for years, especially if, as is so frequently the case, dyspepsia, colitis, or some other chronic cause is in operation. Its prognosis depends chiefly on (i.) its previous duration, and (ii.) the removability of the cause, and (iii.) the age of the patient, being more favourable in younger persons.

TABLE OF DIAGNOSIS.

	Neurasthenia.	*Hysteria.*	*Hypochondriasis.*
Sex.	Both sexes almost equally.	Female sex almost exclusively.	Males chiefly affected.
Age.	Any age—young men slightly predisposed.	Definite manifestations of some kind appear for the first time practically always before twenty-five, generally between fifteen and twenty.	Rare under thirty; predisposition from thirty to fifty.
Causes.	Produced by overwork; gastro-intestinal and other causes of malnutrition, toxæmia, defective metabolism: occasionally shock.	Subjects of the hysterical diathesis are liable to hysterical attacks or symptoms *throughout life*. The determining cause of active hysterical manifestations always an emotional upset.	Solitary, sedentary life. prolonged gastro-intestinal troubles.
Onset.	Starts gradually and runs a fairly even course.	Onset sudden, generally with an attack of some kind; all phenomena vary from hour to hour and day to day.	Starts gradually, and runs an even and intractable course of indefinite duration.
Mind.	Mental exhaustion and inability to think, study, or do work; memory deficient; intellect clouded for business; always tired; temper irritable; depression rather than sadness; sometimes suicidal.	Wayward, impulsive, and emotional; fond of gaiety and amusement; usually joyous, but laughter and tears alternate with great rapidity; memory and intellect sometimes brilliant, rarely deficient; no tendency to suicide.	Introspective habit; observing accessible organs and secretions; habitual sadness; no taste for amusement; patient tries an endless succession of remedies and doctors; always striving for a cure.
Symptoms.	Chronic weakness and nervousness; attacks of vague sensations about the head; convulsions never; gastro-intestinal trouble of some kind in 75 per cent.	Symptoms paroxysmal; seizures of different kinds frequent; flush readily; attacks of globus and syncope frequent; convulsive attacks in nearly half the cases.	No seizures of any kind; runs an even course.
Result.	May last a long time, but by appropriate measures it is gradually CURABLE.	Active manifestations disappear suddenly and unexpectedly; very apt to recur, and therefore only TEMPORARILY CURABLE.	Once established hypochondriasis is impossible to eradicate: progressive and INCURABLE.

Causation.—Prior to 1898 neurasthenia was regarded as of purely nerve origin—exhaustion and heredity playing the leading parts. In that year the author showed that the disease was mostly dependent on a toxæmia of some kind,[1] and he classified the causes of neurasthenia into four groups, compound causes being very frequently in operation : (1) *Toxæmic* causes, which include dyspepsia, colitis, and other gastro-intestinal disorders, such as chronic constipation, dilatation or kinking of the colon, bad teeth or pyorrhœa alveolaris, and various other infective foci. Abuse of alcohol, morphia, or cocaine, and Graves' disease, may also be included here. (2) *Malnutrition* causes, such as post-influenzal (a specially potent cause) and other post-febrile and debilitating conditions, and deficient or defective food. (3) *Fatigue* and over-functioning are factors which include prolonged overwork, worry, anxiety, deficient or defective sleep, severe prolonged pain and excessive venery. (4) *Emotional* and *traumatic* causes—grief, shock (mental or bodily), railway and other accidents, in which the neurasthenia need not supervene until a few weeks after the shock. Injury may produce either " traumatic neurasthenia " or " railway spine " (see Paraplegia). My statistics (*loc. cit.*) show that some underlying gastro-intestinal cause is present in 75 per cent. of the cases. An hereditary predisposition plays a part in the etiology—though not, in my belief, as prominent a one as some hold—either by reason of a neuropathic predisposition in the progenitors, or alcoholism or tuberculosis and other debilitating agencies in the parents. In regard to age, none are exempt, but neurasthenia is commoner in young and middle-aged adults than in advanced age or in children ; and 61 per cent. of the cases under my care were males. A sedentary indoor life and the unhealthy atmosphere of town life appear to predispose, and the rush and strain of modern civilisation favours the occurrence of the disease.

The *Treatment* resolves itself briefly into : (1) The alleviation of the distressing symptoms as far as may be. Bromides are particularly useful in this respect ; morphia and other sedatives require the greatest care. Alcohol and tobacco should be avoided. (2) The removal of, or compensation for, the above causes. Dyspepsia is in evidence in by far the largest number of neurasthenics in out-patient work, and it is surprising how efficacious an alkaline gentian mixture is in the majority of cases. Aperients and intestinal antiseptics are valuable. Regular outdoor exercise is useful. (3) Nerve tonics with hygienic and educational measures conducted with sympathy and encouragement will do much for those in whom hereditary predisposition is strong. Among nerve tonics strychnine is very useful, and arsenic, phosphorus, damiana, phosphates, glycerophosphates, malt, and cod-liver oil may be tried. Turkish and warm baths allay irritability. Cold baths and

[1] " Lectures on Neurasthenia," Glaisher and Co., London, 1st Edition.

judicious hydrotherapy, various forms of electricity, and a regulated diet have all done service in these cases. (4) Complete physiological rest of the nervous system is of the greatest value, but I cannot recommend Weir-Mitchell treatment—at least, in its complete form. Sea voyages are beneficial. (5) In the most intractable cases operations on the colon, as suggested by Mr. W. A. Lane, may be considered.

§ **524. Hysteria** is a word derived from ὑστερον, the womb, in the mistaken belief which was prevalent in the Middle Ages that the disorder, which is almost confined to the female sex, arose within that organ. The disorder may be provisionally defined as a condition of instability of all the emotional, vaso-motor, and all the reflex nervous functions, with a tendency to the development from time to time *throughout life* of many different forms of nervous seizure, and of various motor and sensory disorders closely resembling organic diseases of the nervous system, never leading to a fatal issue. In the author's view, nearly all hysterical disorders are dependent directly or indirectly on an inherent defect of the sympathetic system.[1]

Symptoms.—There is no particular form of facies uniformly attaching to hysteria, but the hysterical disposition is essentially one of unstable equilibrium of the emotional and other faculties of the mind. These patients are easily aroused to violent expressions of feeling, hasty judgments, impulsive actions, and to passionate exhibitions of various kinds. There is a hypersensitiveness to all forms of pain, and a tendency to neuralgia, the favourite seats for which are just below the left breast, or on one side of the head (clavus). The hysterical diathesis in an individual may also be suspected by the presence or a history of any of the following symptoms, which may with propriety be called the "hysterical stigmata."[2] (1) *Flushings* of the face and other parts with or without provocation. Sudden pallor and other vaso-motor skin phenomena are also very frequent in hysterical subjects.[3] (2) *Nervous attacks* of some kind are sure to occur in hysterical subjects sooner or later—"hysterics" (crying and laughing), nervous faints, etc. They are specially apt to occur at the catamenial period, or after some "contrariment" emotional disturbance. (3) "*Globus*" is a sensation as of a ball in the throat, or a sense of choking or suffocation. Flatulence is a frequent accompaniment of globus, and a severe attack of globus with prostration is often terminated by copious windy eructations. (4) "*Ovarie*" or the hysterical ovarian phenomenon, consists of a tenderness on pressure on either inguinal region, which produces an indescribable feeling rising up towards the heart and throat.

[1] "Lectures on Hysteria and Allied Vaso-Motor Disorders," Glaisher and Co., London, 1909.

[2] A stigma etymologically signifies a permanent or abiding mark or sign by which something may be recognised.

[3] See a clinical lecture on the "Skin Symptoms of Hysteria," the *Lancet*, January 30, 1904; and "Lectures on Hysteria."

Pressure in this, the " ovarian " region, may determine some kind of attack. This phenomenon is in no way dependent on the ovary, but as I have elsewhere shown,[1] is specially related to the ilio-hypogastric and ilio-inguinal nerves. Similar hyperæsthetic or hysterogenic zones may exist elsewhere. (5) *Patches of anæsthesia or hyperæsthesia* may exist almost unknown to the patient. The anæsthesia may occupy one half of the body and involve the special senses on that side.

Hysterical disorders may affect any part of the body, closely simulating organic disease. These are described under their suitable sections, and a brief summary only is given here. (1) Disorders of motion—paralysis of the voluntary muscles, hemiplegia, monoplegia, paraplegia, rarely if ever paralysis of the face ; tremors and convulsions, tonic and clonic spasm of one or several limbs, or involving the whole body. (2) The involuntary muscles may also be affected by paralysis or spasm—adductor spasm or paralysis of the vocal cords, aphonia, dysphagia, hiccough, cough, dyspnœa, borborygmi, phantom tumour, vomiting without nausea. (3) Anæsthesia may affect one limb or half of the body ; hyperæsthesia, especially of the spine, and various neuralgiæ are common. (4) Joint affections, with pain and stiffness, may occur. (5) Hyperpyrexia is said by some to occur, with or without local manifestations of hysteria. (6) The special senses may be affected —amaurosis, hemianopsia, retraction of the field of vision, deafness, disturbance of taste and smell. (7) Finally, there may be mental disorder—trance, catalepsy, hallucinations and delusions—especially after convulsive attacks.

The *Diagnosis* of hysteria from neurasthenia has been considered in § 523. The salient features of hysteria are its limitation practically to the female sex, the *paroxysmal occurrence* of all its symptoms, and a previous history of similar symptoms. The diagnosis of the numerous hysterical phenomena will be dealt with under the various disorders which they most resemble.

Prognosis.—The hysterical diathesis lasts throughout the lifetime of an individual, modified from time to time by their state of health and surrounding circumstances ; but once hysterical, always liable to develop hysterical manifestations. The disease never terminates fatally of itself, but it often renders the life of an individual a misery to herself and those around her.

Etiology.—Hysteria is practically confined to the female sex ; it occasionally presents itself in the male sex, but only to the extent of about 2 per cent. Heredity is a potent factor, and can be traced in at least 75 or 80 per cent. ; the influence is transmitted particularly through the mother. In many cases there is a family history of one or other of the diseases dealt with in this group (Group I.). Faulty education or a life of self-indulgence, or any mode of existence which leads to

[1] The *Lancet*, July 20, 1901, p. 122.

introspection, a diminution or abolition of the control normally exercised by the will, undoubtedly foster the evolution of the diathesis. Faulty hygienic and physical development in childhood also favour the occurrence of hysterical phenomena in after life. The favourite ages when evidences of the hysterical diathesis are most manifest, and when the various phenomena are most likely to supervene, are soon after the evolution and at the involution of a woman's sexual life. The determining cause of all hysterical manifestations is some emotional shock, trivial or severe. No anatomical or histological lesions have yet been discovered.

Treatment.—Educational treatment is very important in these cases, and as there are but few mothers possessing the combination of judgment, firmness, tact, and kindness which is necessary in the treatment of these, their own daughters, subjects of hysteria should be entrusted to others. Some regular occupation and interest in life is another means by which the temperament may become controlled. Nothing to do or a frivolous kind of existence is calculated to foster and develop the diathesis. Matrimony thus becomes a valuable adjuvant, because it gives to a young woman occupation, interests, and responsibilities outside herself. Briquet[1] showed conclusively that hysteria had no causal relation to ungratified sexual passions. If the manifestations of the diathesis are sufficiently pronounced, a course of treatment is indicated which comprises (1) removal from the conditions under which the disease is fostered, (2) isolation from sympathetic friends, (3) over-feeding with milk and other easily assimilable foods, and (4) massage, which enables the patient to take and assimilate more food. These four measures constitute the Charcot or Weir-Mitchell method of treatment. The treatment for the nervous attacks, when they arise, consists of the sudden application of cold water to the face, the faradic battery to the limbs, and the internal administration of asafœtida, valerian, and spirits of ether, or chloroform. A hypodermic injection of apomorphine effectually terminates hysterical convulsions and other violent seizures.

§ 525. **Hypochondriasis** is a morbid condition of the nervous system allied to neurasthenia on the one hand, and melancholia on the other. It is an introspective, melancholic, or pessimistic habit of the mind, in which the patient believes, without cause, that he is the subject of one or more serious bodily disorders. He is gloomy, wrapped up in himself, but talkative; a slight pain in the stomach is certainly cancer; a slight palpitation is regarded as mortal cardiac disorder; or the testicles hang too low, and therefore he will certainly become impotent for life. Yet, in spite of all, he is not without hope, for he will spend his life taking physic and frequenting the consulting-rooms of physicians, surgeons, and quacks—where he will argue "learnedly" about his symptoms until the unhappy physician wishes he had not been born. Nor are these patients suicidal—two points in which the condition differs from true melancholia, which is a state of hopelessness and a tendency to self-destruction.

[1] "Traité Clinique et Thérapeutique de l'Hystérie," par le Docteur Paul Briquet, p. 206. Paris, Baillière et Fils, 1859.

Diagnosis.—Hypochondriasis used to be regarded as the representative of *hysteria* in the male sex, but it is in reality a very different malady (table in § 523). In the former one does not meet with the "attacks," paralysis, and sensory alterations so frequent in hysteria. Hypochondriasis bears a closer relation to *neurasthenia.*

Causes.—Hypochondriasis is occasionally seen in the female, about the menopause, but the patients are mostly men of middle age. It is rarely seen before puberty, or, indeed, before thirty, and generally makes its first appearance between thirty and forty. There is often a neurotic family history, and often one of insanity. Dyspepsia, hepatic, or intestinal disorder is always present, and may be looked upon as its most frequent cause—a fact which is interesting when we remember the marked prostration and depression which attend gastric and abdominal disorders. Flatulence is a common symptom, and the stomach is often dilated. I am satisfied that many cases which were formerly, and are still, regarded as hypochondriasis are in reality intractable cases of neurasthenia, due to dilatation, kinking, stasis or sepsis of the cæcum or colon.

Treatment is neither easy nor satisfactory. The dyspepsia should be treated, and a draught of ammoniated tincture of valerian, or pil. asaf. co., or some other anti-spasmodic, taken occasionally for the flatulence. It gives relief, not only to the flatulence but to other symptoms. The bowels should be carefully regulated and otherwise treated. These means, with regular exercise, constant change, cheerful society, help to break through the vicious attitude of the mind ; but complete recovery is rare (see also Treatment of Neurasthenia, § 523).

§ **526. Alcoholism** or excessive indulgence in alcohol is met with clinically in three forms : (1) Acute alcoholism, (2) chronic alcoholism, a phase of which constitutes (3) delirium tremens.

Acute Alcoholism is due to an excessive quantity taken in a few hours. It gives rise to mental disturbance, muscular inco-ordination, and finally narcosis with a marked lowering of the body temperature, and a heavy alcoholic odour of the breath. The stupor of apoplexy, uræmia, and other causes of coma (§ 530), and the muttering delirium (§ 345) which occurs in pneumonia and other diseases, are apt to be mistaken for drunkenness—a serious error which is best avoided *by keeping a patient in the hospital under observation*, and suspending our judgment.

Chronic Alcoholism is due to the persistent imbibition of moderate doses of alcohol over a long period of time. The effects are worse when taken on an empty stomach or in the form of raw spirits. It acts as a tissue poison on the nervous, muscular (voluntary and involuntary), and epithelial elements, and hinders tissue oxidation, so leading to fatty degeneration.

The *Consequences and Symptoms* in the earlier phases resemble neurasthenia in many respects, with special toxic effects added later. The *neuro-muscular system* early shows signs in (i.) the unsteadiness and tremor of muscles, especially those of the hands, and in peripheral neuritis, of which alcohol is the most frequent cause. (ii.) The mental processes become slow, the memory, judgment, and will enfeebled, and later dementia or some other form of insanity ensues. Delirium tremens (p. 750) supervenes from time to time, and sometimes epileptiform convulsions. The *digestive system* is quite as commonly affected. (i.) Chronic gastric catarrh, attended by characteristic morning vomit-

ing, is always present in spirit drinkers, and gastric dilatation in beer drinkers. (ii.) Hepatic congestion occurs in all cases, and cirrhosis (with or without fatty degeneration) in a good number, but not in all. The *heart* dilates and undergoes fatty degeneration, and the vessels become thick and degenerated. The *kidneys* become congested, enlarged, and later cirrhotic. The *facies* of the chronic toper is characteristic—redness of the cheeks and nose, with œdema of the conjunctivæ.

Outbreaks of **delirium tremens** (*delirium e potu*) supervene from time to time in the course of chronic alcoholism; it never occurs as the result of one orgie in a usually temperate person. It is determined sometimes by an unusual drinking bout, sometimes by cutting off the habitual libations, sometimes as the result of an injury or the advent of pneumonia or some other inflammatory affection. Incoherent mutterings or ravings, characterised by hallucinations of vision (reptiles, rats, or monsters), accompanied by muscular tremor, intractable sleeplessness, and in bad cases two or three degrees of fever, are the leading features of the malady, which usually runs its course in two to five days.

The *Diagnosis* of chronic alcoholism is generally easy. The diagnosis of delirium tremens is referred to in § 345. Care should be taken not to overlook acute pneumonia, particularly of the apex. The *Prognosis* of delirium tremens for recovery is generally favourable if the temperature is not much elevated and the strength of the patient can be maintained.

The *Treatment* of acute alcoholism consists of the administration of an emetic, such as zinc sulphate, or apomorphine, $\frac{1}{6}$ grain, hypodermically. A chronic alcoholic habit is rarely abandoned after forty, and residence in a home is advisable in all confirmed cases, to enable the patient to regain his self-control. In other cases much may be done by careful domestic control and medical supervision. The gastric catarrh must be treated, and the uncomfortable "sinking" feelings may be much relieved by tincture of capsicum ℥v., sod. bic. gr. x., in an ounce of peppermint or chloroform water. In acute and chronic alcoholism one of the most distressing symptoms is insomnia, and it is difficult to treat. In the acute form opium is said to be contra-indicated, and it is doubtful if chloral or other hypnotics will shorten the attack. Digitalis in large doses may be tried, but it will be found that large doses of bromides, and in some cases opium, are generally successful. For dipsomaniacs (who have periodic outbreaks) and those who are born with an enfeebled nervous system, little can be done unless they will surrender the control of their lives to others. The great value of strychnine, atropin, and cinchona in the treatment of alcoholism and morphinism has no doubt been known to several observers for some years, and I cannot speak too highly of them as

regards my own experience. Some years ago a "gold cure" was "invented" and advertised,[1] but the active agents of this cure were undoubtedly the remedies above named, not the auric chloride. Strychnine and atropin sulphates (hypodermically), $\frac{1}{60}$ grain and $\frac{1}{120}$ grain respectively, may be given with cinchona bark (by the mouth) four times daily until the throat is dry and the pupils dilated.[2]

The treatment of *delirium tremens* calls in the first place for a reliable attendant (perhaps two or three), for the patient may be violent in his attempts to escape from his horrible visions, and artificial restraint may be necessary. To procure sleep and maintain the strength by nourishment are the main indications. Large doses of bromide, and chloral (if the pulse is not too weak) or hyoscine, $\frac{1}{100}$ grain, may be tried; but, as a rule, the malady runs its course unaffected by drugs. Graduated cold baths and cold packs are often very efficacious in cases with pyrexia (§ 392).

§ **527. Morphinism** (Synonym: Morphia Habit, Morphinomania) and other **drug habits.**—Hypodermically, **morphia** in small doses is a nerve stimulant as well as a hypnotic, and induces a feeling of contentment and well-being. But in the course of twenty-four hours reaction and craving for more occur, particularly when pain is present, and by degrees the dose has to be increased until in the course of a few months twenty to one hundred times the normal dose is necessary to produce a feeling of satisfaction, and can be easily tolerated. The only *signs* by which the *morphine habitués* can be detected are contracted pupils, pallor of the face, and the frequency with which they withdraw to satisfy their craving—a difference being observed in their depression before and their gaiety and brightness afterwards.

If such a patient is suddenly deprived of the drug, the following *symptoms* (which I have been accustomed to call "amorphinism") set in. The pulse, which was previously normal, becomes rapid and of low tension, and the patient prostrate, suffering agonies from the tingling in the limbs, sweatings, sneezing, lachrymation, diarrhœa, vomiting, uncontrollable restlessness, faintings, sinkings in the pit of the stomach, extreme wakefulness, and a host of horrible and indescribable somatic sensations resembling extreme neurasthenia.

Consequences of the morphia habit.—Enormous doses may be taken by gradual increase; one of the largest in my experience was 25 grains a day, reached after a habit of only two years. At first the patient is always gay, and has great capacity for mental and bodily endurance. But if the habit be continued, the character gradually becomes altered, and various moral obliquities become manifest. The patient alienates his friends by his tempers and unreliability; and, one by one, truth, reverence, virtue, and honesty disappear. If there be difficulty in procuring the drug, great craftiness is exhibited, and cases are known of women previously of the highest character selling their virtue and their husband's honour to

[1] The following is the analysis, supplied to me by Mr. Wootton, M.P.S.L., of Luton, Beds, of the "gold cure" in question:

The *injection*, four times daily. Strych. sulph. gr. ½; atropin sulph. gr. ¼; ac. boric. gr. xv.; aq. ℥iv.

The *mixture*, ℥i. every two hours when awake. Ammon. hydrochlor. gr. i.; aloin, gr. ii.; tr. cinch. co. ℥iii.; aq. dest. (better sp. chlor.) ℥i. Morph. used at outset of treatment also.

Red Mixture for alcoholics, ℥i. every two hours when awake. Sodio-auric chloride, gr. xii.; amm. chloride, gr. vi.; strych. nitrate, gr. i.; atropin, gr. ¼; ext. cinch. liq., ℥iii.; ext. cocæ liq., ℥i.; glycerin, ℥i.; aquæ, ℥i.

[2] Mr. S. B. Fenn, *Brit. Med. Journ.*, 1904, vol. i., p. 1008; and Dr. C. A. McBride, *Brit. Med. Journ.*, 1904, vol. i., p. 1006.

procure it. In course of time the mental powers gradually degenerate, and suicide is not infrequent in those who desire but are unable to rid themselves of the thraldom. The body also suffers, and the patients become pale, and generally emaciated. They get careless in the use of their syringe, multiple abscesses form and death may result from septicæmia. Fatty degeneration of the viscera ensues, especially of the heart, as in the case of a girl aged nineteen, which was reported in Paris some years ago. My own belief is that some of the cases of sudden death reported as due to overdose of morphia might be shown to be due to this condition.

Prognosis.—That the habit shortens life is certain, though it may go on for many years. The danger of sudden death, due to causes just explained, or from an overdose, is also considerable. The curability of a case of morphinism depends in my experience on three points: The age of the patient, the duration of the habit, and the curability of the painful affection for which it was first contracted. The actual quantity per diem which has been reached is of small account. A habit of 4 grains daily of ten years' duration was more difficult to cure than one of 22 grains of two years' duration. If carcinoma or some other cause of an incurable and recurrent pain be present, and especially if the patient is aged, the tendency to relapse is great, and it may be impossible to ease the pain in any other way.

Treatment.—(*a*) To break the habit the patient must place himself under the *absolute control* and guidance of a physician in whom he has confidence. Three methods are advocated—sudden cessation, gradual reduction, and gradual reduction combined with the substitution of solid opium or morphia by mouth or rectum. The first of these should never be employed unless time is an object, for the suffering is very great, and if the habit be of long duration, not without danger. In the third, which is advocated by Dr. Oscar Jennings, of Paris, a definite scheme should be written out by the physician, in which about double the quantity of opium, by mouth or rectum, is allowed to replace the gradual reduction in the morphia. Afterwards, when all hypodermics have ceased, the opium internally can be gradually reduced without much difficulty. The second method, gradual reduction, is the one which I have found most successful. There should be no difficulty in reducing, by ¼ grain a day, down to 2 grains, after which the reduction should continue in quantities less and less in a geometrical ratio by the addition to the stock-bottle of sterilised water daily. (*b*) Treatment of the symptoms of amorphinism. The prostration and somatic sensations may be partially relieved by moderate doses of alcohol, ammonia, and other stimulants, or by strychnine, 4 minims hypodermically, and atropin, as recommended in § 525. Digitalis and other drugs which aid the heart and contract the vessels are useful. For the vomiting and diarrhœa bismuth is best, but the diarrhœa should not be checked too much. Dionine in one case under my observation relieved the restlessness and craving. For the sleeplessness, chloral and other hypnotics and analgesics may be tried, but none are of great use. After recovery there are two tendencies: (1) towards relapse, and therefore the patient should be kept under observation; and (2) towards alcoholism, therefore great care is required in the administration of stimulants.

The **cocaine habit** leads to many of the troubles of the morphia habit, only there is a greater tendency to mental symptoms and mania. Morphia and cocaine is a frequent combination, and in such case the cocaine may, with comparative ease, be first withdrawn. Then the morphia reduction may be proceeded with as above described.

The **chloral habit** is not so common nowadays as the preceding. The consequences or symptoms consist of gastro-intestinal disturbance, lowered nutrition, pains, skin eruptions, depression, and irritability, palpitation, and cardiac weakness. Sudden death may occur from slight increase of the dose.

Sulphonal, phenacetin, antipyrin, and other tar products do not so readily engender a craving, but when habitually used the patient cannot do without them, and in course of time symptoms as in chloral hydrate arise.

§ **528. Collapse** (or **Shock**) is an acute condition of extreme bodily weakness, with exhaustion of the nervous system; an extreme state of prostration. An attempt is sometimes made to distinguish shock and collapse, but the two are clinically identical. The term "shock" is applied to that condition which follows any sudden mental or physical injury; the term "collapse" when it supervenes on some less sudden cause, such as cholera. The condition in either case is a serious one. The pathology is still obscure, but in effect there is paralysis, or more properly paresis, of all the muscular tissues of the body, voluntary and involuntary (muscles of the limbs, of respiration, of the heart and arteries). The *Symptoms* may be arranged under the following headings: (1) The skin is pale, cold, and clammy (especially of the extremities); the surface temperature is 2° F. or more under normal; the pupils are dilated, and react slowly to light. (2) The circulation and respiration are very feeble, the pulse being rapid and scarcely perceptible. (3) Loss of voluntary movement (sometimes restlessness and, in cases of profuse hæmorrhage, convulsions). The mind is apathetic, but the intellect is clear. The urine and other secretions are diminished or suppressed. The patient may die, or may pass into a reaction stage, with slight pyrexia.

Diagnosis.—In *coma* the mind is completely obscured, and the respiration laboured and stertorous. Except the functions of organic life, all is in abeyance. In *syncope* consciousness is generally lost, and the condition of prostration is more transient.

The *Causes* of collapse may be divided into those of sudden and those of gradual onset. When the condition is of sudden onset after injury or emotion, it is usually described as shock.

(*a*) Of *sudden* onset: (1) An overdose of chloroform or ether. (2) Surgical operations or severe injury. A vigorous man suffers more from operation than an old man or one who has been in bed for some time previously. Blows on the abdomen or extensive burns are always attended by more or less collapse. A relatively slight injury to a very sensitive part, such as the testicle, may produce collapse. (3) Severe and sudden emotion (terror or grief), or acute pain coming on suddenly, such as biliary or renal colic. (4) Poisoning by the narcotico-irritants (oxalic acid, carbolic acid, phosphorus, etc.) and by the asthenic poisons (hydrocyanic acid, aconite, digitalis, tobacco, veratria). A history in these cases may be absent. Ptomaine poisoning from tinned meats, etc., is attended by profuse diarrhœa. (5) Profuse hæmorrhage or diarrhœa, as in post-partum hæmorrhage and cholera. (6) Intestinal obstruction. (7) Perforation of some part of the alimentary canal, with extravasation of its contents into the peritoneum. (8) Rupture of an abdominal cyst or of an abdominal or thoracic organ. (9) Pulmonary or other embolism. (10) Heat exhaustion after exposure to a very hot sun.

(*b*) Of *gradual* onset : (1) Privation and exposure combined. (2) Profuse diarrhœa, such as usually terminates lardaceous disease. (3) Peritonitis and other abdominal inflammation. (4) The asthenic types of fever, such as may attend enteric and yellow fever. (5) At the termination of many diseases described in the chapter on Debility.

When a patient is found in a state of collapse or shock, the physician has to diagnose the *cause* of the condition. After applying restoratives he should inquire, first, whether there is a history of injury or emotional disturbance, hæmorrhage, etc. ; secondly, ask if the patient was in good health up to the time of onset of the condition of prostration, so as to exclude group *b ;* thirdly, inquire what food the patient has recently taken, and remember the possibility of poison ; fourthly, examine all the viscera, especially the heart and abdominal organs, beginning at the part which is or has been the seat of pain.

The immediate *Treatment* consists in applying warmth to the body by means of hot bottles and warm blankets. The head should be lowered, the feet raised, especially in cases following external hæmorrhage. Stimulants, alcohol or ammonia, may be given by the mouth if the patient can take them. Hypodermics of ether (℥ 20 to 60 every half-hour) or liq. strych. (℥ 10) or brandy should be given. Normal saline solution may be administered subcutaneously (§ 406) in cases where there has been profuse hæmorrhage or diarrhœa, and in many other cases of collapse. It may also be administered per rectum.

GROUP II. MENTAL SYMPTOMS.

Disorders of consciousness and of the mind form a very large group. There are not many serious disorders of the nervous system in which mental symptoms do not appear sooner or later ; and that complex organ, the mind, has also disorders peculiar to itself. These are generally omitted from text-books on medicine, but the scheme of this work would not be complete without a brief epitome of the more important.

It will be found convenient to deal with mental symptoms under four headings :

(α) Sudden interruptions of consciousness—*slight and transient*, such as petit mal ; or *severe and prolonged*, such as coma . . §§ 529 and 530
(β) Partial mental and "one faculty" defects, such as speech defects, or loss of memory . . . . . § 533
(γ) Acute perversions of the mind, such as delirium and mania . . § 536
(δ) Chronic perversions of the mind in adults, adolescents, and children, such as melancholia, dementia, and various special types § 537 *et seq.*

The patient complains of sudden transient **attacks of unconsciousness.** The case is probably one of Syncope or Epilepsy Minor.

§ **529. Sudden,** usually **brief and transient unconsciousness.**—The patient comes, perhaps, complaining of "attacks," "sensations," "faints," "dizziness in the head," or "interruptions of thought."

You have only the patient's account to guide you, and it may be a little difficult to diagnose the condition, but it is probably SYNCOPE, EPILEPSY MINOR, ARTERIAL (or senile) VERTIGO, or SOME CASES OF AURAL VERTIGO.

It is necessary, first, to ascertain definitely whether the patient was really unconscious : Did he know who was beside him, or hear when they spoke to him, and did he fall down ? Secondly, the age and sex of the patient—epilepsy appears for the first time between ten and thirty ; hysterical faints are almost always confined to young females. Thirdly, the history of previous attacks.

I. **Syncope** is a loss of consciousness more or less complete, generally of short duration, due to cardio-vascular failure of functional or organic origin. It does not generally last longer than a few minutes, though the duration and intensity vary considerably. Before the attack the patient is pale, sometimes a useful warning. The process of going off is accompanied by a disagreeable " swimming in the head," which has some resemblance to vertigo, or by an indescribable sinking feeling in the region of the stomach. The process of recovery is more gradual than petit mal. The diagnosis of cardiac syncope from nervous faints (of vaso-motor origin), and these from petit mal, and their causes and treatment, have been given in § 24, p. 39. Many considerations seem to point to the probability that the essential cause of hysterical faints in an instability of the abdominal sympathetic.[1] We know that the blood pressure in the cerebral arteries is largely, if not entirely, regulated by the amount of blood in the splanchnic area, being high when the latter is relatively empty of blood, and low when it is relatively full.

II. **Epilepsy Minor** (Synonyms : Petit Mal, Epileptic Vertigo).—There are two varieties of idiopathic epilepsy, E. major (§ 598), consisting of unconsciousness with convulsions, and E. minor (petit mal), unconsciousness without convulsions. They sometimes alternate in the same individual. There are many degrees, shading from one to the other, and few dispute the identity of the two maladies. Only one circumstance can be seriously urged in this direction—namely, that amyl nitrite will often stop an attack of major, but increases the severity of an attack of minor, epilepsy. Epilepsy minor may be defined as a momentary or brief loss of consciousness, preceded—in about half the cases—by an aura or warning, unattended by convulsions, and often without falling, not followed by the stage of stupor, and the whole lasting rarely more than half a minute to a minute. In the attacks now under consideration, it may be that the patient only pauses in a conversation, or there is only a vacant look, a fixity of gaze, dilated pupils, or momentary pallor of the face, which none but a close observer would notice. More usually, however, the patient—who generally

[1] " Clinical Lecture on Hysterical Attacks," the *Lancet*, July 20, 1901 ; and " Lectures on Hysteria," Glaisher and Co., London, 1909.

refers to these attacks as "sensations"—feels giddy and loses his equilibrium for a few moments. The chief point of difference between nervous faints (p. 39) and minor epilepsy is that in the former there may be no definite loss of consciousness, although the patient falls, whereas in the latter a definite brief loss of consciousness is the invariable characteristic, and the patient may not necessarily fall down. The absence of any apparent rotation of objects helps us to distinguish epilepsy minor from vertigo; and its appearance always for the first time in the earlier decades of life distinguishes it from "senile syncope." Some say that petit mal is more likely to result in insanity than grand mal. The *Treatment* is on the same lines as that of epilepsy major (§ 598).

Masked Epilepsy is a still more incomplete form of epileptic attack, in which the patient, though unconscious, continues to perform automatically various acts during the seizure. Thus, a shoemaker under my care would continue his sewing; and Trousseau mentions a skilled violinist who continued to play with precision. He also mentions a judge who used to leave the bench during an attack of masked epilepsy, micturate in a corner of his robing-room, and return to the Bench again without a return of consciousness. The criminal records show that homicide may be performed during these attacks.

III. **Senile Syncope; Senile or Arterial Vertigo; Apoplectiform Attacks.**—Under these terms may be included various attacks attended by unconsciousness, varying in degree from a transient interruption of thought to a severe loss of consciousness. Those who have much to do with old people are aware that brief losses of consciousness are extremely frequent among them. Sometimes it amounts to nothing more than a momentary confusion of thought. They were variously described by my old patients in the Paddington Workhouse as "dizziness" or "giddy faints." They often give rise to a momentary reeling if they happened to be walking at the time. But sometimes it is a definite unconsciousness, lasting one or two minutes, or longer, in which they fall unless they lean against or catch hold of something. Every gradation is met with between these attacks and a severe syncopal or apoplectic seizure. These attacks are generally associated with arterial hypermyotrophy[1] or some form of arterial degeneration disease, especially when this is combined with cardiac failure. In a few instances of the severer form of attack there were definite evidences after death of minute softenings or hæmorrhages of various dates, sometimes in considerable numbers, with which the attacks were undoubtedly connected.

IV. **Aural Vertigo** (§§ 511 and 618).—Patients often speak of attacks of aural vertigo as "faints," though they are not usually attended by unconsciousness. It is not, however, sufficiently recognised that severe attacks of aural vertigo may be attended by complete loss of consciousness of some duration, and that such attacks may be connected with middle-ear catarrh as well as lesions of the inner ear. The attacks in Ménière's disease are often so attended.

One of the most severe cases of paroxysmal vertigo with unconsciousness, due to middle-ear catarrh, which I have met with occurred in G. C. B., æt. thirty-eight, a cattle dealer, who consulted me in February, 1896, for severe attacks of giddiness, which he had had for two and a half years. The giddiness would come on suddenly, without warning, "like a windmill in the head," and "things went all round"; sometimes he became unconscious and fell down, unless near something to catch hold of; sometimes they were followed by nausea or vomiting. He had lately had two or three a week, and since August, 1894, he had had to give up work. In one very bad attack he became quite unconscious for some time, and *fell out of his cart.* Six months before the vertigo had come on—January, 1893—

[1] Trans. Path. Soc. Lond., 1904.

his hearing had gradually become defective, and ever since then he had had a sensation as of a "kettle singing in his head." On examination, I found his hearing to be about half normal, but the perosseous hearing was good. All his symptoms were attributable to middle ear catarrh. After six months' pharyngeal and aural treatment under Dr. Bolton Tomson (of Luton, Beds), his hearing was quite restored, and he was able to leave off the bromides without a return of any of his troublesome and dangerous attacks.

§ 529*a*. V. **Ménière's Disease** (or Labyrinthine Vertigo) is a disease supervening suddenly, with an apoplectiform attack, with unconsciousness, followed by recurrent attacks of paroxysmal vertigo, associated with nerve deafness due to hæmorrhage into the vestibule or semi-circular canals. The term is sometimes applied to any form of vertigo associated with deafness, but this does not tally with Ménière's original description,[1] and the name is better exclusively reserved for vertiginous attacks attended by loss of consciousness, and associated with deafness.

There are four classical symptoms: (i.) The vertigo is always in paroxysms in which, after the first attack, consciousness is retained, though the patient may fall, owing to the disturbance of equilibrium. Irritation of the left semi-circular canals usually produces a sense of rotation from left to right; destruction the reverse. (ii.) The attacks are commonly attended by nausea, or even vomiting. (iii.) There is deafness in the great majority of cases, on one or both sides, the patient being unable to hear a tuning-fork placed on the teeth or the head (perosseous—*i.e.*, nerve deafness); and (iv.) tinnitus, or buzzing in the ears. In characteristic cases of Ménière's disease these four symptoms supervene suddenly with an apoplectiform attack (accompanied by transient loss of consciousness), which has been shown to be due to hæmorrhage into the labyrinth. (i.) and (ii.) afterwards become paroxysmal, (iii.) and (iv.) more or less permanent. Knapp[2] has observed that the hearing is impaired at first only for the higher and lower octaves; and Charcot states that some patients have the vertigo and buzzing only while the deafness is partial, disappearing when this becomes total. This is probably diagnostic of labyrinthine as distinguished from central vertigo.

In regard to *Treatment*, the disease is undoubtedly very intractable, if not incurable, and our efforts should be directed mainly to the circulatory system, so as to regulate it and avoid a repetition of hæmorrhage. The symptoms may be relieved by bromides and other sedatives. Charcot recommends 5 grains of quinine thrice daily, and he mentions one case which was cured by this means. Trinitrin often relieves the symptoms for a time.

The patient is attacked with **complete unconsciousness,** *suddenly supervening and more or less prolonged.* The case is one of apoplectic or other form of COMA.

§ **530. Coma** is a condition of loss of consciousness coming on more or less suddenly, in which, in its complete form, all signs of vitality, excepting those of organic life, are suppressed. The patient is deprived of all power of movement and sensation. He is neither able to hear nor to understand an order to put out his tongue shouted into his ear. The limbs fall helplessly into any position. The eyelids are closed, and the conjunctival reflexes absent. The respiration is slow and stertorous, owing to the flapping of the palate and the falling back of the tongue on to the posterior wall of the pharynx. The pulse and the respiration are the only signs of life. The temperature is—at any rate, at first—

[1] It seems that all Ménière's original cases came on in an apoplectiform manner. *Gazette Médicale*, 1861.

[2] "Archives of Ophthalmology and Otology," 1871, Knapp and Brunner

normal or sub-normal. The *typhoid state* (§ 346) is a term applied to the comatose condition which supervenes in certain fevers, and may be recognised by the presence of pyrexia, the history, and the muttering delirium. *Syncope* is readily distinguished from coma by the unconsciousness being much less in degree and much more transient. The following is a list of the possible causes of coma :

I. Head injury.
II. Apoplexy and other vascular lesions.
III. Other gross and functional cerebral lesions.
IV. Alcohol.
V. Opium.
VI. Uræmia.
VII. Diabetes.
VIII. Cholæmia.
IX. Heatstroke.
X. Addison's disease, Raynaud's disease, and other rare conditions.

Coma in children arises under somewhat different conditions (§ 532).

The CLINICAL INVESTIGATION of coma is of the highest importance, as it is an emergency of the gravest significance. When called to such a patient, whom one sees, perhaps, for the first time, the question as to the cause is one of the most difficult that we have to solve. The commonest causes of such a condition are drink, cerebral apoplexy, head injury, opium-poisoning, and uræmia, and the prognosis and treatment differ in these several conditions. Your mode of procedure should be as follows : (1) *Examine the head* (and other parts) carefully, to see if there be any signs of injury, and if the case be a medico-legal one, make a precise written note on this point. If there be evidence of an injury, the question of whether the injury has caused the coma or whether it occurred after the patient was seized, should be borne in mind. (2) Note the *odour of the breath.* If this be alcoholic, it does not follow that the condition is due to drink, for stimulants are frequently given by the friends to restore the patient during such an attack. Note also whether the breath has the sweet odour of diabetes. (3) Observe most particularly the state of each *pupil* and the conjunctival reflex. Both pupils are much contracted in opium-poisoning and hæmorrhage into the pons. Hæmorrhage into other and commoner situations within the cranium usually causes inequality or dilatation of both pupils. In the other causes of coma they are usually both dilated. (4) Ascertain whether any *paralysis* of the limbs is present, or whether the face is drawn to one side. This may be a little difficult, but usually in apoplexy the paralysed leg and arm of one side of the body, opposite to that of the lesion, are very rigid or very flaccid as compared with the other side, and by raising the limbs and allowing them to fall it will easily be found whether one side is more rigid or more flaccid than the other. In nearly all cases due to gross cerebral lesion (tumour, abscess, meningitis, etc.), one side is weaker than the other. (5) Count the *pulse*, examine the *arteries*, and auscultate the *heart.* High arterial tension suggests uræmia or apoplexy ; great slowness suggests opium-poisoning ; a presystolic murmur suggests cerebral embolism. (6) Count and observe the *respirations.* A stertorous or snoring quality is simply an indication of the profundity of the coma, being due to paralysis of the tongue and palate. The respirations are very slow in opium-poisoning. In grave cases of apoplexy and uræmia the respiration assumes a Cheyne-Stokes character. (7) Take the *temperature.* It is often very low in uræmia and in opium-poisoning, sometimes a little lowered in apoplexy and in drink. There is sometimes a tendency for it to run up after an attack of apoplexy, and this is of very serious import. (8) Procure (by catheter if necessary) and examine some of the *urine.* The absence of albumen is against renal disease ; a small amount of albumen does not help one much to distinguish between apoplexy and uræmia. In diabetic urine we find sugar ; in opium-poisoning morphia. Atropine and other vegetable alkaloids are also excreted by the urine. (9) Observe

the *age*, and inquire into the *history*—whether the attack came on suddenly in apparent health or after some previous indisposition. As regards the age, coma in childhood is almost confined to post-epileptic coma, meningitis, cerebral tumour, or sinus thrombosis ; about middle age cerebral hæmorrhage is to be suspected. Coma sometimes arises in the course of a disease, the history of which is readily revealed by inquiry, such, for instance, as epilepsy. But, on the other hand, the patient's friends may have been quite unaware of the existence of any disease, such, for instance, as diabetes, contracted granular kidney, or cerebral tumour in the frontal region.

The commonest causes of coma in every-day practice are drink, intracranial hæmorrhage (with or without injury), opium poisoning, and uræmia (see table below).

TABLE OF DIAGNOSIS OF COMA DUE TO DRINK, APOPLEXY, OPIUM POISONING, AND URÆMIA.

	Pupils (P.) and Conjunctival Reflex (C. R.).	*Pulse and Respiration (R.).*	*Paralysis.*	*Depth of Coma.*	*Course.*
Drink.	P. equal, normal, or dilated. C. R. present.	*Pulse rapid and strong, then weak.* R. normal or snoring.	None, but inco-ordination if able to walk or move.	*Can be roused.*	Progressive recovery in twelve hours.
Apoplexy or Fracture with Compression.	P. usually dilated and unequal. *C. R. lost.*	Pulse generally full and bounding. R. stertorous ; may be Cheyne-Stokes.	*Hemiplegic* rigidity or flaccidity and often *facial paralysis.*	Cannot be roused.	Stationary.
Opium Poisoning.	*P. very contracted and equal.* C. R. usually present.	Pulse and R. both very slow. R. stertorous.	General weakness.	Can sometimes be roused.	Progressive towards death or recovery in ten to twelve hours.
Uræmia.	P. normal or dilated. C. R. usually present.	*Pulse slow and arterial tension high.* R. sighing, and may be Cheyne-Stokes.	None.	Cannot be roused.	*Coma alternates with convulsions.*

I. **Head Injuries** may produce either *concussion* (bruising) or *compression* of the brain, the symptoms of which differ more in degree than in kind (table, p. 760). Injury may also be accompanied by conjunctival hæmorrhage, paralysis of the cranial nerves, and inequality of the pupils. Bleeding from the ear and sub-conjunctival ecchymosis do not necessarily (though usually) indicate fracture of the base. Injury to the head may produce compression of the brain, and therefore coma, in four ways : (1) Fracture of the skull, with depression ; (2) hæmorrhage into or upon the brain—in both of which the coma comes on immediately after the injury ; (3) the effusion of inflammatory products, when the coma comes on after an interval of a few days ; and (4) abscess, when coma comes on after a week or two at least.

TABLE OF SYMPTOMS OF CONCUSSION AND COMPRESSION.

Concussion of the Brain.	*Compression of the Brain.*
Symptoms.—As in shock (*q.v.*), *plus* sudden unconsciousness, but patient can be roused in most cases.	Completely unconscious; cannot be roused.
Pupils.—Equal, dilated, sluggish reaction to light.	Immobile, often unequal, at first contracted, later on dilated.
Respiration.—Shallow, slow, sometimes sighing.	Slow, stertorous, sometimes irregular.
Motor System.—Muscles relaxed, but no absolute paralysis.	Paralysis; cheeks blown out with expiration; often rigidity on one side of the body.
Bladder.—Frequent micturition.	Retention till overflow with "false" incontinence.

NOTE.—There is considerable difficulty of diagnosing whether a case has *slight compression* or a *very grave degree of concussion.*

§ **531.** II. **Apoplexy** (a "stroke") is a term which may be conveniently retained to indicate a sudden unconsciousness due to a vascular lesion within the skull. There are three kinds of such vascular lesions—hæmorrhage, embolism, and thrombosis. The older authors used to state that *hæmorrhage* from the rupture of a cerebral artery could be distinguished from simple embolism or thrombosis by the occurrence of *loss of consciousness* (sometimes accompanied by convulsions) in the former, and not in the latter. But further experience has shown that this distinction is only a matter of degree; profound coma may sometimes arise from the embolic blocking of a moderately large artery, or from thrombosis; while, on the other hand, slight hæmorrhage supervening gradually may be unattended by loss of consciousness. The *extent* and the *suddenness* of the vascular lesion, rather than its nature, determine the presence of coma.

Symptoms of Cerebral Hæmorrhage.—A prodromal stage or warning in the form of headache or vertigo for some days before may be complained of, connected undoubtedly with the high arterial tension which is its leading etiological factor; or the coma may come on very suddenly without warning. It may be followed or not by convulsions. Sometimes the paralysis comes on with faintness and vertigo only; or the paralysis may come on more gradually, followed later by unconsciousness (ingravescent apoplexy). Sometimes it comes on during sleep. The lesion causes paralysis on one side (usually) of the body, which is indicated at first either by a greater rigidity or a greater flaccidity, or by the absence of spontaneous movements on that side. The pupils are unequal, the conjunctival reflex lost. The temperature, particularly in large hæmorrhages, is usually at first one or two degrees below normal. In the course of twenty-four to forty-eight hours the thermometer usually shows a rise of one or two degrees, at which point

it remains for several days. A rapid elevation of temperature within a few hours of the seizure indicates hæmorrhage at the base, and therefore a speedily fatal termination.

Diagnosis.—In view of the importance of differentiating this condition in an emergency, a table is given above (p. 759). It should be remembered that apoplexy frequently supervenes in the course of chronic Bright's disease, and therefore uræmia and apoplexy may be concurrent. The diagnostic features of the greatest value in apoplexy are the state of the pupils, particularly their inequality, the loss of the conjunctival reflex, and the presence of hemiplegia. The diagnosis of the various *forms of vascular lesion* is given in a table below.

TABLE OF THE DIAGNOSIS OF CEREBRAL HÆMORRHAGE, EMBOLISM, AND THROMBOSIS.

	Cerebral Hæmorrhage.	*Embolism.*	*Thrombosis.*
Age.	Middle and advanced age.	Any age, but frequently young.	Any age.
Causes.	1. Arterial degeneration. 2. Vascular strain. 3. Excitement.	1. Cardio-valvular lesions, especially mitral stenosis. 2. Thrombus in the peripheral vessels.	1. Syphilitic endarteritis. 2. Cerebral atheroma. 3. Exhausting disease; phthisis; anæmia. 4. Slowing of blood—*e.g.*, cardiac enfeeblement.
Onset.	Coma usually sudden, sometimes with convulsions.	Sudden onset of paralysis, but usually no loss of consciousness or convulsions.	Paralysis may be sudden, or after premonitory symptoms, vertigo, convulsions. Coma unusual.

As regards the *locality* of the hæmorrhage, the usual position (about 76 per cent.) is the *internal capsule*, from the lenticulo-striate artery (Fig. 156) giving rise to hemiplegia of face and body on the side opposite to the lesion. In about three-fourths of the cases of hæmorrhage into the *ventricles* there is paralysis or rigidity of all four limbs, and the condition is uniformly fatal. Marked contraction of both pupils, or crossed hemiplegia, suggests hæmorrhage into the *pons*. Hurried or Cheyne-Stokes respiration is more common with hæmorrhage in this position, and the prognosis is grave. *Meningeal hæmorrhage* is suggested by the absence of definite paralysis and the presence of initial and recurring convulsions. *Pachymeningitis hæmorrhagica* (hæmorrhage into the thickened meninges) is a condition giving rise to attacks of coma which differ (in most of the cases I have seen) from the other causes now under consideration in their slow advent. It far more often occurs in lunatics and in the dementia of old age. *Conjugate deviation of the head and eyes* towards the paralysed side is frequent when the hæmorrhage involves the motor tract.

Prognosis.—About half the cases of apoplexy, taking all cases together, recover from the attack, but with remaining paralysis. The depth and duration of the coma are fair measures of the extent of the mischief, and therefore of the prognosis. The signs indicating deep coma, which are therefore of unfavourable import, are loud stertor, completely insensitive conjunctivæ, flapping cheeks, and increasing cyanosis. Convulsions, or the early appearance of rigidity, or a sudden rise of the temperature, are unfavourable. Coma coming on slowly and progressively increasing (that is, ingravescent apoplexy) is more unfavourable than that which comes on more suddenly, with less complete coma. A more unfavourable form still is when, shortly after the first attack of apoplexy, a second supervenes ; from this the patient rarely recovers. As regards *locality*, the worst positions are intraventricular hæmorrhage, basal hæmorrhage, and hæmorrhage into the pons.

Etiology.—Cerebral hæmorrhage is more frequent in the male sex, and in those over forty or fifty. The rarer cases of "apoplectic seizure" in a person under forty are almost invariably due to embolism or thrombosis. Heredity plays an important part by reason of the tendency to vascular disease which runs in families. There is a marked predisposition in plethoric persons to suffer from cerebral hæmorrhage —the stout, thick-necked build, with ruddy cheeks. Disease of the vessels is an almost necessary precursor to their rupture. High arterial tension is a most important factor in the causation of apoplexy ; it predisposes to arterial disease, and may also determine the hæmorrhage. The causes of high arterial tension are given in § 61 ; but the commonest cause of high tension is chronic Bright's disease, and that is why this morbid condition is so frequently associated with cerebral hæmorrhage. Leukæmia, purpura, and other blood diseases may occasionally cause cerebral or meningeal hæmorrhage.

Cerebral Embolism.—The preceding remarks have reference to cerebral hæmorrhage, but cerebral embolism involving a fairly large artery may give rise to all the symptoms of apoplexy. An "apoplectic stroke" in persons under forty is almost invariably due to embolism or thrombosis, though hæmorrhage may occur in children. The age of the patient, the presence of cardiac, especially mitral, disease, or some other condition giving rise to embolism, aids us in diagnosis (see table, p. 761).

Thrombosis of the Cerebral Arteries is also a cause of apoplectic seizure. In most cases thrombosis arises from a *gradual* occlusion of the lumen of a vessel by chronic arterial disease in the aged, or by syphilitic endarteritis in the young. The supervention of symptoms, however, is usually sudden, and in only a proportion of cases attended by coma (see table above).

Thrombosis of the Cerebral Sinuses is not common, but may give rise to coma and all the symptoms of apoplexy. It may arise from caries of the skull of syphilitic or tuberculous origin, extension from a cerebral abscess, and occasionally

from the pressure of an aneurysm, gumma, or other tumour ; or in association with meningitis. *Non-pyogenic thrombosis* (especially of the superior longitudinal sinus) also occurs with cachectic conditions, chronic diarrhœa, enteric fever, and marasmus in children. *Septic thrombosis* and the differential signs of thrombosis of the lateral, cavernous and longitudinal sinuses are described under Intracranial Inflammation (§ 551).

The *Prognosis* of cerebral embolism as regards life is usually good, though the paralysis remains, and if the causal condition remains, it is apt to recur. In thrombosis the prognosis is good when due to syphilitic endarteritis, less favourable when occurring in the aged, and extremely grave when associated with exhausting disease and anæmia.

The *Treatment of an apoplectic seizure* is not very hopeful. Perfect rest and quiet are very important. The patient should, as a rule, be left in the room where the seizure occurred—a mattress being placed on the floor, if necessary, rather than incurring the movement necessary to raise him on to a bed. The head and shoulders should be raised, and the patient turned gently over to one side to prevent the tongue falling back into the pharynx. The administration of food is, as a rule, undesirable, at least by the mouth, for fear of it passing into the air passages, and alcohol must be absolutely forbidden. The bladder should be watched, and the catheter carefully passed if necessary. The patient will do no harm for a day or two without nourishment by mouth, and the lips may be moistened by a feather dipped in water. In cases due to *embolism* nothing further can be done excepting to prevent a recurrence ; in *thrombosis* (other than syphilitic) stimulants are indicated. In *hæmorrhage* a brisk purge should be given ; two drops of croton oil or 4 to 8 grains of calomel on the tongue is a good method, followed, if necessary, by an enema of castor oil or turpentine. The chief indication is to prevent any extension of the hæmorrhage. If the pulse is full and bounding, it is a good practice to bleed to the extent of 3, 5, or even 10 ounces ; and these patients nearly always do well. The administration of aconite or veratrium viride has been suggested to reduce the blood-pressure in lieu of venesection. An ice-bag or a cooling lotion to the head is recommended by some, and may be of some benefit if care be taken that after it is once started there is no intermission, for the reaction in such an interval may increase the mischief by determining blood to the head. Blisters to the back of the neck, and mustard plasters to the calves or soles of the feet " to rouse the patient," are in my belief worse than useless. Hæmostatics, ergot, gallic acid, acetate of lead have been recommended, and belladonna may be of use.

III. **Other Gross and functional Cerebral Lesions.**—Generalised convulsions are generally attended by some disturbance of consciousness, and sometimes by coma, during and sometimes following the attack. But in such circumstances the convulsions constitute the major feature,

and their causes are therefore given under that symptom (§ 597). It will there be seen that the list of these causes corresponds very closely with the causes of coma, and this is what one would expect; for in general terms cortical *compression* is manifested by coma, cortical *irritation* by convulsions, and toxæmia may be evidenced by either. If the history is wanting, difficulty in diagnosis may be experienced. Among the chief **causes of both convulsions** and **coma** are the following:

1. In Post-Epileptic Stupor the unconsciousness is not so complete as in apoplexy; there is no hemiplegia, and within a few hours the patient wakes. There is a history of previous attacks if the patient is an adult.

2. Cerebral Tumour and Abscess may give rise to attacks of coma, which, in the absence of a history of previous ill-health, are difficult to distinguish from apoplexy. In such gross lesions of the brain there is (1) optic neuritis; (2) paralysis of the cranial nerves, and perhaps hemiplegia. Tumour of the frontal lobe, however, may cause no paralyses. (3) The coma not infrequently alternates with or is attended by convulsions. (4) A history of headache, giddiness, and vomiting may be present.

3. In one-fifth of the cases of Disseminated Sclerosis attacks of coma occur (Charcot), lasting a day or two, and then passing into a state of stupor. In these the face is flushed, pulse rapid, and temperature elevated to 104° or 105° F. The tremor and other symptoms are always worse as the patient emerges from these attacks.

4. General Paralysis of the Insane at some stage is almost invariably accompanied by fits of various kinds; sometimes they are comatose, sometimes epileptiform, with partial or complete loss of consciousness, and sometimes without unconsciousness. Unconsciousness is a very bad sign, and the patient frequently dies in such attacks.

5. Certain Acute and Subacute Cerebral Lesions, such as tuberculous and simple meningitis, cerebro-spinal meningitis, and septic sinus thrombosis, may cause coma. Coma in such conditions is usually of late onset, due to compression of the brain, and a history and other signs are obtainable (see Intracranial Inflammation, § 547). However, in tuberculous meningitis coma may be of sudden onset, especially in children.

IV. **Intoxication,** or Acute Alcoholic Poisoning.—The coma which supervenes after heavy drinking, or a single large dose of alcohol, may very closely resemble apoplexy and cerebral compression from head injury. In any case of doubt it is wise to admit the patient to the hospital, or to treat him on the supposition of the more serious condition. If house surgeons would bear this in mind, we should see less of those paragraphs in the newspapers headed "drunk or dying." The smell of alcohol in the breath is fallacious, as friends may have given alcohol to restore the patient. The chief differential features of the coma of intoxication are: (1) The coma is rarely so profound as in apoplexy, and the patient can generally be roused. (2) The absence of inequality of the pupils, hemiplegia, or convulsions (see table, § 530). (3) Procure some of the urine, and add one or two drops of the urine to fifteen drops of a chromic acid solution, made by adding one part by weight of potassium bichromate to 300 parts by weight of strong sulphuric acid. The solution turns a bright emerald green if alcohol be present in quantity (Anstie).

V. In **opium poisoning** the patient becomes progressively drowsy, and (1) coma succeeds gradually. (2) The pupils are equal and extremely contracted; (3) the pulse and respirations are slow; (4) there is no hemiplegic rigidity or flaccidity of the limbs; (5) the coma gradually deepens, the face becomes cyanotic, and the pulse and respiration gradually cease together.

For the symptoms of poisoning by other narcotic drugs the reader is referred to manuals on toxicology, but it is well to remember that chloral and chloroform, coal-gas, belladonna, the bromides, cannabis indica, and occasionally hydrocyanic acid, may all act as narcotic poisons.

VI. **Uræmia** ("serous apoplexy" of older authors[1]) may lead to coma in the advanced stages of kidney disease. (1) The coma is rarely quite so profound as in apoplexy; it is more of a stupor or drowsiness, which gradually deepens. (2) In the great majority of cases, stupor alternates with convulsions, and in many cases it alternates with muttering delirium. (3) There is an absence of hemiplegic rigidity or paralysis. (4) There is albuminuria. (5) If a history is obtainable, the earlier symptoms of uræmia will be revealed (§ 270).

VII. **Diabetic coma** supervenes very suddenly, often in apparent health, and is very profound. Its two characteristic features are (1) the sweet odour of the breath, somewhat resembling that of chloroform, and (2) the presence of sugar in the urine. It almost invariably results in death, being one of the most frequent modes of termination in that disease.

VIII. **Cholæmia.**—Hepatic diseases which result in destruction of the secreting tissue of the liver—notably the later stages of cirrhosis and acute yellow atrophy—give rise to a condition clinically resembling uræmia. Clinically this condition is in most cases differentiated by the jaundice, slight in the concluding stage of cirrhosis, very marked in acute yellow atrophy. The coma under these circumstances has been called cholæmia, under the impression that it is due to the presence of bile in the blood; but bile freed from mucus injected into the blood does not give rise to symptoms.

IX. **Heat-stroke** (sun-stroke) is classified into (1) a "cardiac" variety, in which the patient suddenly goes off into a dead faint, with symptoms pointing to failure of respiration and circulation; and (2) a "cerebro-spinal" variety in which coma gradually supervenes. The circumstances under which it occurs are the only means of its differentiation. The comatose form is described in § 375.

X. **Certain rare diseases**—*e.g.*, **Addison's disease** and **Raynaud's disease**—are occasionally attended by sudden coma, perhaps with vomiting and hemiplegia. A malignant form of **malaria** (§ 378) is attended with coma which, coming on suddenly, may lack a history; and English trained medical men newly arrived in India may mistake these cases for apoplexy. *Excessive muscular exertion* has caused coma, probably due to accumulation of toxic products (Dr. V. Poore, the *Lancet*, 1894, vol. i., p. 1066).

The *Prognosis* of coma is always grave, and the gravity increases with the depth and the duration of the coma. The coma after head injury usually comes under the care of the surgeon. The coma of apoplexy and other vascular lesions has been already dealt with. In

[1] The older authors recognised the clinical resemblance of uræmic coma to the coma of cerebral apoplexy, but they found no intracranial hæmorrhage. In cases of chronic Bright's disease the cerebral convolutions waste, and the space is occupied by the effusion of serum on the surface of the brain, which was therefore regarded by them as the cause of coma; hence the term "serous apoplexy."

post-epileptic coma, if the patient does not recover within a few hours, the status epilepticus is present. Such a condition being very rare, the diagnosis should be carefully reconsidered. Coma due to degenerative conditions of the nervous system is usually recovered from, leaving behind perhaps a temporary paralysis. Coma occurring with tumour of the brain or acute lesions is usually fatal. The prognosis of opium poisoning depends upon the time which elapsed before the patient was seen, the treatment adopted, and the vigour with which it was carried out. Uræmic coma is not so unfavourable as might be thought ; cases recover with proper treatment, but sooner or later the condition recurs. In diabetic coma the patient rarely rallies.

Treatment.—Apoplexy has been already dealt with, and the treatment of head injury is carried out on similar lines, bearing in mind that surgical aid is necessary in many cases. Many cases of coma (other than apoplexy) may be relieved, temporarily at any rate, by lumbar puncture (pp. 921 and 930)—fracture of the skull, cerebral tumour, intracranial inflammations, and *even* uræmia—and cures have been reported. It deserves extensive trial. For alcoholism and poisoning a prompt emetic should be given ; a hypodermic injection of apomorphine is one of the best remedies. Opium poisoning is treated by frequent washing with the stomach-pump. Coffee, atropine, or strychnine are given if the heart or respiration be failing. The patient must be kept awake by walking him about, applying electricity to the limbs, ammonia to the nostrils, and artificial respiration. For uræmia eliminate the poison in the blood by brisk purgatives, hot packs, venesection, and saline injections.

§ **532. Coma in Children,** apart from injury, may be due, in order of frequency, to post-epileptic stupor, tuberculous meningitis, post-basal meningitis, suppurative meningitis, cerebral tumour, syphilitic pachymeningitis, sinus thrombosis, and hæmorrhage ; diabetes, abscess, and cysts are rare causes. The history, mode of onset, and associated symptoms aid the diagnosis. Tuberculous meningitis is by far the most frequent cause. Out of 86 cases of coma in childhood, Dr. F. E. Batten found tuberculous meningitis in 50 cases, non-tuberculous meningitis in 17, and cerebral tumour in 16 cases. Cerebral hæmorrhage occurs chiefly in association with the specific fevers, such as small-pox and whooping-cough, also with rickets and scurvy. In marasmic conditions thrombosis of the longitudinal sinus (§ 551) may ensue, together with meningeal hæmorrhage, giving rise to convulsions followed by coma. Thrombosis of the veins of Galen (§ 625), and lateral sinus thrombosis, which occurs with ear disease, may cause coma.

β. Partial Mental and "One Faculty" Defects.

One faculty of the mind, such as the memory or attention, may be affected, and there may also be various partial mental defects which

do not amount to insanity, though they may constitute its earlier phases. These will now be considered—

Defects of speech (motor and mental).
Defects of memory (partial and entire).
Defects of attention (deficiency and excess).
Defects of other mental faculties.
Hypnotism.
Catalepsy and trance.

§ 533. Defects of Speech and other Signs of Thought may be purely motor, or they may be of central or mental origin (ideo-motor or ideo-sensory), but it will be convenient to consider them both here, as they are so frequently associated. All of these defects may be simply and graphically represented in the following scheme :

Defective Communication with others.

Defects in the **outgoing** processes . { motor defects (paralysis). ; ideo-motor . . . { speech. ; writing.

Defects in the **incoming** processes . { ideo-sensory . . . { visual. ; auditory. ; blindness or deafness.

Motor defects of speech are recognised by an **error in articulation.** The patient says the words, but pronounces them badly, just as in cases of motor defect of writing the patient can sometimes write, but writes badly. The muscles of speech are those of the lips, tongue, palate, larynx, and respiration, and these, like other muscles, may be the seat of paralysis, tremor, spasm, or inco-ordination.

Clinical Investigation.—In order to investigate a case of motor defect of speech, we must (1) pay attention to each of the different muscles just named. In the course of conversation with the patient we can generally detect any of the motor defects which are mentioned below. If not, we may ask the patient to repeat the alphabet through, and we shall readily detect any paresis or other defect of the muscles. (2) Notice whether there be any tremor of the lips or of the tongue when protruded ; any separation of syllables ; syllabic utterance ; or, on the other hand, any slurring or running together of the words. (3) The presence of any hemiplegia, facial paralysis, or other paralysis should be noted. (4) If all these tests be negative, and still the patient is unable to communicate his thoughts properly, turn to Mental Aphasia, § 534.

1. **Paralysis of the muscles** of the lips, face, and tongue is met with in most cases of *hemiplegia* in the early stage. Here one side of the face is generally obviously affected, especially its lower part, and the tongue, when protruded, deviates to the paralysed side. The speech is thick, and often quite unintelligible. But as the face and tongue recover, the speech returns. This is a pure defect of the muscles of articulation.

2. **Partial paralysis** of the muscles of articulation is met with in its most typical form in the case of *bulbar paralysis* (glosso-labio-laryngeal palsy) and pseudo-bulbar paralysis. At first, when the tongue is chiefly affected, difficulty occurs with the linguals, L, N, T, and there is an obvious difficulty in pronouncing words, so as to give the impression of the tongue being too large for the mouth. Soon after this the labial letters also give rise to difficulty (as P, B, M) ; then the palate becomes involved, the speech becomes nasal, and difficulty occurs with guttural letters—K and G (hard). Towards the end of this affection speech is totally lost (alalia), and the patient can only utter meaningless grunts.

3. **A fine tremor of the lips** with **slurring speech** is very characteristic of *general paralysis of the insane.* The syllables are run together as in intoxication—" British Constitution " becomes " Brish Conshon." It is also met with in delirium tremens, some cases of meningitis, and occasionally in disseminated sclerosis.

4. A **drawling, indistinct,** monotonous speech is very characteristic of *paralysis agitans.*

5. **Syllabic or staccato speech** is more characteristic of *disseminated sclerosis* than the preceding ; there is a pause between each syllable, and each syllable of a word is equally accentuated (con-sti-tu-shon), as in scanning Latin verse ; hence sometimes called " scanning speech." Occasionally this kind of speech is met with in tabes dorsalis.

6. **Stammering** is a spasmodic disorder of the muscles of articulation and respiration. The condition resembles a spasmodic tic (§ 593) of the muscles of respiration initiated each time by an attempt to speak. Other tics occur in these patients. Most difficulty is experienced with words commencing with explosive or labial letters—B, D, P, T, K, or G. Excepting in bad cases, the patient can whisper or sing without a defect. In severe cases the spasm tends to spread to other muscles of the face or other parts ; the patient, for example, remaining with his mouth wide open, or his face screwed up into some contortion for an appreciable time before any sound is uttered. The affection appears in childhood, and there is very generally some hereditary or other manifestation of defect in the nervous system. *Syllable stumbling* is a variety of stammering in which one or more syllables of a word are repeated. The curability of stammering depends a good deal upon its previous duration and the age of onset. It is worse when it comes on in the adult. The *Treatment* is a matter for very careful education, and there are some very good teachers who make the cure of it their speciality. Much can be done by teaching the patient to manage his breath, and always to *take a deep breath before starting to speak.* I have frequently been able to assist such patients by instructing them to beat time with their hands, and, without actually singing, to adopt a sing-song method of speech.

7. **Lalling** or infantile speech is that in which the letters difficult to pronounce—*e.g.*, R, L, C, Sh—are avoided ; British is pronounced Bitty.

8. In **idioglossia** the child has a speech of his own, which is unintelligible excepting to those accustomed to the child. It is due to a defective power to *reproduce the sounds* of words said to him. He has to be taught by a system of lip reading.

9. **Rhinolalia operta** is the speech met with in cleft palate and paralysis of the soft palate—*e.g.*, diphtheritic—in children. In it the speech has an unmistakable nasal quality. In **rhinolalia clausa,** on the other hand, which is due to a spasm or stiffness of the soft palate, the normal nasal quality of the speech is wanting, and this produces the somewhat affected style met with in some hysterical cases.

§ **534. Aphasia** is, as Trousseau aptly put it, " loss of memory for the signs of thought." In some cases the patient's speech and power of writing are so much deranged that he cannot communicate his ideas to you ; in other cases you are unable to communicate with the patient because he cannot recognise words spoken to or written for him.

For speech and writing, the two **outgoing** processes by which we communicate to other people, two structures must be intact, and their functions normal :

(1) The motor *apparatus* of speech and writing ; and

(2) Initiating *centres* in the cerebral cortex which have been gradually educated up to the function of speaking and writing. These centres are appropriately called **ideo-motor** centres, and contain a series of educated recollections of the complicated movements necessary to produce speech or writing.

For the recognition of spoken or written language, the **incoming** processes of communication, two parts also are necessary (Fig. 154) :

(1) The sensory *tracts* of vision and hearing ; and

(2) Certain receptive *centres* in the cerebral cortex, **ideo-sensory** centres, educated up to the art of recognising and naming words heard and things and words seen (see tables, p. 770).

In mental aphasia we are concerned solely with the ideo-motor (or outgoing) and the ideo-sensory (or incoming) centres in the cerebral cortex. Defects of the motor apparatus of speech have been fully considered (§ 533), and defects of the

organs of vision and hearing are given elsewhere. In what follows we must assume that both of these are healthy.

(*a*) **Ideo-Motor Aphasia.**—Are the patient's powers of **speech** or **writing** defective ? We will first consider the out-going forms of aphasia : aphemia (ideo-motor defect of speech) and agraphia (ideo-motor defect of writing).

The commonest instances of this form of aphasia are associated with right hemiplegia. In certain cases of hemiplegia we find after a time that, although the patient recovers the use of his lips, tongue, and arm, he is still unable to name things properly or to converse, except by writing or by pantomime. This disability may exist in many degrees ; in some cases the patient only occasionally says a wrong word, in others he cannot use a single word correctly. In some of the worst cases the patient makes use of one or two words only (recurring utterances) for all he wishes to say. His *articulation* of those words which he can speak is good ; he has full power in the muscles of the face and the arm, and his hearing and vision are good. But there is a *defect in his mind* which prevents him from recalling the movements of articulation which indicate the word he wants to use. He knows the right word when he sees or hears it ; hold up a pen and suggest to him that it is a haddock ; he shakes his head and gets angry ; write on paper or say " pen," and he nods and smiles. Such a case represents one of the commonest kinds of aphasia ; it is a *loss of memory of the co-ordinated movements of speech*, and is known as **aphemia.**

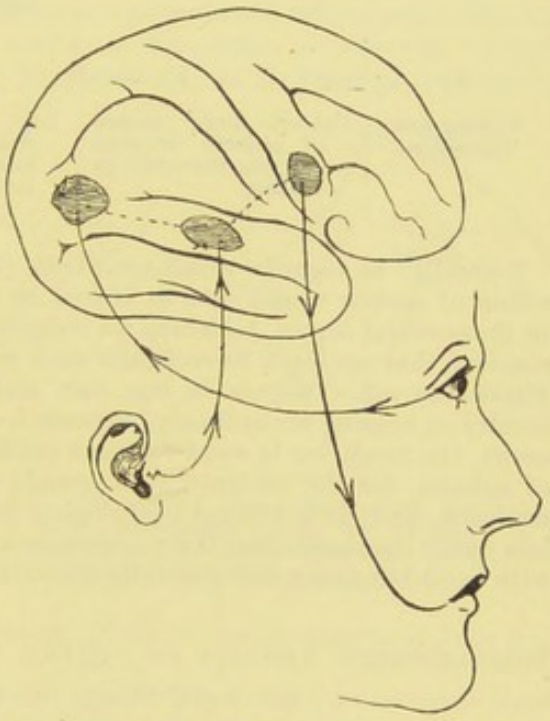

Fig. 154.—APPARATUS OF SPEECH (represented in error in the right cerebral hemisphere).—The auditory and visual ideo-sensory centres and the ideo-motor centre of speech.

Take another and rarer case, and suppose that, instead of speech, it is only the power of writing which the patient has lost. His motor power is good, his speech correct, and his vision and hearing perfect. But he cannot write a word, although he knows the word he wants to write. In short, he has *loss of memory for the co-ordinated movements of writing*, and the condition is **agraphia.**

Thus we have two kinds of possible defect of a patient's outgoing, initiating, or ideo-motor centres in the cortex corresponding to the two principal means by which he communicates his thoughts to the outer world (see table, p. 770).

(*b*) **Ideo-Sensory Aphasia** (Sensory Aphasia).—Can the patient understand **written** or **spoken words ?** Let us now consider the incoming forms of aphasia—visual aphasia (word-blindness) and auditory aphasia (word-deafness). They may be unattended by any defect of speech, vision, or hearing for ordinary purposes. But the patient has simply a loss of memory for the signs of other people's thoughts as expressed in written or spoken words.

First, as regards WORD-BLINDNESS (visual aphasia). We have a visual recording-board (Figs. 154 and 155) in the cerebral cortex, upon which our recollection of printed or written words is recorded, so that after seeing them a few times, or being educated to read, we can recognise and name them at once when seen again. If the patient has *lost the faculty of understanding and recognising written or printed words*, the condition is word-blindness (visual aphasia). This form may be met with alone. The vision is good enough for ordinary purposes, but it is a curious

circumstance that in most of such cases the patient cannot see with the left half of each retina (hemianopsia), because it so happens that lesions in the occipital lobe which produce word-blindness also cut off the optic radiations which come from one optic tract.

IDEO-MOTOR APHASIA *i.e.*, defect in the cortical mechanism of the centres of speech and writing.

	Tests.	*Nature of Defect.*	*Position of Lesions in Cortex Cerebri.*[1]
Speech loss (aphemia).	Cannot talk correctly or say names of objects.	Loss of the educated recollection of the movements required for spoken words.	Posterior end of third left (in most people) frontal convolution (Broca's) and lower end of ascending frontal (Fig. 155).
Writing loss (agraphia).	Cannot write names of objects or convey his thoughts in writing.	Loss of the educated recollection of the movements required for writing.	Posterior end of second left (in most people) frontal convolution.

Secondly, as regards WORD-DEAFNESS (auditory aphasia)—*i.e.*, the non-recognition of spoken words. As in vision, so in audition, we have a recording-board in the cerebral cortex, by which we recognise spoken words. It is by the auditory memory that we learn to recognise such sounds as " mamma " and " papa," and afterwards other sounds in our own and other languages. When a patient's faculty of hearing for ordinary purposes is intact, but *he cannot understand spoken words*, the condition is word-deafness (auditory aphasia). This is the rarest form of aphasia, for the recognition of sounds and spoken language is the first thing we learn, and, as a general neurological principle, the faculties first to come are less easily damaged than later acquirements. It is practically always associated with word-blindness, and generally also with ideo-motor aphasia.

IDEO-SENSORY APHASIA *i.e.*, defect in the cortical receptive centres for word-vision or word-hearing.

	Tests.	*Nature of Defect.*	*Position of Lesions in Cortex Cerebri.*[1]
Word-blindness (visual aphasia).	Can see, but cannot read or recognise *printed* or *written* characters. Usually with left homonymous hemianopsia.	Loss of the educated visual memory for written (or printed) signs.	Angular gyrus (visual speech centre); or in the left occipital lobe, so situated as to sever the optic radiations between the angular gyrus and the visual centre.
Word-deafness (auditory aphasia).	Can hear, but cannot understand or recognise *spoken* words.	Loss of the educated auditory memory for speech.	Posterior half of the superior temporo-sphenoidal (Wernicke's) convolution (auditory word centre).

[1] Note that these lesions are ordinarily situated in the left hemisphere, but when the patient is left-handed they are situated in the right hemisphere.

I have described these four varieties of memory of the signs of thought singly in order to make the subject clear, but they are all interdependent one on another. The complete memory for a pen, for instance, involves a visual recollection of the object and its printed sign, an auditory memory of the sound " pen," and an ideo-motor recollection of the movements used for saying or writing " pen." In point of fact, the four varieties of aphasia which correspond to these forms of memory are nearly always mixed in various proportions, aphemia being the most common, agraphia the next, word-blindness the next, and word-deafness the rarest. This renders their investigation difficult. Par-aphasia was a term used by Kussmaul to indicate a slight degree of mental aphasia with varying admixtures of aphemia, word-blindness, and word-deafness, leading to the misplacing and misapplication of words. Par-agraphia was used by him to indicate slight degrees of mistakes in writing.

Lesions.—Pure cases are, as just mentioned, very rarely met with, but now and then such cases have been observed, and we are thus able to locate the position of the several lesions in the cortex. These are given in the tables, and in Fig. 155. Usually the lesion involves the grey matter of the cortex, occasionally the white strands beneath. The changes may be structural, as in cases of hæmorrhage, embolism, thrombosis, or tumours; or functional, and leave no trace behind them.

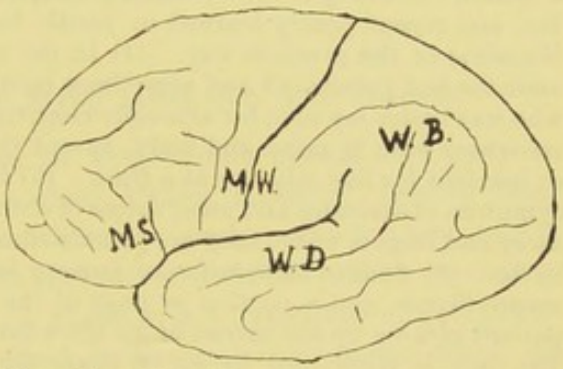

Fig. 155.—M.S., ideo-motor centre for speech. M.W., ideo-motor centre for writing. W.B., centre damaged in word-blindness. W.D., centre damaged in word-deafness.

Clinical Investigation.—One so seldom meets with aphemia, agraphia, word-blindness, or word-deafness alone that the investigation of a case of aphasia is not easy, and it becomes a question as to which of these predominates.

(1) First ascertain whether there is any, or how much, defect in the articulation or motor apparatus of the face, tongue, or arm.

(2) You will find it convenient to proceed next to investigate the patient's power of comprehending your communications to him, in the inverse order of the description I have given above.

(3) Word-deafness, the rarest form of aphasia.—Give the patient some simple spoken order—*e.g.*, " shut your eyes "—but do not accompany your spoken orders by any gesture. If he responds, there is no word-deafness; if he does not respond, he is either deaf (an unlikely thing on both sides), or he is word-deaf; and you must ascertain that he is not deaf by seeing if he turns his head towards a sudden noise or musical sound.

(4) Word-blindness.—Ascertain if his sight is good, and particularly if he appears to be able to see equally well with both halves of his retina, for word-blindness is commonly accompanied by homonymous hemianopsia. Give him the newspaper (upside down at first), and ask him to identify some of the letters. Then write out some simple instruction and show it to him; if his sight is good (except for hemianopsia), but he does not respond, there is visual aphasia (word-blindness). In some cases you can place a pen, a penknife, and a watch before him, writing out their names on pieces of paper, and ask him to place them on the objects.

(5) Agraphia.—Can he express his thoughts to you correctly in writing? Ask him to write his name, for instance, or write an account of his illness.

(6) Aphemia.—Can he remember words he wants to say, or does he use them incorrectly in conversation? Can he name objects correctly? Hold up a pen and suggest wrong names for it, finally saying or writing " pen," and learn by his gestures if he knows which is the right name.

§ **535. Defects of Memory** (Amnesia) may be (*a*) *entire*—forgetfulness for everything—or (*b*) *partial*—forgetfulness for certain facts.

(*a*) **Loss of memory as a whole** may be indicated by a forgetfulness for recent facts, such as what day of the week it is ; or for remote events, such as incidents of youth ; or by a forgetfulness of what the patient wished to say or do (intention amnesia), or where he had placed things.

Causes.—(1) *Neurasthenia* and various *debilitating conditions*, such as anæmia, convalescence from severe illnesses, general ill-health, and exhaustion from overwork are commonly attended with some defect of memory. The activity of the memory, like that of the attention (or power of concentration of the thought), is indeed a measure of the activity of the mind, and when the brain is " tired," the memory flags. (2) The memory is also temporarily impaired under the use of certain *drugs—e.g.*, large doses of the bromide—but it readily recovers when this drug is stopped. (3) In *advancing years* the memory may become permanently deficient as a normal phenomenon (senile amnesia and senile dementia). The peculiarity of senile amnesia is that it applies chiefly to recent events and to recently acquired knowledge. The patient perhaps can give us full particulars of his early life, and repeat poetry learned in youth, but is unable to mention any event of the same or the previous day. (4) In the mental condition of *chronic alcoholism*, amnesia is a prominent and sometimes incurable condition. For instance, a lady who was under my care for alcoholic neuritis and delirium three years ago, has now recovered both in mind and body, except that she is unable to recall a single fact or incident for five minutes at a time. (5) Defective memory is also met with as a symptom of *dementia* and other forms of chronic insanity. The patient puts things away and forgets where they are ; he also forgets where he is and what he was going to do. (6) *Sudden obliteration of memory* is not unknown. It may occur after a severe illness, and a portion or page of the recollection becomes blotted out, the patient picking up the thread of his life where it left off ten or twenty years before. The case is mentioned by Sir William Gowers of a clergyman aged sixty who obstinately believed he was forty, and picked up the thread of his life at that age. Sometimes such sudden obliteration occurs after epilepsy or without known cause, as in cases reported in the papers from time to time of persons who have forgotten their name and all particulars about themselves.

(7) *Dual personality* (Synonyms : " dual consciousness," " alternating consciousness ") is another mental condition, in which loss of memory is a prominent feature. It is a rare and interesting condition, in which a patient lives alternately two different lives, or rather, has two alternating mental states, which may for purposes of description be called State A and State B. The essential peculiarity of the condition is that the patient when in State B has no recollection of his thoughts and acts while in State A, and *vice versa*. In each state he picks up the thread of his life when he was last in that state. Moreover, while in State B he may behave in a totally different manner to that in which he behaved during State A. He is subject, in other words, to alternating states of consciousness and character, in which the whole mental attitude and mental record is changed. As a temporary condition dual consciousness may occur after epilepsy (masked epilepsy, or minor epilepsy chiefly), or after hysterical attacks. As a more permanently alternating condition it is found in some without this association.

(*b*) Among the **partial losses of memory**—

Mental aphasia may be mentioned. It is, as we have just seen (§ 534), a loss of memory for the signs of thought.

Verbal amnesia is really a slight degree of ideo-motor aphasia manifested by an inability to recollect the words or names when the patient wishes to speak. It occurs with a tired brain or after some emotional shock.

Visual and Auditory Amnesia.—Many interesting observations of late years go to show that the memory is compounded of different constituents. Apart from the association of ideas, there are two distinct means by which different individuals

recall an idea. (i.) Some recall an idea by a visual impression, such as the form or colour of the object it represents, or by the image of the printed word representing it which they have seen or read. This is called a *visual memory*, and people who chiefly use this form, remember those ideas best which were originally conveyed to them by pictures or objects seen, or by reading a descriptive passage to themselves. (ii.) Others can best recall an idea which is conveyed to them through their ears—*e.g.*, by sounds or by words read aloud to them—and these have what is called an *auditive memory*. Though everybody possesses both these forms of memory, they unconsciously make use more of one than the other. The visual memory of most people is better than their auditive memory, and they recall objects and pictures seen much more readily than sounds heard ; hence the great value of kindergarten and demonstrative methods of education. Charcot narrates a remarkable case of a highly educated man, a banker, who, having an unusually good visual memory, suddenly, after a severe emotional shock, lost it completely. He was conscious of a great blank in his mind, and was unable to picture to himself any of the forms with which he had formerly been familiar, such as the shape of a building, a column of figures, the colour of his wife's hair, etc. By degrees, however, he learned to substitute his auditive memory, which hitherto had lain dormant, and so he gradually became able to carry on his business again.[1]

γ. *Acute Perversions of the Mind (Delirium and Mania).*

§ **536.** Acute mental exaltation or excitement occurs clinically in two forms—delirium and mania—which differ, however, less in their clinical features than in the circumstances under which they occur. I. *Delirium* is the term applied to mental excitement which is clearly traceable to some bodily disorder of which it is a symptom or complication. II. *Mania* is the term applied to mental excitement when no such source can be traced, the mental condition being the only—or, at any rate, the principal—symptom. III. *Active* or *acute* melancholia is occasionally met with ; it only differs from chronic melancholia (§ 539) in the misery and depression of the patient being of a more aggressive character. Mania is practically always acute ; melancholia nearly always chronic.

Clinical Investigation.—The first and most important point in any given case of delirium or mental excitement to which you may be called for the first time is to ascertain the temperature, for the most useful clinical division of the causes of delirium is into *Febrile* and *Non-febrile*. Secondly, it is important to make a thorough and complete investigation of all the organs of the body, to ascertain whether there be any local inflammatory disorder, such as pneumonia, with which delirium may be connected, either directly or indirectly. I remember once overlooking a case of latent pneumonia, and consigning it to the lunatic ward of the workhouse. The urine also should be carefully examined for albumen, sugar, or other abnormality. Thirdly, an inquiry should be made into the history of the malady and of the patient, especially as regards alcohol. In reference to the etiology of delirium, three important *predisposing causes* have to be borne in mind. First, there is a marked predisposition in some nervous people to develop delirium in presence of a slighter cause than would be operative in others. Secondly, there is a marked hereditary tendency towards the same vulnerability ; and thirdly, excessive drinking predisposes to the occurrence of delirium after an injury, operation, and many diseases which are not usually so attended.

[1] " Leçons Clinique sur les Maladies du Système Nerveux," tome iii., Leçon 13.

I. The causes of **delirium** have been considered (§ 345), and need only be here enumerated.

Febrile.	*Non-Febrile.*
Diseases of the brain—especially meningitis.	Delirium tremens.
Acute visceral inflammations.	Chronic renal disease.
Acute specific fevers.	Post-febrile delirium.
Delirium tremens (rare cases).	Reflex delirium.
	Deliriant drugs.

II. **Acute Mania** may supervene suddenly—(1) during convalescence from exhausting diseases (as previously mentioned); (2) in the course of other diseases of the nervous system—*e.g.*, G. P. I.; (3) in the course of some other form of insanity. Its onset is usually rapid, tongue-tremor being often met with in the early stage (Dr. G. H. Savage). The stage of excitement is soon reached—loquaciousness, sleeplessness, continual restlessness, incoherence, in which delusions and ideas succeed each other with great rapidity, sometimes relating to moral and religious, at other times to intellectual topics. After lasting some weeks or months, recovery (sometimes quite suddenly) ensues; sometimes it is followed by moral or mental obliquity or dementia; rarely it passes into chronic mania. The temperature is normal throughout. In most cases there is a tendency to relapse.

Acute Delirious Mania (Bell's Mania) is an acute maniacal condition coming on suddenly in a person in apparent health, attended by pyrexia, usually running a rapidly fatal course, no lesions being found after death. It is happily a somewhat rare disease. The symptoms come on abruptly, and quickly amount to frenzy, accompanied by outbreaks of great violence. The temperature ranges irregularly from 100° to 104° F., and in the course of one to three weeks the disease terminates in great bodily prostration, and usually in death. Acute delirious mania differs from acute mania in the elevation of temperature, the rapid wasting, and its more rapid and fatal termination. It resembles some cases of enteric fever very closely, acute pneumonia and acute meningitis, but their proper symptoms are absent.

The *Treatment* of mania consists mainly in the administration of food (with stimulants if the pulse so indicates). Narcotics and depressants may be tried, and Dr. Savage mentions a case which apparently got well under frequent small doses of opium. For acute delirious mania the wet pack or the graduated bath (§ 392) may be given. Professor Osler recommends venesection.

III. **Acute dementia** ("**stupor**") is a rare condition of sudden ablation of the mental faculties.

δ. *Chronic Perversions of the Mind* (*Insanity*).

§ **537.** This is a more complex group than any of the preceding, and it forms the collection of morbid conditions of the mind, known as insanity. The subject may be briefly dealt with under—

Clinical Investigation.—I have already, in § 513, referred to the importance of tact and general knowledge in investigating psychical disorders, and I may here mention two other points: (1) Get your data in chronological sequence as far as possible; and (2) never be in a hurry. Any careless, inept, insistent, or

rapid questioning will only confuse and silence your patient and defeat your object. Gain his confidence ; let *him talk to you* first of his favourite hobby, then of his thoughts and feelings, and finally of his delusions or hallucinations. The main points to investigate in mental cases are sleep (and dreams) ; speech ; writing ; memory ; decision and will ; reasoning power ; moral and ethical standards ; delusions ; hallucinations ; and, lastly, whether the patient regulates his conduct according to these—*i.e.*, whether he (or she) is a potential danger to himself or others. Before committing yourself to an opinion never omit to ascertain from the relatives how far the patient's present differs from his previous character and conduct, for conduct that is mad in one person is normal in another.

The three terms, *delusion*, *illusion*, and *hallucination*, strictly speaking, have different meanings, but they are used somewhat laxly and indifferently. A hallucination is a false sense impression without any external stimulus—*e.g.*, the hearing of voices and the seeing of snakes. *Illusions* are distortions of external stimuli, as in alcoholic delirium, when a lady's muff is thought to be a cat. *Delusions* are false ideas existing in the mind of a patient, as when he believes himself to be the Deity. Hallucinations are met with particularly in mania, delirium, and mania *e potu*, and it is surprising what minute details can be given to us about these creations of the mind. Hallucinations of sight (rats and snakes) are much less common than those of hearing (hearing voices). Hallucinations of taste and smell are present not infrequently in association with delusions of being poisoned. Hallucinations of common sensation are both frequent and various, and are especially frequent at the climacteric and in hysterical subjects who become insane. Amongst the varieties of sensation hallucinations of the male or female sexual organs are not infrequent, and in this way false accusations may be made against those (especially doctors and nurses) with whom the patient comes in contact.

In regard to all delusions, illusions, or hallucinations, it is important to estimate as far as possible how far such perversions of the mind influence, or are likely to *influence, the acts or conduct of the individual.*

After noting the age of the patient, the first question to investigate in the history is whether the attack came on with excitement or with depression. If with excitement, the case may be one of mania or general paralysis of the insane or delirium (§ 536). If the case came on with depression, it may be melancholia or G. P. I. If the patient is an adult, start at § 538 ; if an adolescent, at § 545 ; if a child under ten, start at § 546.

§ **538.** I. **Chronic Mania** is simply a prolonged form of acute mania, as previously mentioned (§ 536), lasting with less excitement for years, instead of for weeks or months. Recurrent mania is that which recurs, sometimes at the menstrual epoch. " Folie circulaire " is an alternation of mania and melancholia, with lucid intervals. Monomania was used by Esquirol to indicate a form of delusional insanity (§ 542) dominated by one fixed idea ; it is not really mania.

§ **539.** II. **Chronic Melancholia** is a morbid condition of miserable self-consciousness and self-negation without hope. Melancholia occurs under three circumstances : (1) It may be part of some other mental disorder, such as G. P. I., or a stage (first or third) of mania ; (2) it may constitute the whole of the mental disease without previous ill-health ; or (3) it may supervene on neurasthenia or some bodily ailment. The onset is usually insidious, and commences with extreme self-consciousness, combined with sadness, as indicated by tears without cause, and when the patient is remonstrated with he is irritable. There are morbid dreads of impending calamity which cannot be named, sleepless nights, and a suicidal tendency. Among the physical signs commonly noted in such cases are feeble circulation, as evidenced by cold feet and chilblains, and constipation. Melancholia differs from hypochondriasis in the " hopelessness " of the former and apathy to surrounding conditions.

Four *varieties of melancholia* are described—active, passive, and simple melancholia, and melancholic stupor. (1) and (2) *Active* and *passive* melancholia depend upon the degree to which patients give expression to their grief. In the former they are always imparting their trouble to someone ; it is in reality an acute melan-

cholia. In the latter they sit for hours together in a dejected state, and it is sometimes difficult to elicit their leading delusion. (3) *Simple* melancholia may be described as melancholia without definite delusions. It consists simply of misery, sleeplessness, self-blame, and inability to continue at work. This form is common in the overworked or much-worried, and in women at the climacteric. Suicide is not uncommon in these cases, and precautions, which are sometimes neglected on account of the simplicity of the affection, should not be omitted. Otherwise the prognosis is favourable. (4) In melancholia with stupor (melancholic stupor) the patients remain speechless and motionless, with an aspect of abject misery. Their limbs may be flaccid or in cataleptic rigidity. They are abstracted and oblivious to all external stimuli. They resist external interference, but are not usually violent. The condition may come on suddenly or gradually, and last for months or years. Some cases end fatally in a short time. Some have recurrent periods of exaltation. Some terminate in a condition of permanent weakmindedness, and a few recover. It is equally common in both sexes, but is more frequent in the young than the old. Sometimes it follows a severe and exhausting illness, and sometimes it follows acute mania.

Course and Prognosis.—The melancholic process is longer than the maniacal one. The duration varies considerably, but lasts an average of some three to twelve months. Relapses are not infrequent. The slower the advent of the disease, the slower is the recovery. Recovery is common enough in the young, the prognosis being worse as age advances. Suicide is frequent in all forms, but death from the disease is rare. Heredity is an important factor, and the nutrition of the body at the time is another. The melancholia of pregnancy is favourable, but the melancholia of lactation is always grave. It is interesting to remember that cases of melancholia may recover even after a very long time (nine, ten, and thirteen years have been recorded). There is a distinct *suicidal tendency* in all cases of melancholia, but those are specially liable, according to Dr. Savage, who have delusions of impotence, of being followed or persecuted, of hearing voices, of being the cause of injury to their relatives, or who are suffering from great physical weakness or bodily disease. The tendency to suicide appears to increase with age, and suicidal cases generally have an insane heredity, and often a suicidal heredity also. Patients generally have a special predilection for some particular mode of death—one to poisoning, another to drowning, another to hanging, and another to blowing his brains out, and they will often avoid other means which may happen to present themselves.

In regard to *Causation*, melancholia depends in most cases more upon physical and external than upon mental and moral causes. Some of the commonest causes have been referred to under the variety Simple Melancholia. No doubt want of society, solitary habits, combined with a sedentary life, in which the person is debarred from genial companionship, are the prominent causes of this condition. A general depression of the vital powers—*e.g.*, from bodily disease, fevers, heart disease, etc.—is an important factor. Any age may be affected, but it mostly arises at or after middle life.

Treatment.—In the simpler cases, such as those referred to under Simple Melancholia, a few weeks' rest under supervision, with a pleasant companion and complete absence of the conditions under which the disease arose, followed by a few months' easy travel, will generally set the patient right. If the interest can be aroused, and the attention attracted for a sufficient time, much can be done to relieve the condition and even remove the delusions. Feeding is necessary, and in case of refusal it may be done by means of (*a*) a spoon, pouring the fluid into the cheek beside the teeth, or (*b*) by the nasal or stomach tube. The quantity thus administered should be equal to 5 pints of milk, 2 pints of strong beef-tea, 6 eggs and 3 to 6 ounces of brandy per diem. Suicide must be prevented by removal to an asylum or careful watching at home.

§ **540.** III. **Dementia** is deficiency of all the mental faculties, coming on in adult life. It comes on as a *primary* condition in (*a*) chronic alcoholism, and (*b*) advanced life (senile dementia). The first, as we have seen, shows itself especially

by a loss of memory. The second also has the same peculiarity, with the additional feature that the memory is lost for recent events only. Dementia comes on as a *secondary* condition in (*a*) general paralysis of the insane, and as the concluding stage in many other forms of mental disease ; and (*b*) after vascular and other gross intracranial lesions. Even after a small lesion of the brain the mental capacity for business is hardly ever as good as before its occurrence, and the patient often becomes childish, peevish, forgetful, emotional, and by degrees in severe cases, completely demented.

The **Special Forms of Insanity** are general paralysis of the insane ; delusional insanity ; emotional insanity ; moral insanity ; alcoholic, syphilitic, and puerperal insanity ; catalepsy and trance.

§ **541.** IV. **General Paralysis of the Insane** (G. P. I. ; Paralytic Dementia) is a progressive generalised muscular weakness and tremor, accompanied by mental symptoms, often of a grandiose character, occurring almost entirely in young men or men in the prime of life ; due to atrophy and a scattered sclerosis of the cortex cerebri. This change represents a later stage of some nutritional alteration in the nerve-cells ; Dr. W. F. Mott has shown that the changes in the cerebral cells (swelling) produced by ligature of the cerebral arteries, and so cutting off the nutrition, resemble those found in the early stages of G. P. I. He has also shown that this, like locomotor ataxy, is undoubtedly syphilitic. The disease depends on a parasyphilitic process ; *i.e.*, it is an indirect or nutritional effect of the syphilitic toxin (resembling tabes dorsalis in this respect), not a direct manifestation of syphilitic lesions (see also footnote 1, p. 838).

Symptoms.—Paralysis of the limbs may sometimes exist for many years without mental symptoms (*vide infra*). In my own experience, mental have generally preceded the physical symptoms, but this order varies. The characteristic symptoms are mental alteration, general weakness, tremor, and alterations in the pupils and the speech. They are divided for convenience into three stages, each of which lasts about one year. In the first or *premonitory stage* (the stage of irritability and muscular tremor) irritability, restlessness, perversion of the moral sense, and loss of the faculty of attention are among the most usual features, though a variety of other mental aberrations are met with. A man of even temper, who has been a fond husband and father, becomes irritable over trifles, gives way to coarse and blasphemous language, exhibits sexual aberrations, or commits thefts. He becomes egotistical, showing the delusions of grandeur so characteristic of the malady, and squanders his money. He may believe himself to be very strong, very wealthy, or very high born. Sometimes, on the other hand, great depression is the characteristic of this stage ; and thus, even in this period, we find two groups, one with a tendency to expansion and restlessness, the other with a tendency to despondency and loss of energy. Accompanying, preceding, or following the mental symptoms are various *physical changes*, amongst the commonest of which are

(i.) tremor (fine, small, and rhythmical) of the hands, (giving rise to characteristic handwriting), and of the lips and tongue (giving rise to a very characteristic speech—viz., a slurring of the words as in intoxication). (ii.) The pupils in this stage are usually small, very contracted, and immobile to light ("pin-point pupils"); very often unequal. (iii.) Headache, neuralgia, and various subjective sensations are complained of. (iv.) Weakness of the limbs, always generalised, and sometimes combined with some inco-ordination. As a rule, the knee-jerks are increased in G. P. I. Sometimes the symptoms of lateral sclerosis are present. Anæsthesia and other alterations of sensation and of the special senses are frequent. The *second stage* (stage of fits) is characterised by (i.) mental enfeeblement, which replaces the exaltation in the first stage; (ii.) increasing muscular weakness, difficulty in walking any distance, and especially in the act of turning, sometimes combined with giddiness; (iii.) fits are almost invariably present at some time during this stage; they vary in character, but are usually syncopal or epileptiform, with or without the loss of consciousness. Sometimes they consist of attacks of numbness of the limbs, or aphasia, or coma. The *third stage* is the stage of progressive mental extinction. The speech becomes inarticulate, the paralysis extreme, and may be accompanied by contracture, so that the patient cannot feed himself. His mind undergoes progressive extinction, and there is loss of all its faculties. The urine and fæces are passed involuntarily.

Many different *varieties* have been described, but these only exist in the earlier stages of the disease; they all tend to one common form of progressive mental enfeeblement. (1) The expansive variety is the commonest, and forms the basis of the above description. (2) The melancholic variety is characterised by great depression and melancholic delusions. (3) In the paralytic variety, paralysis and tremor predominate, with few or no mental symptoms, excepting occasional outbursts of emotion and some change in character. Sometimes the paralysis predominates in the legs (ascending variety); the legs may be rigid (spastic form, Savage); and there is an ataxic form closely resembling tabes dorsalis at the outset. (4) A congestive variety has been described, chiefly characterised by fits of various kinds.

Course and Prognosis.—The duration varies widely from a few months to three or more years, and the proportion occupied by these various stages differs greatly. One thing is very characteristic of the disease—remarkable intermissions of comparative or complete return to health. But these cases always break down on attempting to resume their former state of life. The expansive form above described usually runs its course in about three years. Where depression and melancholia are marked features, the prognosis is worse (Bristowe), and the disease is more rapidly fatal. The spinal paralytic or ascending form occupies a much longer period, and may extend to six or eight years. When

the malady is once established, it invariably progresses towards a fatal termination.

On account of its great variety, G. P. I. has to be *diagnosed* from many different complaints : (*a*) From other forms of *mental disorder*, especially alcoholic insanity, chiefly by the tremor, speech, the pupillary changes, and the progressive lethal paralysis ; (*b*) from other diseases giving rise to *generalised paralysis* (see § 573) ; (*c*) maladies attended by tremors and other neuro-muscular symptoms, such as *disseminated sclerosis* and *paralysis agitans*. Chronic *alcoholism* and *peripheral neuritis* are difficult to differentiate sometimes ; they are recognised by a history or evidence of alcoholic dyspepsia, and by absent knee-jerks. *Bulbar paralysis* is recognised chiefly by its symptoms being confined to the mouth, tongue, and throat, the mind being usually normal. Lumbar puncture (pp. 921 and 923) greatly aids our diagnosis of G. P. I. The diagnosis from *tabes dorsalis* is not usually difficult, but these two diseases are very apt to occur in a mixed form.

Causation.—(1) Adult males, in the very prime of their strength and manhood—that is, between thirty and forty—are the favourite subjects of the disease, but it may occur at any age. The youngest I have known was sixteen, the oldest sixty (an elderly man who married a young wife). It is generally said to be four times as common in men, but I believe it to be commoner. It is more frequent in the lower classes of life. A neurotic heredity is said to be in operation in as much as 30 per cent. of the cases. There seems but little doubt, however, that the disease is in its essence a syphilitic, or rather parasyphilitic process, as above mentioned. Alcoholic, sexual, and other excesses, anxiety, and mental fatigue are accessory causes.

Treatment.—Something may be done in the way of *prevention* when there is a history of heredity by the avoidance of overstrain and of alcohol. Patients with a hereditary taint and premonitory symptoms such as the above should certainly avoid matrimony. The most important *remedial* measure consists of the removal of the patient from the conditions under which the disease has arisen, and especially avoidance of business and all causes of anxiety or mental strain. He should live a regular life, with outdoor exercise and amusements which take him out of himself. Iodides and nerve tonics (especially cod-liver oil) sometimes do good in the earlier stages. When excitement is present the head should be kept cool (possibly with an icebag) and the feet warm, and the bowels should be freely opened. Physostigma or hyoscyamin may be given ; and if much excitement is still present, 30 minims tinct. digitalis every four hours, or a warm bath followed by an icebag. The cold pack is also useful, and bromide and sulphonal. In the melancholic varieties, arsenic, iron, and quinine are the only remedies that have been found useful. Galvanism to the central nervous system has not, so far, been attended with much success.

§ 542. Other Special Types of insanity are named according to their clinical features, such as delusional, hysterical, and moral insanity; or according to their etiology, such as alcoholic, syphilitic, and puerperal insanity.

V. **Delusional Insanity** is a chronic form of insanity, in which the leading or sole mental alteration consists of a fixed delusion or hallucination, which modifies the conduct of the individual (compare Clinical Investigation, § 537). A delusion, illusion, or hallucination may arise under three conditions: (i.) It may arise in a person who is otherwise perfectly sane; (ii.) it may be associated with other evidences of insanity; or (iii.) it may, when no other symptom is present, constitute in itself delusional insanity—when, that is to say, it controls the conduct of the individual. Delusions, especially on religious subjects, are not at all uncommon in the so-called sane. But when these delusions modify the acts or conduct of the individual and lead him to act in an unusual manner, the condition becomes one of insanity. Patients who believe they are persecuted or followed are apt to be dangerous, and either homi- or sui-cidal. **Paranoia** is a term which has long been in use in Germany, and has gradually been introduced into England. My conception of the term is that it is almost identical with delusional insanity, but with a tendency to hallucinations. It does not always amount to insanity, since it may be applied to the eccentric personages who have some marked and permanent delusion, but who are otherwise sane. Such subjects are, however, responsible for many crimes. Voice-hearers and those with delusions of love or persecution are the most dangerous. **Monomania** is a term, originally used by Esquirol, which has now almost fallen into disuse. It indicates, strictly speaking, a mental state which is dominated by one fixed idea or delusion—usually of self-importance—and in this sense it might be a symptom of G. P. I. Later on, its significance has become more general, and it has been used by most to indicate a chronic form of insanity (which may follow an acute attack) in which the patient, though fairly sane on most subjects, has some one intellectual peculiarity or delusion. Many such persons are at large, and pass for sane people.

VI. **Hysterical or Emotional Insanity.**—The mental perversions to which hysterical subjects are occasionally liable are (1) emotional insanity; (2) hystero-epilepsy with insanity; (3) ecstasy; (4) catalepsy; and (5) trance. The first is an emotional mania often of a religious kind—a religious veneration for the curate, for instance. The patients are rarely or never suicidal or melancholic, though they may be passionate, mendacious, mischievous, crafty, noisy (screaming and singing hymns), and given to various kinds of movement, such as hammering and dancing, or to the striking of attitudes (as in ecstasy). Perversion of the tastes and the appetite and a general capriciousness are very characteristic.

Cases of the class under consideration are relatively frequent, and in general terms the *Prognosis* of such cases is more favourable than many other forms of insanity, especially if no hereditary mental taint exists, and due skill and judgment are employed in their treatment. In the *Treatment* of these hysterical mental affections we should, as far as possible, avoid putting them to bed, for complete want of will—that is, lack of energy and initiative—is a characteristic feature, and they will soon become bedridden. Change of environments, interests in life, and judicious companions are the central points. It is seldom necessary to send them to an asylum. Artificial feeding may be required, but should be discontinued as soon as possible. Savage suggests adding salt to create an artificial thirst, and then placing fluid nourishment in the way of the patient when, as sometimes happens, she is too lazy to feed herself. Laziness leads to dirty habits and the passing of fæces in the bed. We should look out for tubercle in these cases. Organic disease of the brain has sometimes been found.

VII. **Moral Insanity** is recognised by some as a special form of insanity, in which the mental disorder consists principally, and sometimes solely, of a marked deflection from the normal standard of morality. The intellect and the will may be normal, and the emotions under control. In the adult, moral and ethical perversions occur (1) very frequently as an *early phase* of G. P. I., and many other forms of insanity, for, as Esquirol remarked, moral alienation is but the "first step to

madness." (2) It is also met with in the adult *after recovery* from an attack of mania or other mental disorder. The patient, especially if young, is frequently left with a sort of moral scar, and the lower or animal side becomes prominent. (3) Occasionally one finds in adults a moral defect as a *substantive condition* without previous insanity and without any other mental defect. But often the childhood of such patients presented some similar defect in a less degree, for the condition is more frequently hereditary and congenital. *Kleptomania* (in which the patient is afflicted with an irresistible impulse to thieve) belongs to this class. Other patients have irresistible erotic or amorous tendencies, and others have a tendency to unnecessary exaggeration and lying. Dipsomania is a paroxysmal, irresistible craving for drink. (4) *Moral insanity* as a substantive disorder is chiefly met with *in children*, particularly those of alcoholic, insane, or epileptic parents. In early childhood they may be perverse, mischievous, cruel, untruthful, or thieving. They are often precocious, and they may even be intellectually gifted. Nevertheless, such children generally need incarceration in course of time, if they do not find their way into prison. It seems probable that a large proportion of the criminal convictions among the children of the lower orders are subjects of this malady. The condition, as a rule, is incurable, unless by educational and disciplinary measures.

VIII. **Alcoholic Insanity.**—Alcohol is generally accorded the chief place in the causation of insanity. It may result in two special forms: (*a*) delirium tremens, and (*b*) alcoholic dementia; it may also result in (*c*) delusional and other forms of insanity.

IX. **Syphilitic Insanity.**—(*a*) After contracting this loathsome disease it will prey on the minds of some persons to such an extent as to produce an extreme degree of hypochondriasis, to which the term *syphilophobia* is aptly applied. In every symptom, normal or abnormal, they see the disease. (*b*) Syphilitic endarteritis may lead to a *dementia* indistinguishable from senile dementia except by the age of the patient. Various forms of mania and melancholia may also be associated with arterial and gummatous lesions, or with concurrent cachexia. (*c*) General paralysis of the insane is a parasyphilitic process (§ 541).

X. **Puerperal Insanity** is a generic term for the mental disorder which arises under three different conditions. (*a*) The mental perversion which arises during *pregnancy* is always a form of *melancholia*, and varies in degree from a simple exaggeration of the morbid longings and perverted tastes which are more or less usual during gestation to melancholia of a pronounced type, accompanied, perhaps, by delusions and a suicidal tendency. It is not generally serious unless heredity is in operation, and usually disappears after the confinement. (*b*) Puerperal *mania* (or puerperal insanity proper) arises usually between the first and fourth weeks after delivery, coming on usually suddenly with maniacal symptoms. (*c*) The insanity of *Lactation*, or post-puerperal insanity, is a form of *melancholia* which arises during the first two or three months, or any time during the first twelve months after confinement. All three forms are apt to recur in succeeding pregnancies, but unless there is a hereditary taint of insanity the prognosis is good for recovery. Abundant food and removal from home are indicated, and special measures should be directed to meet the tendency to suicide or infanticide which is frequently present even in the mildest cases, and gives no indication of its presence until some untoward event occurs.

XI. **Catalepsy** (*καταλεψις*, a seizure or attack) may be defined as a state in which the patient is deprived of sensation and voluntary motion, in which the limbs remain in any position in which they are put. The patient may appear, but it does not follow that she is, unconscious to the surroundings. The eyes may be open, but she appears totally oblivious to all the outside world, and she may lie for hours, or days, perhaps, passing her motions under her. These patients are nearly always of the female sex, and are invariably the subjects of some other hysterical manifestations, to which category, indeed, the condition belongs. Sometimes these cataleptic attacks are ushered in by hysterical convulsions or a hysterical faint. They are usually determined by a fright or some emotional storm.

XII. **Trance** is a condition allied to catalepsy, in which the limbs are either rigid, or, more usually, flaccid, and lacking the feature of remaining in any one position in which they are placed. The patient may remain for weeks or months in what seems to be a faint, taking no notice, eating no food, making no movement, and scarcely breathing; though here again she may not be as unconscious as she seems. The pulse is hardly perceptible at the wrist, and unless the patient is forcibly fed she may ultimately die; but it is surprising the length of time she may live with hardly any nourishment.

§ 543.—**Prognosis and Treatment** of insanity in general.—The *Course* and *Prognosis* in several of the various forms of insanity have been referred to. In general terms the chief points on which the prospect of recovery depends are (1) the absence of heredity, especially direct heredity; (2) the rate of onset of the attack, being more favourable in a rapid than a slow, insidious advent; (3) the duration of the attack before the patient comes under treatment; and (4) the kind of insanity present.

PROSPECT OF RECOVERY IN CHRONIC MENTAL DISORDERS.

Good.	*Moderate.*	*Bad.*
Hysteria and emotional. Alcoholic. Syphilitic (mostly). Puerperal.	Delusional. Mania. Melancholia.	G. P. I. Moral insanity. Dementia.

The *Treatment of insanity* in detail has been referred to under the different forms, but the general principles resolve themselves into four indications: (1) Feeding; (2) change of environment; (3) placing under restraint; and (4) treatment of any physical defect discoverable. Hypnotism (as below) is available for some of the slighter cases, especially where alcoholism is in question.

The question of **removal to an asylum** depends on many things, chiefly (i.) the manageability of the patient; (ii.) the means at home for control; and (iii.) the character of the mental disorder and its potentiality for homicide or suicide.

Any mental patient, however mad, can be taken care of by his or her relatives *without certification*, provided it is done without payment, they being responsible for the patient's safety.

Cases of slight eccentricity and *uncertifiable* mental aberration may be received into the house of a medical man or other householder for payment; but directly a case becomes *certifiable* (in the opinion of the Commissioners) it must be placed under certificate. The penalties for breach of this are very heavy. No medical man or other householder may retain in his house more than one certified patient at a time.

A mental patient can be received at a private asylum as a voluntary boarder *without certification* if the patient writes a letter to the Commissioners in Lunacy (Victoria Street, London, S.W.) stating that he wishes to go there as a voluntary boarder.

Procedure for Removal of Lunatics and Alleged Lunatics.—The procedure for removal is somewhat intricate, and it is useful to remember that the relieving officer of the parish is a most convenient person to apply to, bearing in mind, however, that it is no part of his duty to undertake private cases, but that, nevertheless, if he be approached with due regard to the importance of his office, he may save those concerned a great deal of trouble, and supply them with all the necessary forms and particulars as to modes of procedure. He is also in constant relation with the lunacy justices.

A person deemed to be a lunatic, and found *wandering at large* not under proper care, can be apprehended by a "constable, relieving officer, or overseer" of the

parish, and taken to the workhouse. Any person, either *pauper* or *non-pauper*, deemed to be a person of unsound mind can, for his own safety or that of others, be removed from a dwelling-house by a relieving officer to the workhouse. In either case the patient can be detained there for three days upon the certificate of such constable or relieving officer, and, further, upon the certificate of the medical officer of the workhouse, for a total of fourteen days. Meantime the procedure under No. 3 (*c*) below can be instituted. This method is now often utilised for persons in all classes of life who are dangerous and away from their friends.

In *private cases* the urgency order (1, below) can be used in urgent cases. This holds good for three days from date of signature; if not urgent (2) is the usual method.

All the different forms necessary are procurable from Shaw, Fetter Lane, London, or, as previously mentioned, from the relieving officer.

A patient can be removed to an asylum in England or Wales in five ways:

(1) Under an Urgency order signed by a relation (or guardian) and one doctor.

(2) Under a Reception order of a Justice obtained by petition of relative on two doctors' certificates (used also for certification in a case for single care).

(3) Under a Summary Reception order of a Justice,

(*a*) On information from the police or relieving officer that a non-pauper is cruelly treated or neglected, a Justice calls in two doctors, who certify insanity.

(*b*) On information from the police or relieving officer that any person, pauper or not, is wandering at large deemed to be a lunatic, a Justice calls in one doctor who certifies insanity.

(*c*) On information from the relieving officer that a pauper is deemed to be a lunatic, a Justice calls in one doctor who certifies insanity.

(4) Under an order after Inquisition, being a written authority from the "Committee" of the person, together with an office copy of the order of the Court of Chancery appointing the "Committee." The "committee" is a legal phrase for the guardian appointed by the court.

(5) Under a Reception order by two Commissioners (rarely done), who call in a doctor who certifies insanity.

Idiots and imbeciles (from early life) can be removed to an idiot or imbecile asylum on one medical certificate and a statement by a relative.

The procedure in Ireland and Scotland is somewhat different.

Testamentary Capacity.—The intricate technicalities of the lunacy law cannot be entered upon here, but a knowledge of what constitutes the testamentary capacity of a patient is of great importance to the practitioner, because it is often on his evidence that courts of justice decide such matters. The testamentary capacity of a person of unsound mind depends practically on three questions:

1. Did he at the time understand the nature of a will and its effects, and did he understand the extent of the property of which he was disposing?
2. Did he provide for his relatives, or, if not, why did he leave them out?
3. Had he any delusion bearing on testamentary matters?

If these questions can be satisfactorily answered and proven, the will is valid, however eccentric the patient may have been, or even if he was at the time a certified lunatic. The fourth question—undue influence—is a non-medical question.

§ 544. **Hypnotism.**—Hypnosis may be defined as a condition resembling sleep, in which the subject's capability to receive and act upon suggestions is greatly increased. This increased suggestibility is made use of by the operator for the implanting of new and healthy conceptions and the removal of morbid ideas, the object being to influence the body through the mind. It is worthy of study both from a psychological and a medical point of view. It explains many of the cures performed at Lourdes and other shrines, and by faith-healers in all parts of the world.

Bernheim asserts that 80 per cent. of his hospital patients are hypnotisable to the extent of somnambulism, characterised by amnesia on waking; in his private

practice the proportion was much less. Dr. Lloyd Tuckey and the author, in the course of some experiments at Paddington Infirmary, estimated that only about 5 per cent. of the patients there were hypnotisable to that degree. Anæmic young women are perhaps the most easily hypnotised, but strong and healthy men are often susceptible, and I have often found other people in whom there is not the faintest suspicion of hysteria are subject to hypnotic influence. Wingfield found the undergraduates at Cambridge particularly good subjects, and soldiers and others accustomed to respond to the word of command are very amenable to hypnotic suggestion. Children, too, are good subjects when able to understand what is expected of them, whereas the insane and imbecile are generally unhypnotisable, and hysterical women are by no means the best subjects. It is only by trial one can determine whether a person is hypnotisable.

There are various *methods of hypnotising* which are easily acquired. The patient should be put at his ease and seated in a comfortable chair. He is made to relax all his muscles and to fix his gaze on a bright object held about 12 inches above the eyes, so as to cause a slight strain and convergent strabismus. This is the method used by Braid, and most susceptible persons will fall into a cataleptic or somnambulic condition when submitted to it without further procedure. But hypnosis is often assisted by verbal suggestion of sleepy sensations, such as heaviness of the eyelids and limbs, and increasing torpor of mind and body. "Passes," as the mesmerists called them, made by passing the hands, fingers extended, about ½ inch from the face so as to create a slight draught of air, are often helpful in producing and deepening hypnosis. Whatever method is used, the *rationale* is the same—it consists of monotonous stimulation of one or more senses, with corresponding inhibition of others, leading to a condition of altered consciousness, in which organic functions as well as mental states become more under the control of the will. The deeper the hypnosis, the greater, as a rule, the effect of suggestion, but good therapeutic results are obtainable when only a slight drowsiness is produced. The patient should be allowed to rest quietly for half an hour, and should be told to awake without shock or feeling of discomfort at the expiration of that time, either spontaneously or on a given signal. The suggestions should be given in an authoritative and impressive manner, and may be accompanied by manipulation of the affected part as practised by Braid. They are directed towards the removal of pain, spasm, and other symptoms, and the re-establishment of normal functions. Such suggestions often act immediately, and the action in successful cases is continuous and sustained.

Hypnotism should be used only by medical men, and with proper *precautions*. The consent of the patient and his friends should be obtained, and a third person should be present during the operation. In competent hands no bad effects result from its employment even over prolonged periods, but much evil, moral and physical, might follow the abuse or misuse of this powerful agent. Its use for purposes of public exhibition should be forbidden by law.

Uses.—Therapeutically, hypnotism has been employed to relieve pain, to procure sleep and rest, to remove delusions and obsessent ideas—*e.g.*, agoraphobia—to cure the effects of grief and shock, to reform alcoholics and moral perverts, to cure various neuroses, such as nocturnal enuresis, writer's cramp, to relieve various hysterical manifestations such as anæsthesia, attacks, and paralysis. Witterstrand, of Stockholm, claims to have cured many cases of epilepsy by keeping patients in a state of profound hypnosis for three weeks continuously. In exceptional cases hypnotism has been employed as an anæsthetic in surgical and midwifery practice.[1]

§ **545. Mental Deficiency in Adolescence** (ten to twenty years) may (1) consist occasionally of one of the chronic mental disorders of adults

[1] Those who wish to study the subject of hypnotism more closely should consult "Psychotherapeutics; or, Treatment by Hypnotism and Suggestion," by Dr. Lloyd Tuckey, 5th edition, London, 1907, Baillière, Tindall and Cox; "Hypnotism: Its History, Practice, and Theory," by Dr. Milne Bramwell; or the classical works of Charcot and Bernheim.

previously described, and especially the MORAL INSANITY described in § 542 ; or (2) it may be a SEQUENCE of either of the two groups below (congenital and non-congenital mental deficiency of childhood) (see § 546). (3) There is also a PRIMARY MENTAL DEFICIENCY inherent in the individual, and manifested for the first time in adolescence, and it is this last class with which we are now concerned ; it includes the " borderland cases," and constitutes at once the puzzle of the alienist and the trial and burden of relatives and guardians.

Symptoms.—Various kinds and degrees of defect may be met with. It is nearly always between the ages of ten and twenty that such cases come under notice for the first time, sometimes because " they will do stupid things," sometimes for " romancing " or lying, sometimes because they do not learn as rapidly as others, or " are not so bright "; sometimes because they are unruly, or have taken to drink or other vices. In the last-named case, when they have a moral obliquity, they may be possessed of brilliant intellectual gifts, but more often there is an all-round deficiency, and they are and remain childish all their lives. This deficiency leads them to consort with all sorts and conditions of men and women, whose habits and language they quickly imitate ; if sent for a sea voyage, they are generally to be found in the forecastle or steward's pantry, and rapidly assume the indelicate language of the seafaring man. Their parents expostulate with them, and they promise, and do try, to avoid repeating the offences, but they soon break out again. Much trouble accrues to the parents or guardians of such persons to keep them, when belonging to the well-to-do classes of life, from squandering a fortune, forming an ill-judged liaison, or getting into other and worse troubles, and when belonging to the lower classes, to keep them out of prison.

Causes.—In nearly all such cases there is a neuropathic family history on one or both sides. A few cases can be traced to some of the causes of acquired mental deficiency previously mentioned. Inbreeding, such as occurs in the aristocracy, may be a contributory cause. The condition would naturally be aggravated by faulty education and mode of life, but without the hereditary and inherent mental defect it cannot be produced.

The *Treatment* is a question of careful training and education, and a good deal can be accomplished if no expense is spared. It is always best, I am sure, to remove these cases from home, for nearly always a state of friction arises between the girl or youth and one or other parent, which is detrimental to their progress. If the patient is liable to outbursts of passion, these must be gently curbed, and the system of education made attractive by utilising any particular taste which they have. The amount of improvement which such cases are capable of depends upon the possibility of fixing the attention, and this depends largely on their having some taste which can be so utilised, such, for instance, as music. The possibility of errors of refraction may be remembered, as in the cases quoted on p. 788.

§ **546. Mental Defects in Children under Ten** is a very different matter to the mental disorders of adults. Moral perversion or insanity, as already mentioned (§ 542), is not infrequent, but mania, melancholia, and other adult varieties are extremely rare. Mental deficiency as a whole is the characteristic of this age period, and this is what will concern us now. It may be CONGENITAL—*i.e.*, due to ante-natal causes —or ACQUIRED (non-congenital)—*i.e.*, due to causes arising after birth.

The *Symptoms* and causes of these two groups differ considerably. In both varieties the children are " backward," deficient in all the faculties of the mind in greater or less degree ; they carry evidences of this in their manner and behaviour, and sometimes, but not always, in their face. CONGENITAL cases always present some well-marked *alteration in the facial or cranial and bodily conformation*, and

very often they are dwarfed in body as well as in mind. Children belonging to the NON-CONGENITAL class of mental deficiency do *not present these physical alterations ;* their expression is bright, their cranium and face natural, their limbs well made, and, excepting in the paralytic class, they can generally walk and run well. Unfortunately, they are also distinguished from the congenital class by a mobility and restlessness which is an indication of the difficulty of fixing their attention, and therefore of teaching them. They may also be distinguished from the congenital cases by a history of their having been mentally sound at birth and for some years afterwards, but among the lower orders a history of any diagnostic value is often wanting.

The practical point to ascertain in all cases of mental deficiency in childhood is their teachability, and this depends on two questions : (1) Can their attention be attracted and fixed ? and (2) Are they imitative ? (3) Their facial and bodily conformation should be observed ; (4) the history or evidences of paralysis noted ; and (5) if they have been to a Council School in England, the standard they have reached—the average in the infants' school is anything under five years ; in Standard I., seven to eight years, with an increment of one year for each succeeding standard, Standard VII. being reached by normal children at thirteen or fourteen.

The **congenital varieties of mental deficiency** in childhood (**idiocy**) date from birth, and the children generally present evidences in the facial and cranial conformation as well as in the mind. In a good many of these cases the palate is too high, or V-shaped, too narrow from side to side, or otherwise deformed. The palate described by Dr. Clouston, and called by him the "neurotic palate," is one midway between the normal palate and the V-shaped palate, and is found in persons of nervous temperament, who are liable to hysteria, neuralgia, and migraine. Dr. Fletcher Beach found that out of 700 feeble-minded children 28 per cent. had V-shaped or otherwise deformed palates, and 60 per cent. had neurotic palates. On the other hand, a high or V-shaped palate does not necessarily imply congenital deficiency, as was at first thought.

The VARIETIES OF IDIOCY (Congenital Mental Defect in Childhood), as given by Dr. Fletcher Beach,[1] are six in number.

1. *Simple Congenital Idiocy* includes children *without any obvious* abnormality of the cranium or limbs, only in the face or palate (*supra*). In some the facial expression may be fairly intelligent, but most of the lower grade present an animal expression, thick lips, pug-nose, large coarse ears, broad, thick, depressed bridge of nose, narrow or hairy forehead, and underhung jaw.

2. The *Mongol* or *Chinese* type of congenital deficiency is so called from the resemblance of the face to that of the Chinese, the palpebral fissures sloping downwards and inwards. With flat face, flat back to the head, and constant protrusions of the tongue, this form of idiocy presents an unmistakable physiognomy. The fingers also are stunted and the little fingers incurved. Congenital heart disease occurs in about 30 per cent. They may be regarded as "unfinished" children, as they are often born of mothers who have suffered from continued ill-health during pregnancy ; sometimes they are the youngest of a large family, or born of parents advanced in life. These children are imitative, and therefore educable to a limited extent, but they make no progress beyond a certain point.

3. *Microcephalic* idiocy includes children whose heads are smaller in circumference than normal, which averages about 19 inches. The head may measure 17, 15, or even 12 inches ; the forehead is narrow, and slopes backwards, corresponding with the deficiency of the frontal development of the brain. The features are frequently normal, eyes large, and nose aquiline. These children rarely make much improvement, for they have but little power of attention, though some of them are imitative. The question of craniectomy was raised some years ago in connection with these cases, as the small size of the brain was thought to be due to premature union of the cranial sutures, but this is now known to be incorrect.

4. *Scapho-Cephaly* is so called from the boat-shaped character of the head,

[1] *Clinical Journal*, August 4, 1897 ; and "The Treatment and Education of Mentally Feeble Children," Fletcher Beach, 1895, Churchill, London.

the antero-posterior being considerably longer than the transverse diameter. Mental defect is not *necessarily* associated with this cranial conformation. But too much dependence must not be placed on the shape of the head, unless it is combined with mental defect. The children are sometimes deficient in intellect, but teachable.

5. *Hydrocephalic* idiocy occurs in some of the children born with hydrocephalus. Some retain their intelligence for a considerable while, but usually they drift into imbecility.

6. In *Paralytic* cases the child has had hemiplegia, paraplegia, or diplegia, *dating from* birth. The paralysis may be due to some inflammatory condition of the membranes in utero, or pressure or injury before or at the time of birth (birth palsy, *q.v.*). Cerebral atrophy, congenital malformations, and other lesions have been found. Mentally these children generally make good progress under education, but physically little can be done for them.

7. In *Sporadic* and endemic *Cretinism* the markedly stunted growth is the most characteristic feature. The head is usually large, flat at the top, spread out at the sides (Fig. 6, p. 27). Hair coarse and dry, like that of a horse's tail. Voice hoarse and squeaky. Speech generally limited to a few words, and often monosyllabic. Their temperament is placid and good-natured. Under treatment by thyroid gland or extract these cases make very remarkable progress, as in the two illustrations given. The treatment must be continued during the patient's lifetime, otherwise they relapse into their old condition.

Causes of Congenital Mental Deficiency in children.—*α*. Causes acting *in the parents before birth* (heredity) occupy a very prominent place. Beach and Shuttleworth have exhaustively investigated this question in 2,400 cases. Abnormal conditions in the mother during pregnancy account for as much as 29 per cent., among which may be mentioned injuries, worry, anxiety, fright, or illness. Phthisis in the parents accounts for 28 per cent., insanity and imbecility 21 per cent., epilepsy and other neuroses 20 per cent., and intemperance in the parents 16 per cent. Consanguinity acted as a cause in only 4 per cent., even when consanguinity in the grand-parents was taken into account. This is somewhat at variance with the generally supposed evil of marriage between blood relations, but it is a fact about which there seems no doubt, and Dr. Beach adds: "Even in those cases where consanguinity was present there were other hereditary tendencies, of themselves quite sufficient to produce the affection (congenital mental deficiency) without the presence of consanguinity." Syphilis in the parents was responsible for only 1·7, the effect of syphilis in the parents apparently being not so much the procreation of idiots as of children apparently healthy at birth whose nervous systems break down at the age of puberty.

β. Among the causes acting *at the time of birth* prolonged or tedious parturition is undoubtedly the most potent. It is sometimes supposed that injury by forceps is a potent cause of the production of congenital mental deficiency, but it is worthy of note that in only 3 per cent. of Beach and Shuttleworth's cases was there a history of delivery by forceps. His facts go to show that it is far better to put on the forceps early than to allow labour to be prolonged indefinitely. Such prolongation leads to great compression of the cranium, the brain being crushed, distorted, and otherwise injured. Such children when born are very often in an absolutely helpless condition. Others are subject to convulsions, and when death ensues, meningeal hæmorrhage and cortical laceration are very often found.

Acquired (or **non-congenital**) **mental deficiency in children** under ten is rarely difficult to differentiate from the congenital deficiency (*vide supra*). The varieties depend upon the causes. Among the *Causes* infantile convulsions was in operation in 27 per cent. of the 2,400 cases which Dr. Beach collected, epilepsy and other cerebral affections in 8 per cent., head injury in 6 per cent., fright or shock in 3 per cent., febrile diseases such as scarlatina, measles, whooping cough, enteric, small-pox in 6 per cent., and over-pressure at school was only mentioned in 0·16 per cent.

1. *Infantile Convulsions*, if only occurring once or twice, may have no evil result, but if continued for some time throughout the early months of life, they generally result in a certain degree of weakmindedness. Many such cases occur in the children of insane or epileptic parents. They generally make little or no improvement, and drift into imbecility.

2. *Epilepsy*, as a rule, does not commence until after ten years of age, but in some rare cases where it starts quite early in life it is attended with marked mental deficiency, although the fits may be few in number. Some cases improve if the fits cease, but mostly they go from bad to worse. Infantile convulsions cease before two years, epilepsy never occurs till after two, and very rarely till after fourteen years.

3. In *Paralytic Cases* the mental deficiency is associated with hemiplegia, and very often epileptiform seizures (*q.v.*) which have started *after birth*. They are mostly due to cerebral hæmorrhage and the scar which results.

4. *Traumatic Causes.*—Injuries to the head in childhood frequently result in mental deficiency. The injury, as previously mentioned, may have occurred during childbirth (compare β, p. 787), though the symptoms may not have arisen until later. But quite as often the traumatism occurs after birth, and its history is a little difficult to make out. I frequently see mentally defective children in the out-patient room in whom careful inquiry reveals the history of a fall from a perambulator or cot, which had been disregarded by the mother. One should always suspect an injury (perhaps concealed by the nurse) when the mental symptoms are associated with spastic hemiplegia. But very often there is no sign of paralysis or scar on the head, and the fits or mental defect from which the patient suffers did not supervene till many months or a year or so after the injury, being, in fact, due to the cerebral scar which results.

5. *Inflammatory Cases.*—The acute specific fevers, meningitis, and other inflammatory conditions may be followed by mental deficiency. It seems to me probable that most of these cases are due to concurrent or consequent inflammation of the brain or its membranes. The mental symptoms do not necessarily follow directly after the illness which has caused the mental defect, as in the case about to be mentioned, and therefore we may have only the history to rely upon. These cases are among the worst which we have to deal with. They are analogous to cases of mania occurring in the adult, excepting in their universally unfavourable course. In October, 1897, a little girl, æt. three and a half years, was brought to me by her mother. She ran perpetually around the room from object to object, resting her attention on nothing for more than a second or two at a time, taking no notice of anybody. The mother said she was excitable at night and often mischievous. The birth had been natural, and the child normal at birth and up to the age of two years. Then she was laid up with a feverish attack, which was called " influenza," attended by very severe headache, with which she was ill for several weeks. She recovered completely, and it was not until the age of three that she began to be unmanageable and destructive, and the present condition slowly supervened.

6. *Hypertrophy of the Brain* is included by Dr. Beach under the heading of inflammatory cases, because the post-mortem appearances of patients who died of this disease under his care showed that there was, or had been, chronic inflammation of the brain. In these cases both the brain and the skull are enlarged, the shape of the head being more of a square than in hydrocephalus.

7. *Hereditary Syphilis.*—Among the symptoms of this protean disorder is mental deficiency in childhood. It does not seem to affect the brain in infancy or early childhood, but about the period of the second dentition, or between that and the time of puberty, the nervous system sometimes shows signs of progressive degeneration. Sometimes this is manifested by disseminated sclerosis or other spinal disease, sometimes only by a steady deterioration of the mind.

8. *Reflex Causes.*—Mental symptoms of a pronounced kind arise in association with asthenopia in children. Two little girls, aged six and thirteen years, were brought to me for great despondency, and crying without cause. The mother of

each stated the child was constantly crying without a reason, and was constantly saying that her parents and others were against her and hard on her. No heredity was in operation. The vision was found by my colleague, Mr. Work Dodd, to be six-twelfths and eight-twelfths of normal, and on this being adequately corrected by glasses the mental symptoms entirely disappeared.

9. Theoretically, it is possible for some of the forms of mental alienation met with in adult life to appear in childhood, but mental disease practically only arises in childhood when one of the above causes is in operation.

The *Prognosis* of ACQUIRED MENTAL DEFICIENCY in childhood is always very grave. Indeed, there are few cases where much improvement takes place when once the deficiency is established. If the mental symptoms seem to have come to a standstill, and the child is in any degree educable, something may be done in this direction ; but, as a rule, less can be done for acquired cases than for congenital, and their tendency is to get worse.

In the TREATMENT OF MENTAL DEFICIENCY IN CHILDHOOD, whether CONGENITAL OR ACQUIRED, a certain amount can be done by education and discipline. The first and fundamental principle is to remove the child from its home and from the surrounding conditions which have been unconsciously adapted to its defective habits. The second point is to fix the child's attention, and this can best be done by employing music, pictures, and other things which please and attract the child. It may be taken as a general principle that the educability of a child—that is to say, the prospect of recovery—depends entirely upon the facility with which the attention can be attracted and the efficiency with which it can be retained when once secured. The shape of the head and aspect of the face cannot be relied upon ; the brightest looking children are often the most volatile. On the other hand, the dull-looking children who take an interest in their work will make more progress. As a rule, the prognosis is much worse in irritable, restless cases than in quiet cases. It is usual to commence with the education of the senses—touch first, then sight, hearing, taste, and smell. Speech also has to be educated by means of speech drill. Any vicious habits must be gently corrected, and punctuality and discipline enforced. Later on, perseverance, and ideas of justice, duty, self-reliance, prudence, and forethought have to be inculcated. The progress of any case under treatment depends very much on the age at which it is commenced. It should always be begun as early as possible, and the idea which some mothers have of the symptoms passing off at seven or fourteen by an abrupt change from mental enfeeblement to mental brightness should be met and combated as wholly fallacious, and very damaging to the prospects of success.

GROUP III. INTRACRANIAL INFLAMMATION.

§ **547.** Pyrexial disorders of the nervous system are not numerous, and are practically confined—if acute anterior poliomyelitis and certain exceptional cases of cerebral abscess and cerebral hæmorrhage be omitted—to acute inflammation of the meninges. They consist of the following intracranial inflammatory conditions, all of which present *cerebral symptoms with pyrexia* and its attendant symptoms. The onset in most is acute, though in some it is insidious.

I. Tuberculous meningitis.
II. Acute meningitis.
III. Post-basic meningitis.
IV. Epidemic cerebro-spinal meningitis (§ 373).
V. Intracranial abscess.
VI. Sinus thrombosis (especially septic thrombosis).
VII. Hæmorrhage, chronic degenerative diseases, etc.

Clinical Investigation.—1. The *cerebral symptoms* in intracranial inflammation may be grouped into those of irritation and compression. The symptoms of cortical *irritation* which are the most usual, especially in the early stages, consist of headache, vomiting, tonic or clonic spasms or convulsions, sleeplessness, restlessness, delirium, quick pulse, and contracted pupils. The symptoms of *compression*, which usually ensue later, are mental dulness, paralysis of the limbs and cranial nerves, a slow full pulse, dilated or unequal pupils, and stupor passing on to coma. The earlier or irritative stages may have to be diagnosed from other causes of convulsions (§ 597); the later compression stage from the typhoid state (§ 346) or coma (§ 530).

2. The *scalp and cranial bones* should be examined for any swelling or tenderness on percussion—*e.g.*, any œdema (sinus thrombosis), erysipelas or Pott's puffy tumour (an œdematous swelling of the scalp which is usually a sign of subcranial extra dural abscess in that situation). When there has been a *wound of the scalp*, the skin may have healed up externally though pus has formed beneath, and the infective products carried along the perivascular sheaths or lymphatics into the skull. But the patient may go about for days or weeks before symptoms of intracranial inflammation present themselves.

3. The *ear* and *sinuses* near it, the nose, and the pharynx should also be carefully examined for any discharge or signs of disease.

4. The *cranial nerves* need to be examined severally (§ 608 *et seq.*). Inflammation, like tuberculous meningitis, which has a predilection for the base, nearly always reveals cranial paralysis at some time; but those which affect the convexity of the brain, such as simple meningitis, give rise rather to convulsions.

5. The *lungs* and other organs should be examined. Many cases of tuberculous meningitis are secondary to tuberculosis of the lungs or peritoneum.

6. The *age* and *history* of the patient, especially as regards any previous otorrhœa, may be noted. Acute meningitis may occur at any age, and runs a course of about two days to two weeks; tuberculous meningitis chiefly affects children, and runs a course of two weeks to two months (including premonitory stage); post-basic meningitis is almost confined to infants under one year, and runs a prolonged and indefinite course.

7. The *temperature* should be carefully investigated, and a chart obtained. Diurnal intermissions suggest tuberculous meningitis (§ 548). A nondescript or continued temperature suggests acute meningitis (§ 549). The temperature falling after a few days of initial rise and becoming subnormal suggests cerebral abscess (§ 550). Wide and irregular intermissions suggest septic sinus thrombosis (§ 551).

8. Lumbar puncture is a valuable aid both to diagnosis (pp. 921 and 923) and treatment (p. 930).

§ **548.** I. **Tuberculous Meningitis,** tuberculosis of the cerebral meninges, is the commonest cause of intracranial inflammation in children. The cerebral pia mater, especially at the base and in the Sylvian fissure, becomes studded with grey miliary tubercles. It is more frequent in children, and in the male sex. Tuberculous meningitis may (i.) supervene in a case of tuberculosis elsewhere, of which previous symptoms have existed; (ii.) it may be ushered in primarily and suddenly with vomiting and convulsions in a child previously in good health; or (iii.) it may come on insidiously as a primary affection of the meninges, with a long prodromal period.

The *Symptoms* are usually insidious and variable. There is a prodromal and three other stages, the latter running a course of two or three weeks, and passing imperceptibly into each other. In the *prodromal* stage, which may last a considerable time, the child gets thinner, loses its appetite, is peevish and listless but with intervals of bright-

ness, when the mother thinks it is becoming well again. Intermittent pyrexia may be revealed from time to time. (1) The *irritative* stage is ushered in very often suddenly, with (i.) severe headache, vomiting, or convulsions, and there may be delirium, and the child may lie curled up, shrinking from light or sound. (ii.) There is a moderate degree of fever, usually of a diurnally intermittent type. (iii.) The pulse is quick, and the pupils contracted. (iv.) The head generally shows a characteristic retraction with rigidity of the muscles of the neck. (v.) The abdomen is retracted. When vomiting is present in a child, without diarrhœa, tuberculous meningitis, or other head mischief should be suspected. (vi.) The peculiar cry, known as the "hydrocephalic cry," may accompany this stage of the disease, and last till the end. As a rule, the irritative stage lasts only a few days to a week. (2) The *compression* stage is announced by symptoms pointing to paralysis of the cranial nerves as in all diseases situated chiefly at the base of the skull. (i.) The pupils are unequal, and become dilated, and strabismus is common owing to paralysis of the sixth or third nerves. (ii.) The patient is drowsy, with slow pulse, which may be irregular, and sighing respiration. (iii.) There is vasomotor paralysis, indicated by the red streak when the finger-nail is drawn along the skin. This condition is known as the "tâche cérébrale" (Trousseau), from the idea, now known to be mistaken, that it was found only with cerebral lesions. (iv.) Optic neuritis or tubercle of the choroid may develop. (3) The *final* stage is marked by increasing irregularity and weakness of the pulse and the respiration. The drowsiness passes into coma, and there is incontinence of urine and fæces. Mucus gathers in the bronchial tubes, and breathing is laboured, and often takes the Cheyne-Stokes form. Just at the end the temperature may rapidly fall, or it may rise very high (106° or 107° F.) and local paralyses—*e.g.*, of the arm or leg, or ptosis may occur, with convulsions. This stage lasts only a few days.

Tuberculous meningitis in adults is often secondary to tubercle in the lungs, abdomen, or elsewhere. It differs from the disease in children in: (1) The advent is most insidious and prolonged. I once saw a typical example of this where headache and intermitting pyrexia were the only symptoms for nine weeks. (2) Severe and persistent headache with intermitting pyrexia are always very prominent features. Hemiplegia, or, rather, hemiparesis, may be the earliest and principal symptom, and aphasia is not infrequent because of the invasion of the Sylvian fissure by the tubercle; while (3) optic neuritis, strabismus, or other evidence of cranial nerve paralysis, such as facial paresis, or inability to swallow, are present as in children. Vomiting and convulsions are rare. (4) The course lasts from five to twelve or more weeks.

The *Diagnosis* of tuberculous meningitis may often be settled by lumbar puncture (pp. 921, 923, and 929). *Enteric fever*, with head symptoms, and no rash or diarrhœa, may for a week or longer be mistaken for meningitis; and, on the other hand, the meningitis which has little headache may be mistaken for enteric fever. But the course of the disease and the aid of the Widal reaction soon reveal its true nature.

Any one of the *specific fevers* may be ushered in with headache so severe as to give rise to a suspicion of meningitis; but in all such cases the headache ceases when delirium begins. Irregularity in the breathing and the pulse, and evidences of basal paralyses are conclusive signs of meningitis. *Simple meningitis* is distinguished from the tuberculous variety by its rapid course, which rarely exceeds a few days, by there being no basal paralyses, and by the presence, perhaps, of a local cause, such as injury or otitis media. A rapidly growing *tumour—e.g.*, tuberculous or gliomatous—may simulate meningitis. Here the character of the optic neuritis present aids diagnosis—if very intense, with swelling and hæmorrhages, it is probably due to tumour. The early stages of tuberculous meningitis in young women may resemble *hysteria*, but the eyes should be examined for papillitis. *Post-basic meningitis* occurs in infants under one year, and has a longer course. In *marasmic conditions* in children drowsiness and convulsions may arouse the suspicion of tuberculous disease being present, but in such cases the fontanelle is depressed because the intracranial pressure is not raised.

Prognosis and Treatment.—Tuberculous meningitis is essentially a chronic disorder, though its average duration varies considerably—from three weeks to three months are about the limits. The prognosis depends mainly on three things—first the period of the disease at which the patient comes under treatment; secondly, the height and range of the temperature, which is the measure of the activity of all tuberculous processes; and, thirdly, the extent of the tuberculous mischief in other organs. Until recent years it was regarded as necessarily fatal, though cases of undoubted recovery have been published (Trousseau, Rilliet, Carrington). Curiously enough, the *treatment* which was most successful consisted of calomel and pot. iod., remedies which are directed against syphilitic lesions, and it seems possible that some of the alleged cases of recovery may have been syphilitic meningitis. Lumbar puncture (p. 921), originally suggested by Quincke simply to relieve the intracranial pressure, has in several instances resulted in recovery (Henkel, Barth, and others). It probably acts by "flushing" the meninges and the introduction of fresh opsonins; 10 or 20 c.c. may be withdrawn and repeated. Ice-bags applied to the head, bromides or chloral internally, or evaporating lotions to the shaven scalp, relieve the headache. Mercurial and iodoform ointment rubbed into the scalp is recommended in chronic cases.

§ **549.** II. **Acute Meningitis** (Leptomeningitis; Arachnitis; Acute Hydrocephalus) is a diffuse inflammation of the pia mater and arachnoid (leptomeningitis), simple or purulent, according to its exciting cause, chiefly affecting the convexity of the brain.

Symptoms.—There are symptoms and signs of cortical *irritation* (§ 547), followed by signs of *compression—i.e.*, increased intracranial pressure. The inflammation affects the convexity rather than the base of the brain, the reverse of tuberculous meningitis, and the symptoms consist, therefore, of muscular twitchings, spasms, sometimes convulsions, and later on paralysis of the muscles of the body, rather

than paralysis of the cranial nerves as in tuberculous meningitis. There, is no prodromal stage, the invasion being sudden. The course of the disease lasts usually a few days to one or two weeks. (1) The temperature runs a pyæmic course, and there may be rigors and wide variations. Other symptoms are (2) severe persistent headache ; (3) delirium ; (4) retraction of the head, rigidity of the muscles of the neck, diffuse hyperæsthesia and pain, especially about the neck, from involvement of the spinal nerve roots. Optic neuritis and ocular paresis are rare. (5) There may also be vomiting, flushing of the face and conjunctivæ, the " tâche cérébrale " of Trousseau, and herpes on the lips or face. Kernig's sign (§ 514, *c*) is present if the inflammation extends to the spine.) (6) There is usually a history or evidence of some cause, since the disease is rarely primary, the most common cause being disease of the petrous bone, with or without ear disease, attended by a purulent discharge.

Etiology.—The most frequent cause of purulent meningitis is (1) extension of inflammation from adjacent parts, either from *without—e.g.*, in caries or necrosis of the cranial bones, erysipelas of the face or scalp—or from *within—e.g.*, a cerebral abscess or sinus thrombosis. Any of the bones may be involved, but disease of the petrous bone is the most frequent cause, with or without otitis media ; nasal or ethmoid disease is a much less common cause. In disease of the cranial bones a chronic localised thickening of the meninges may prevent a localised collection of pus, a subcranial abscess, from spreading laterally ; but the edges are liable at any time to become softened, and so permit of a generalised purulent meningitis. (2) Punctured wounds of the scalp may cause meningitis in the same way as in cerebral abscess formation (*q.v.*). (3) Pyæmia and malignant endocarditis may be attended by a purulent, and rapidly fatal form of the disease. (4) The pneumococcus is probably one of the commonest causes of suppurative meningitis. A *primary* meningitis may be due to pneumococci found in the cerebro-spinal fluid, without pneumonia being present. (5) *Secondary* to other constitutional disorders such as the specific fevers, influenza, diphtheria, acute rheumatism, measles, small-pox, erysipelas, scarlet fever, anthrax, gonorrhœa, and actinomycosis, also, it is said, to gout, chronic nephritis, and heart disease in their terminal stages. (6) A primary cerebro-spinal meningitis also occurs in the sporadic and epidemic form, due to the diplococcus intracellularis (§ 627). (7) *Tuberculous* and *post-basic* meningitis are special forms described respectively in § 548 and below.

Diagnosis.—Acute meningitis has to be diagnosed from other forms of meningitis and from *acute specific fevers*, pneumonia, and other acute diseases, which may simulate meningitis at their onset with headache, vomiting, and absence of local signs. *Pneumonia* may be very deceptive at first before lung signs are definite, but the rapidity of respiration affords us a clue. *Tuberculous* meningitis has a more insidious onset, a prodromal stage and a prolonged course. When there is otitis media, meningitis may require to be differentiated from *cerebral abscess*, which has more localised symptoms, a low temperature after the first day or so, and the headache lasts longer before the onset of coma. In septic *sinus thrombosis* the temperature is marked with recurring rigors, and local signs of the sinus involved are usually seen, such as the brawny swelling in the neck in lateral sinus thrombosis. The diagnosis of the *cause* also requires investigation, especially in children. When due to otitis, the patient usually holds the hand to the head. Lumbar puncture (§ 626) and bacterial cultures may aid us in the diagnosis of the cause.

Prognosis.—The disease usually runs a fatal course in a few days, but it may completely recover, or if traumatic in origin, it may pass into a chronic localised meningitis. Recovery has been reported after the primary pneumococcus form, though this and the other septic forms are generally fatal.

Treatment.—The patient must be kept in a dark room, perfectly quiet, the head shaved, and an ice-bag applied. A purge should be given, preferably of calomel. Some recommend blisters, or the cautery to the back of the neck. Only milk diet is allowed. Mercury, especially in the form of inunction, is recommended, to-

gether with large doses of iodide of potassium. The cause must be carefully investigated, and where local causes are in operation, such as injury or disease of the scalp, these must be treated surgically. In children and infants especially, puncture of the tympanum, with consequent outlet of a little pus, has often resulted in the prompt relief of symptoms of intracranial irritation. Lumbar puncture (§ 626) not only affords a clue to diagnosis of the primary malady, but may, by the relief of tension, result in recovery.

III. **Posterior Basic Infantile Meningitis** may be **acute,** but is usually more **chronic.** It is a disease occurring in infants under twelve months, due to inflammation in the posterior fossa of the skull, associated with a special diplococcus, the diplococcus of Weichselbaum. This is the same microbe as that of epidemic cerebro-spinal meningitis (§ 373), and post-basic meningitis is now (1909) generally regarded as a sporadic, sub-acute form of that disease. The exudation glues together the cerebellum and medulla, leading ultimately to the blocking of the foramen of Majendie and distension of the ventricles (hydrocephalus). The characteristic *Symptoms* are (1) the gradual onset of the retraction of the head, which may amount to opisthotonos of the spine with flexor and extensor spasm of the limbs; (2) staring of the eyes, with blindness, appears quite early in the disease, unassociated with optic neuritis, for it is due to involvement of the occipital cortex; (3) vomiting; (4) rigidity of the limbs, which may be general or localised to one extremity; (5) paroxysms of high fever lasting a day or two. The disease occurs in infants from three to twelve months old, only occasionally in older children. It is diagnosed from tuberculous meningitis by the age of the patient, the greater degree of cervical opisthotonos, the longer course of the disease, the absence of optic neuritis in post-basic meningitis; and by lumbar puncture (p. 923).

Prognosis.—The disease often runs a very prolonged course of weeks or months. Death has taken place after nineteen months. Cases may undoubtedly recover; Drs. Lees and Barlow say one in six recover completely. In others grave sequelæ remain: hydrocephalus resulting from the distension of the ventricles; blindness (without optic neuritis); deafness (with consequent loss of speech); and defective intelligence; or a combination of these.

Treatment.—Mercurial ointment should be rubbed into the neck, and iodide of potassium in doses of 1 to 3 grains every two hours is believed to cause absorption of the exudation. Lumbar puncture affords both a clue to *diagnosis* (p. 925) and a means of *treatment* (p. 930).

IV. **Epidemic Cerebro-Spinal Meningitis** has already been described in § 373.

§ 550. V. **Intracranial Abscess.**—Abscess of the brain may occur in an acute or chronic form.

The symptoms of cold or *chronic abscess* are identical with those of any other cerebral tumour, apart from the history and the symptoms referable to the condition which caused it. In quite a number of cases the history of a cause is wanting, and such cases present only the features of intracranial tumour (*q.v.*). The commonest cause of cerebral abscess undoubtedly is suppurative middle ear or bone disease.

The *Symptoms* of acute abscess of the brain may be divided into three stages. The *initial* or inflammatory stage lasts from twelve hours to three days; in some cases it has lasted a week. Very often this stage may not be noticed; and in most instances the patient does not come under observation until it is over. The three chief symptoms which characterise this stage are *pain, vomiting,* and *rigors.* (i.) The pain in the head may be burning, shooting, and continuous or intermittent, and there is tenderness over the seat of the abscess. (ii.) The vomiting occurs without nausea, and has no relation to food. (iii.) The rigors vary greatly in severity, from a slight shivering to a shuddering. If the rigors are frequent, there is probably present some systemic infection or sinus thrombosis (*q.v.*). The temperature in this stage is above normal, but not high. If the disease be due to otitis media, the discharge stops. The *second or collapse* stage[1] shows symptoms

[1] Some call this the *latent* stage, but Sir William Macewen objects to the use of such a term in reference to a condition where symptoms are present.

similar to the first stage, only much less marked. (i.) The pain is diminished ; the patient may lie quietly moaning with the hand over the affected part. Tenderness is brought out on percussion, and is useful in localising the site of the mischief. (ii.) Vomiting and vertigo occur on movement only. (iii.) Cerebration is slow ; the patient will answer questions correctly, but only after long delay, and he may fall asleep in the middle of a sentence. (iv.) The temperature in this stage is normal or subnormal, if the abscess is uncomplicated. (v.) The pulse is slow and full (30 to 60). Respiration is slowed. (vi.) Optic neuritis develops ; and (vii.) paralyses may occur, which aid in diagnosing the site of the lesion. This stage lasts from one to five weeks. In the *third* or paralytic stage the abscess terminates in one of three ways : (i.) By compression symptoms—deepening stupor, coma, and death. (ii.) The abscess may open on the surface of the brain, leading to meningeal symptoms with high temperature, quick pulse, vomiting, convulsions, etc. (iii.) The abscess may open into the ventricles of the brain, which is characterised by lividity, dilatation of the pupils, stertorous breathing, high temperature, convulsions, coma, and death within twelve hours.

Diagnosis.—Abscess of the brain has to be diagnosed from *acute meningitis*, in which the symptoms are more of an irritative character, with high temperature and quickened pulse. In *sinus thrombosis* there are recurring rigors and local signs of the involvement of the sinus. It must be remembered that both meningitis and thrombosis may accompany abscess, and in this case the symptoms are confusing. If meningitis and abscess occur together, the presence of the latter will be indicated by a slower pulse than would be met with in uncomplicated meningitis. *Intracranial tumours* resemble abscess. The slow progress of the symptoms, the greater amount of optic neuritis, and more definite focal phenomena, with the absence of rigors, and of any source of abscess formation, are in favour of a diagnosis of tumour ; while the presence of a subnormal temperature is in favour of abscess.

Causes of cerebral abscess.—Many valuable observations have been made in this domain within the last few years by Sir William Macewen. Briefly, the causes of cerebral abscess may be said to belong to three categories—disease or injury of the cranium or scalp ; pyæmic infection from the scalp or other parts ; and softening of a tuberculous mass (very rare). The locality of the abscess depends a good deal upon its source. The chief causes are : 1. *Middle ear disease* is by far the most common cause. The abscess occurs in the temporo-sphenoidal lobe, or less commonly in the cerebellum. There is usually necrosis or tuberculous disease (caries), or occasionally syphilitic disease of the petrous bone. (i.) The inflammatory process spreads through the meninges, over the tegmen tympani (roof of the tympanum), and if the local adhesions be present to prevent lateral extension, the process goes on, even without any erosion of the bone, to ulceration of the brain, and consequent pus formation within the brain substance. (ii.) Or cerebral abscess may form in the white matter, the grey matter of the cortex of the brain remaining uninvolved. Here the inflammation spreads along the perivascular sheaths of the vessels or along the veins entering the brain. Briefly, the foregoing means that abscess may occur (*a*) by contiguity or (*b*) by extension along the lymphatic sheath of the vessels, or along the veins from a distance. 2. *Disease of the antrum of Highmore or nasal bones* causes abscess in the frontal lobe. 3. *Injury, such as compound fracture*, with necrosis of the bone, may cause an abscess, the position of which depends upon the seat of the injury. 4. *Wounds of the scalp or abscess*, where the skin is healed, but there is pus formation beneath the bone. 5. *Carbuncle of the face* (rare) or *scalp* causes abscess when septic matter passes by the facial vein and pterygoid plexus, or by the ophthalmic vein, to the cavernous sinus. 6. *Disease of the orbit* causes abscess by a similar method of extension. 7. *Erysipelas* (rare) more often causes leptomeningitis. 8. *Abscess of pyæmic origin* may occur in any part, but the most common situation is the occipital lobe. The pyæmic sources are septic embolism—*e.g.*, in malignant endocarditis—and pyæmia.

Prognosis and Treatment.—Early diagnosis and correct localisation of this serious condition is most important, for surgical treatment is successful, but untreated

cases usually die. In some cases the abscess undergoes apparent cure, and, if small, it may remain encapsuled for years, or may even dry up. Macewen mentions a few cases where the abscess discharged externally through eroded bone. The usual course of an acute uncomplicated abscess is from two to six weeks. A chronic abscess may last months or years, but is always a source of danger. After remaining without symptoms for years, it may suddenly burst into the meninges or into the ventricles. Uncomplicated abscess, if discovered in good time, is readily amenable to *treatment* in the hands of a skilled surgeon, but if symptoms of meningitis or sinus thrombosis accompany it, the prospect is much more grave, especially if the meningitis be diffuse.

§ **551.** VI. **Sinus Thrombosis,** thrombosis of the cerebral sinuses, may be PYOGENIC, and accompanied by pyrexia and stupor, or NON-PYOGENIC, accompanied by stupor only.

a. In **Pyogenic Sinus Thrombosis** there is pyrexia of a pyæmic type—*i.e.*, wide variations with rigors. Any one of the sinuses may be involved, but the lateral sinus (secondary to suppurative middle ear or temporal bone disease) is the favourite position.

The *Symptoms* common to septic thrombosis of any of the sinuses are (i.) severe headache, vomiting, and high fever of a pyogenic type, accompanied by rigors and sweats (see chart, § 383) ; (ii.) optic neuritis supervening in a day or two, and perhaps photophobia ; (iii.) drowsiness deepening into coma, and if prompt operative measures be not adopted, death ensues.

The *Localising Signs* of the particular sinus thrombosed depend on the extension of the inflammation along the corresponding extracranial veins, and on the presence of the cause. Thus, in *lateral sinus* thrombosis there are pain and tenderness in the mastoid region, together with the other signs of a suppurative otitis media or disease of the petrous bone ; the inflammation spreads down the course of the jugular vein on the same side and backwards behind the mastoid ; and consequently there is generally some hard brawny swelling in these positions ; if there has previously been a discharge from the ear, it mostly ceases. When the *longitudinal sinus* is thrombosed, the localising signs consist of œdema of the scalp, distension of the veins over the forehead, and sometimes strabismus, associated with convulsions at the onset. This is the sinus which is most often affected by non-pyogenic thrombosis, and when pyogenic in origin, the cause is usually some septic lesion of the face or scalp. When the *cavernous sinus* is affected, the localising signs are œdema of the eyelids and root of the nose, sometimes also of the pharynx, exophthalmos, and paralysis of the second, third, fourth, ophthalmic division of the fifth, and sixth nerves. Pyogenic thrombosis of this sinus may arise from some septic lesion of the orbit, nose, pharynx, or face.

The *Diagnosis* of pyogenic sinus thrombosis from *septicæmia* is very difficult, unless the local signs are pronounced. *Acute meningitis* is also differentiated with difficulty, but here we get a lower temperature without rigors and sweats, and retraction of the head and neck. In *cerebral abscess* the temperature is normal or subnormal after the initial rise, and it is accompanied not so much by stupor as by headache and paralysis of the cranial nerves.

The *Prognosis* of pyogenic sinus thrombosis is exceedingly grave, unless it is promptly dealt with by surgical measures, such as trephining over the mastoid cells, tying the jugular vein below, cutting down upon the lateral sinus, and turning out the clot. With these measures cases may recover, not without.

b. **Non-Pyogenic Sinus Thrombosis** gives rise to coma (§ 530) mainly in marasmic conditions. It is met with among infants suffering from prolonged diarrhœa, among the aged, and less frequently in adults in the last stages of exhausting diseases, such as phthisis or cancer. The superior longitudinal is the sinus most often affected. Headache, passing on to drowsiness and coma, are the leading symptoms ; the temperature may be slightly raised, but it never forms a prominent feature. In adults delirium may mark the onset, but the condition is difficult to diagnose with certainty. In infants the coma supervenes almost imperceptibly upon the drowsiness which accompanies their exhausted condition. In children,

epistaxis and convulsions should make one suspect the condition, even in the absence of localising signs. The localising signs are mentioned under Septic Sinus Thrombosis.

The *Prognosis* is grave, especially in adults. Children may recover, but only with impairment of intellect.

Treatment.—The patient should be kept at rest, with the head and shoulders slightly raised. The neck must not be bent, so that the blood be not hindered in its return. Tonics and stimulants may be given.

VII. Pyrexia, usually of short duration, may accompany certain **intracranial lesions of acute onset,** such as (i.) hæmorrhage, especially into the pons, when there is sudden coma, contracted pupils, and hyperpyrexia (see Apoplexy, § 531); (ii.) lesions occurring in the course of chronic degenerative disease of the central nervous system, such as disseminated sclerosis and G. P. I.

GROUP IV. MOTOR DISORDERS OF THE NERVOUS SYSTEM.

Motor disorders of the nervous system may consist of Paralysis (*below*), Inco-ordination (§ 577), Increased Muscular Action (§ 582), or Muscular Atrophy (§ 600).

a. Paralysis.

Paralysis may take the form of—

The patient complains of weakness or **paralysis** *of the limbs on* one side of the body. The case is one of Hemiplegia, which may be either of sudden onset or of gradual onset.

§ **552. Hemiplegia,** paralysis of one side of the body, is due to a lesion of the internal capsule or some other part of the motor tract of one hemisphere, somewhere above the decussation in the medulla.

The Clinical Investigation of a case of hemiplegia comprises an examination of the paralysed limbs, the cranial nerves, the cardio-vascular system, the urine, and the viscera, and a study of the history and mode of onset. Our chief object is to ascertain the *position* and *nature* of the lesion.

Symptoms.—In nine cases of hemiplegia out of ten the loss of power in the limbs is on the same side as that in the face; when it is on opposite sides it is known as "crossed hemiplegia," and the lesion is situated in the pons. The arm is usually more affected than the leg, and the leg than the face. Only the lower part of the face is affected, the food collects round the teeth on that side, but the patient can close both eyes. If the tongue is paralysed, during protrusion it is turned to the affected side, due to the unbalanced action of the healthy genio-hyoid and genio-hyoglossus. Hemianæsthesia on the paralysed side is not present unless the hinder limb of the internal capsule is also

involved. There may be an initial or *early* rigidity, coming on at the outset and lasting a few hours (as in cases of hæmorrhage), or coming in a few days' time and lasting for a few weeks (as in cases of irritative lesion). There is also a *late* rigidity, which comes on in all cases of hemiplegia after a few weeks or months, and lasts permanently, and is due to descending lateral sclerosis.[1] In the course of years the muscles may undergo a certain amount of *organic contracture*, due to intrinsic changes in the muscular substance, and the hemiplegic muscles are also liable to various kinds of tremor, mobile spasm and athetosis. These are especially common after the hemiplegia of childhood. The other features are those of an upper neuron paralysis—viz., (1) the deep reflexes are increased ; (2) there are no electrical changes ; and (3) there is no wasting beyond that of disuse. Finally, there are *vaso-motor alterations* in hemiplegia. There are known to be centres in the cortex, irritability of which produces coldness and paleness of the limbs. If the lesion producing the hemiplegia causes irritation of these centres, the hemiplegic limbs are paler, colder, or bluer. If, on the other hand, it gives rise to suppression of function, then the paralysed limbs are congested, sweating, and sometimes œdematous. These considerations explain the apparently contradictory observations made as to general dropsy, which sometimes avoids and is sometimes present in excess in the paralysed limbs, and the observation that the pulse is sometimes smaller, sometimes larger in the paralysed arm, and, finally, of the fact that the pupil is generally contracted on the paralysed side, either from paralysis of the sympathetic or from irritation of the third cranial nerve. In all organic diseases of the brain which produce hemiplegia there is generally more or less mental disturbance, and this is particularly marked in cortical lesions, such as tumours of the cortex, hæmorrhages on to the cortex, and various meningeal affections, particularly when the front of the brain is involved. Aphasia (§ 534) is associated with right hemiplegia in many cases owing to the involvement of Broca's convolution ; in left-handed persons left hemiplegia may be so associated.

The chief *Causes of hemiplegia* (ten in number) may be conveniently divided into those of *sudden onset—i.e.*, in the course of a few minutes or a few hours—and those of more *gradual onset—i.e.*, in the course of days, weeks, or months.

A *sudden onset* always indicates a vascular lesion—

Injury.
Hæmorrhage.
Embolism.
Thrombosis.
In hysterical hemiplegia also the onset is generally sudden.

[1] Rare cases have been recorded in which the paralysis remained flaccid (hémiplegie flasque of Bouchard).

A *gradual onset* may be due to—

Intracranial tumour.
Abscess.
Chronic cerebral meningitis and pachymeningitis.
Altered blood states (chorea, diphtheria, anæmia, pregnancy, etc.).
Chronic degenerations of the nervous system.

The *age* of the patient is an important aid in the diagnosis of the nature of the lesion in hemiplegia.

In the *first* half of life suspect in order: Embolism; thrombosis from syphilitic endarteritis; hysteria (in females); tumour; abscess; hæmorrhage in childhood (occasionally); post-febrile and other blood conditions.

In the *second* half of life in order of frequency we get: Hæmorrhage; thrombosis from arterial degeneration; tumour; aneurysm; chronic degenerations of the nervous system.

Syphilis plays a very important part in the causation of hemiplegia, which may be produced in several ways: (1) A syphilitic endarteritis and thrombosis; (2) the formation of a gummatous tumour; (3) meningeal affections; or (4) aneurysm.

(*a*) The **Sudden Causes** (vascular lesions) are much more frequent than those of gradual onset.

(1) Injury to the Head may produce hemiplegia by depressed bone, extravasated blood, or, later on, by the formation of an abscess or a cicatrix. It is known by (i.) the history of injury; (ii.) by being preceded or accompanied by loss of consciousness, stupor, or convulsions; and (iii.) early rigidity in the affected limbs.

(2) Hæmorrhage (apoplexy, § 531) from rupture of a vessel or an aneurysmal dilatation occurs most frequently in the lenticulo-striate artery (Fig. 156, p. 800) in the internal capsule, though it may occur anywhere. Its differential characters are as follows: (i.) The onset is usually very sudden, with coma, and often convulsions, the degree of these symptoms depending on the locality and the extent of the hæmorrhage; occasionally, when the hæmorrhage is small, headache, giddiness, or vomiting alone mark the advent. (ii.) The patient is usually of middle or advanced life, and often presents a history of chronic interstitial nephritis, though cerebral hæmorrhage is also known to occur in children. (iii.) The rigidity of the affected limbs comes on with the hemiplegia, and is succeeded by late rigidity and sometimes by tremor. Meningeal hæmorrhage rarely occurs unless the meninges are previously unhealthy, as in general paralysis.

(3) Embolism (§ 531), blocking of a cerebral artery, often of the middle cerebral artery, may arise from a fragment of a vegetation from a cardiac valve, or by some septic or other embolus. It causes a localised cerebral ischæmia and softening. It is differentiated by (i.) sudden hemiplegia, accompanied and often preceded by severe headache and giddiness; but (ii.) rarely by convulsions, and as a rule

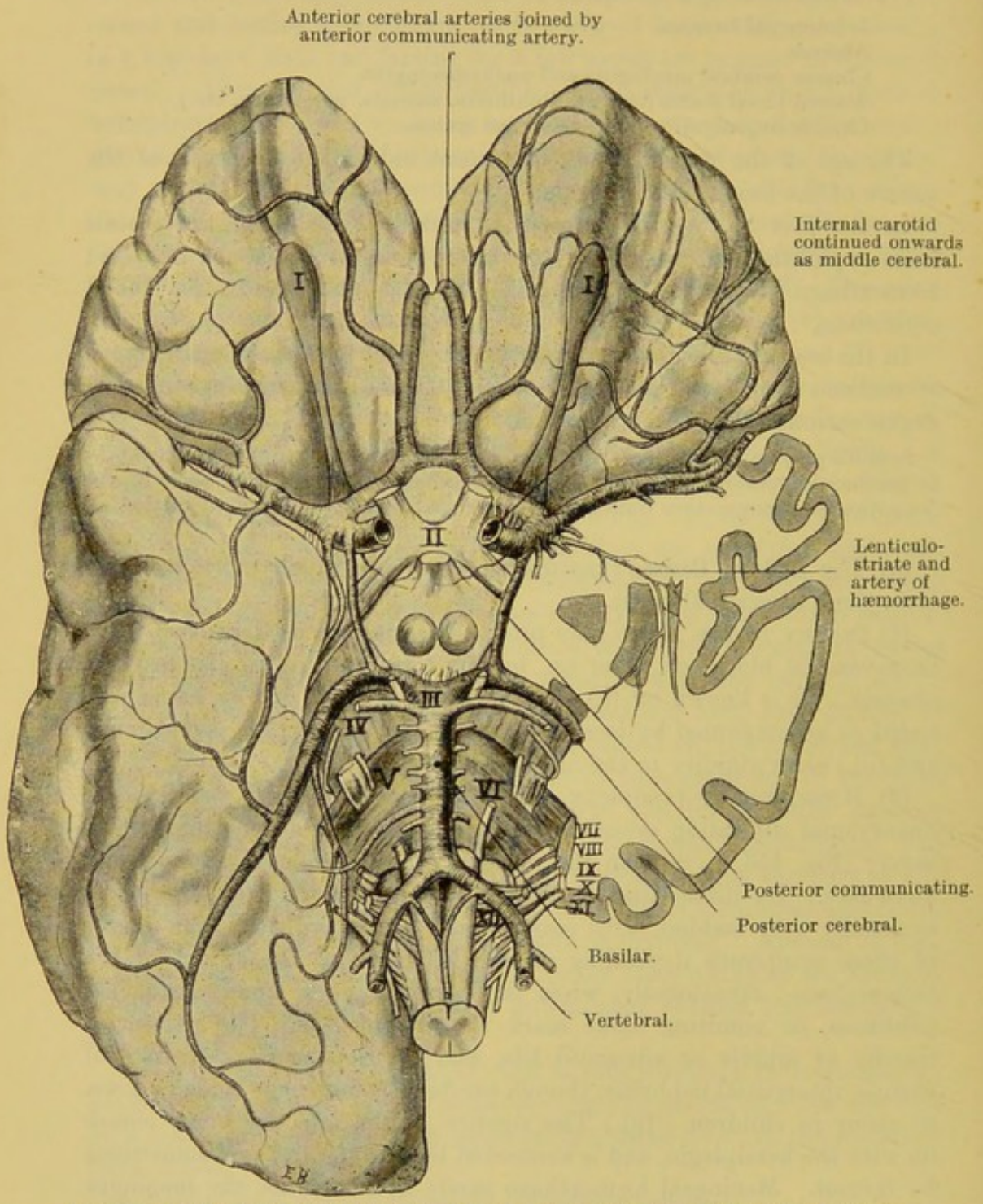

Fig. 156.—The BASE OF THE BRAIN, showing the arterial distribution and the cranial nerves.—In the oblique section of the left hemisphere are seen from without inwards—grey matter of the island of Reil ; claustrum (grey) ; external capsule (white) ; lenticular nucleus (grey); internal capsule (white) with artery of hæmorrhage ; and caudate nucleus (grey). I., Olfactory lobe ; II., optic chiasma ; III., bifurcation of vertebral artery between the third nerves ; IV. (on right crus cerebri), beside fourth nerve ; V. (on pons varolii), beside fifth nerve ; VI., sixth nerve (abducens) ; VII., facial nerve ; VIII., auditory nerve ; IX., glossopharyngeal nerve ; X., vagus or pneumogastric ; XI., spinal accessory ; XII. hypoglossal nerve.

consciousness is not lost ; (iii.) the patient is often young ; (iv.) there is often a history of rheumatic fever, and cardiac valvular disease, especially mitral stenosis ; (v.) the muscles are generally flaccid at first, and the hemiplegia is more often on the right side, because of the continuity of the left middle cerebral artery with the aorta.

(4) Arterial Thrombosis (§ 531) consists of coagulation of the blood within an artery owing to its occlusion by disease of the wall, or a thrombotic condition of the blood, as in phthisis. Senile change in the wall of the artery, associated with cardiac enfeeblement, is its commonest cause. In young subjects syphilitic endarteritis is very often in operation. Venous thrombosis is not necessarily associated with hemiplegia ; it is referred to under Coma. Thrombosis of an intracranial artery is recognised by (i.) moderately sudden advent of hemiplegia, but without unconsciousness or convulsions ; (ii.) the paralysed muscles are flaccid at first, and there is no early rigidity ; (iii.) the patient is mostly advanced in years, unless the arterial disease be syphilitic.

Arterial Occlusion is the commonest of the cerebral syphilitic lesions, and in that way becomes the commonest cause of hemiplegia under forty. A syphilitic endarteritis gradually occludes an artery, and results in thrombosis and " softening " of a circumscribed area, just as does atheroma of the cerebral vessels. The symptoms of a syphilitic softening resemble atheromatous thrombosis except in two particulars—namely, (1) the extent of the softening, and therefore of the paralysis is often more limited (*e.g.*, one arm or one leg) ; and (2) the patient is generally young or middle-aged. A thrombotic hemiplegia in a person under forty or forty-five is almost certainly syphilitic. This kind of cerebral syphilis is distinguished from the meningeal lesions or gumma by the absence of irritative signs. The advent, contrary to what one would expect from the *gradual* occlusion of an artery, is always more or less sudden, unaccompanied by unconsciousness, convulsions, delirium, rigidity of limbs, optic neuritis, or paralysis of cranial nerves.

(5) Hysterical Hemiplegia is recognised by the following characters : (i.) The advent is usually sudden, and often dates from an emotional shock or hysterical seizure ; (ii.) the paralysis is usually flaccid, incomplete in degree, and the face is exempt ; (iii.) it is often accompanied by hemianæsthesia of the same side, sensitive and sensorial ; (iv.) the condition varies from day to day, and may disappear suddenly, unexpectedly, and completely ; (v.) the youth and sex of the patient are characteristic, and she presents other evidences of the hysterical diathesis (*q.v.*), and a previous history of nervous attacks.

(*b*) Hemiplegia of **Gradual Onset,** in the course of weeks or perhaps months, points to one of the following conditions : Intracranial tumour, abscess, chronic cerebral meningitis (and pachymeningitis), altered blood states, or chronic degenerations of the nervous system. Nevertheless tumour, aneurysm, and abscess may sometimes run a latent or prolonged course, and then suddenly develop hemiplegia.

§ **553.** (6) **Intracranial Tumour**, the sixth cause of hemiplegia, may exist for a long time without any symptoms, especially if the tumour be situated in the frontal region, but it is generally characterised sooner or later by a progressive and insidious hemiplegia. As regards the nature of the tumour, syphilitic gumma is one of the most frequent in adult life, and is especially apt to affect the meninges at the base or

over the convolutions. It is well to bear in mind that malignant tumours may occur primarily in the brain; that cancer is confined to advancing years, but sarcoma occurs quite early in life. A tuberculous nodule is only met with in the young, and a history or the existence of tubercle elsewhere point to the tumour being of this nature. An aneurysmal tumour is not common.

The *Symptoms* of intracranial tumour vary very considerably with its locality (which will be discussed in § 554), but most cases present the following *symptoms in common:* (i.) Headache, vertigo, and causeless vomiting from time to time, precede and accompany the illness, especially if the tumour be situated near the cortex. (ii.) Jacksonian epilepsy (§ 598) is also frequent if the tumour be situated in or near the cortex. (iii.) Œdema of the optic discs, leading to atrophy, is present in a considerable proportion of the cases (five-sixths, Sir William Gowers[1]). For the occurrence of these the tumour need not be near the optic nerve; it is believed to be due to increased intracranial pressure. The two symptoms—headache and a marked degree of optic neuritis—are alone very strongly suggestive of tumour of the brain. (iv.) Paralysis of other cranial nerves, depending on the position, but especially of the sixth, fourth, and third, owing to their relatively long course within the cranium. (v.) Incomplete hemiplegia, coming on slowly and insidiously, but progressively, with rigidity and the other features of the upper neuron paralysis, accompanied by other symptoms according to the situation of the tumour (§ 554). Mental apathy and delayed cerebration are usually present.

(7) Abscess of the Brain, the seventh cause of hemiplegia, is rarely primary. It may arise from (1) direct extension, as from disease of the cranial bones, syphilitic, tuberculous, or after injury, or following diseases of the mastoid, ethmoidal, frontal or nasal sinuses. (2) Abscess also arises from septic blood conditions—pyæmia, emboli from malignant endocarditis, or from the lungs when affected with tubercle, bronchiectasis, gangrene, or abscess. It has followed influenza and the specific fevers. The symptoms of cerebral abscess resemble those of tumour, and differ according to the site. Abscess of the temporal lobe after middle ear disease is not uncommon, and in this case the hemiplegia is of slow onset, is ill-defined, and the face is notably involved. The other symptoms are described in Intracranial Inflammation, § 550. Pyrexial symptoms are only found at the onset and the conclusion of the case.

(8) Chronic Meningitis and Pachymeningitis (fibrous thickening of the meninges) sometimes occur (though rarely) as solitary lesions. The condition is mostly due either to chronic alcoholism, syphilis, or injury, and some say that in the absence of these, chronic meningitis may be excluded. Tuberculous meningitis may also cause hemiplegia, especially in children. (i.) The advent is usually gradual; (ii.) the degree of the hemiplegia is slight; (iii.) optic neuritis is frequently present; (iv.) very generally some of the cranial nerves are involved as they pass through the membranes—lesions of the sensory nerves causing pain, disease of the motor nerves causing atrophic paralysis with R. D. (v.) There are always some signs of mental alteration, and not infrequently epileptiform convulsions from irritation of the cortex. Indeed, epilepsy occurring for the first

[1] "The Diagnosis of the Nature of Organic Brain Disease," Phonographic Medical Library, vol. i., 1897.

time in a person of thirty or forty is very probably due to syphilitic disease of the meninges.

Syphilitic Meningeal Affections may also take the form of a gummatous deposit, either primarily or secondarily to osseous lesions. These are distinguished clinically by severe and continuous headache, and the presence of irritative signs.

(9) Altered Blood States, such as those due to chorea, severe anæmia, pregnancy, typhoid fever, influenza, scarlet and other fevers, may give rise to hemiplegia. The lesion is possibly a thrombosis or some more transient vascular change. It is recognised by (i.) the antecedent history, and (ii.) the incompleteness and transitory nature of the paralysis.

(10) Hemiplegia may also form a minor feature in the course of several Chronic Degenerative Conditions of the nervous system, such as disseminated sclerosis, general paralysis of the insane, and bulbar paralysis, the diagnosis of which is dealt with elsewhere.

Prognosis of Hemiplegia.—If the paralysis has been considerable, one can hardly hope for complete recovery. The most favourable kinds, both for recovery and for life, are those due to injury, hysteria, and an altered blood state. Next in order comes syphilis, which, if diagnosed early and treated thoroughly, may generally be cured. All the other causes are more serious. Localised cerebral tumours are sometimes removable, and life may be prolonged, but the paralysis rarely disappears entirely. Abscess is capable of surgical treatment. The most unfavourable kinds of hemiplegia are those due to embolism, thrombosis, and hæmorrhage, the last-named being the gravest of all ; and the usual history in these cases, even if the patient recover from the apoplexy, is that a recurrence takes place during the ensuing year or two, in which the patient dies. As regards symptoms, it is usual for the leg to recover before the arm ; if the converse of this happens, the prognosis is certainly less favourable, as showing that the damage is too great for the substitution of function which leads to the recovery of the leg. The formation of blisters or sloughs over the gluteus is of bad prognostic significance. The general condition of the patient, and the existence of some other disease, such as chronic granular kidney, may also constitute unfavourable circumstances.

Treatment of Hemiplegia.—In hæmorrhage the patient should at first be kept at perfect rest ; the treatment of hæmorrhage, embolism, and thrombosis should be directed to the cardio-vascular system (see § 531). Intracranial tumour and abscess, unless we can positively exclude syphilis, should first be put on full doses of iodide and mercury while the locality is being accurately diagnosed, with a view to surgical interference should these remedies fail. Headache and the other symptoms of tumour may be relieved by lumbar puncture (pp. 921 and 930). For aneurysm full doses of iodide of potassium are useful. Chronic meningitis, if of a syphilitic nature, should be treated accordingly ; if due to injury, and the position of the focus of the disease can be diagnosed, surgery may be invoked. As regards the treatment of the paralysed limbs, a great deal can be done by the judicious application of massage, galvanism, and galvano-

faradism. The first improves the nutrition of the muscles considerably, the second and third maintain and improve their functional activity. Galvanism only may be applied to rigid muscles, and general hygienic measures will aid. Electricity should not be started till two months after a cerebral hæmorrhage, and should be stopped if headache, faintness, or fatigue appear. If the motor tract is definitely destroyed, nothing will restore its functions completely, but a good deal may be done by the means just mentioned, and by the education of other centres to take on the functions of those destroyed. Strychnine must be avoided when the muscles are rigid.

§ 554. The **Localisation of Intracranial Lesions** may be considered first in regard to certain recognised types of paralysis; secondly, certain groups of symptoms corresponding to the three basal fossæ; and, thirdly, the symptoms pointing to positions in the encephalon.

A. Lesions in certain positions give rise to certain TYPES OF PARALYSIS.

(1) Hemiplegia is usually due to a lesion in the internal capsule.

(2) "Crossed hemiplegia" (face on same side as lesion, body on opposite side) is typical of a lesion in the lower part of the pons.

(3) Paralysis of the facial and sixth nerves on same side, hemiplegia of opposite side, signifies lesion in pons at level of exit of facial nerve (Millard-Gübler syndrome).

(4) Incomplete paralysis of the third nerve on same side, with hemiplegia or hemiataxy and facial paralysis on the opposite side, is typical of a lesion in the crus cerebri (Weber's syndrome).

(5) Oculo-motor paralysis of the same side as the lesion, with tremor of the leg and arm on opposite side, indicates a superficial lesion of one crus extending into the tegmentum and the neighbourhood of the red nucleus (Benedikt's syndrome).

(6) Conjugate deviation of the eyes indicates a lesion at or generally above the ocular nuclei. With destructive lesion of the cortex the eyes look towards the same side as the lesion, but they look *away* from *irritative* lesions.

B. Intracranial tumours in the—

CORTEX (see Figs. 147 and 148, pp. 714 and 715) usually produce convulsions.—*Rolandic area*, Jacksonian convulsions starting in part controlled by affected area; tender spot on skull over Rolandic area. Paralysis of opposite side flaccid at first; monoplegia, rarely complete hemiplegia; impaired muscle sense; ataxy in finer movements (*e.g.*, fastening buttons). If *left inferior frontal* involved, aphasia (motor speech). The convulsions are preceded by aura of flashes of light, if the lesion is in the occipital cortex; by noises or music, if superior temporo-sphenoidal cortex; by odour, if tip of temporo-sphenoidal cortex.

POSTERIOR FOSSA—resemble symptoms of cerebellar (below), pontine, and medullary tumours, but without conjugate deviation. Cranial nerves from VI. to XII. may be involved (Fig. 156, p. 800).

MIDDLE FOSSA—lesion of one third nerve, and symptoms of involvement of crus cerebri (hemiparesis).

ANTERIOR FOSSA—paralysis of nerves entering the orbit, loss of sight and smell on side of lesion. Sometimes no symptoms except mental dulness.

C. Destructive lesions in different positions in the encephalon are indicated as follows—

FRONTAL LOBE—mental torpor and depression, sometimes exophthalmos on the side of a tumour, and perversion of smell. Usually no motor or sensory disturbance.

OCCIPITAL LOBE—altered field of vision (hemianopia); if bilateral, blindness; if involving left angular gyrus, word-blindness.

TEMPORO-SPHENOIDAL LOBE—deafness; if in left superior convolution, word-deafness; if tip, taste and smell affected.

Corpus Callosum—(i.) Gradual onset of hemiplegia, with vague hemiplegic symptoms on other side; (ii.) mental torpor; (iii.) coma and death without involvement of cranial nerves; (iv.) headache and vomiting rare throughout.

Internal Capsule—hemiplegia, face and body, of opposite side. When posterior part involved, hemianæsthesia, hemianopia, disturbed hearing, tremor (especially when optic thalamus also involved), hemichorea, and athetosis. In a linear (antero-posterior) lesion, paralysis of face and leg, arm escaping.

Optic Thalamus—hemianopia, hemianæsthesia, post-hemiplegic chorea. Cannot with certainty be distinguished from lesions involving the sensory part of internal capsule and optic radiations. Paralysis of emotional movements of face together with post-hemiplegic choreic disorders are in favour of a lesion involving opposite thalamus.

Corpora Quadrigemina—*superior corpora :* nystagmus, loss of pupillary contraction to light and accommodation, ataxy if fibres to medial lemniscus involved. *Inferior corpora :* auditory disturbance, partial deafness of both and especially of opposite ear. Defective mastication from involvement of motor root of V., and the fourth nerve is sometimes involved. Lesions of corpora quadrigemina simulate locomotor ataxy or cerebellar tumour. Ophthalmoplegia and reeling gait, especially if associated with bilaterally defective hearing, render diagnosis probable.

Crus Cerebri—simultaneous onset of III. nerve paralysis on side of lesion, and of hemiplegia on opposite side, is very characteristic; and if *tegmen* involved, hemiataxy of opposite side.

Pons—very varied symptoms; most characteristic are combination of paralysis of V., VI., and VII. on side of lesion, and of body on opposite side. *Upper part of pons :* Paralysis and anæsthesia of face and body on side opposite to lesion. *Lower part of lesion :* "crossed paralysis," face on side of lesion, body on opposite side; VI. and VII. paralysed together; conjugate deviation to side away from destructive lesion; in acute lesions, contracted pupils and hyperpyrexia.

Medulla—difficulty in articulation and swallowing, associated with disturbances of heart and respiration and paresis or paralysis of limbs on one or both sides. All cranial nerves from VIII. to XII. affected. Often secondary to cerebellar or pontine disease.

Cerebellum—ataxy (reeling), static and dynamic, not affected by closing eyes; movements fairly co-ordinated when in bed. Vertigo. Nystagmus. Paresis of trunk or limbs. K. J.'s sometimes absent. Hydrocephalus by pressure on veins of Galen leading to distended ventricles. Compare p. 845.

§ 555. Hemiplegia in Children differs from that in adults in several respects. It is met with in congenital and in acquired forms, the latter being usually under ten years old. The child has a sudden fit of convulsions, followed by coma and hemiplegia. In after life, athetosis and post-hemiplegic chorea are common sequelæ, and Jacksonian epilepsy is also seen. The affected limbs do not grow so well as the others, though the muscles are not really wasted, and some may be hypertrophied from the constant movement in athetosis. The mental condition in such patients varies—it may be normal or merely dull; others, especially the congenital cases, are idiots, with squint.

The *Prognosis* as to recovery is bad, but the duration of life is not affected by this disease except in the congenital and mental cases, who frequently die in early life from intercurrent diseases.

Etiology.—Congenital hemiplegia occurs from (i.) injury at birth, or protracted labour causing meningeal hæmorrhage, or crushing the hemispheres together (Little's paralysis); (ii.) porencephalus (cavities in the brain); and (iii.) atrophy of one hemisphere. Acquired hemiplegia comes on in the majority of cases before the second year, rarely after the tenth year. The hemiplegia in such cases is apt to come on during or after an acute specific fever, and has been found after death to be due to embolism, thrombosis, or hæmorrhage. Tuberculous meningitis and tumour, cerebral abscess, polio-encephalitis superior and trauma are also causes of hemiplegia in children.

Treatment.—The possibility that infantile convulsions are associated with cerebral lesion should be remembered, and bromides and a purge should be given. The hemiplegia may profitably be treated by electricity and massage. The rigidity and contractures are treated by massage and exercises.

The patient complains of weakness or **paralysis** *of* BOTH LEGS. The disease (general debility being excluded) is PARAPLEGIA.

§ **556. Paraplegia** is most often due to some structural or functional disease of the spinal cord, for it is here that the motor tracts of the two sides of the body run side by side, and can therefore be affected by a single transverse lesion. Paraplegia may also be due to disease of the peripheral nerves.

It will be found convenient for clinical purposes to make three groups of paraplegias :

(*a*) In **upper neuron, spastic** or **rigid paraplegias,** the paralysis is persistent and progressive, the muscles are rigid, with no tendency to atrophy, beyond that due to disuse, the deep reflexes are increased, and there are no electrical changes .. .. .. § 557

(*b*) Paraplegias of the **lower neuron** type are **flaccid.** In this type the paralysis is persistent and continuous from day to day, the muscles are atrophied, the deep reflexes are absent or diminished, and there is the reaction of degeneration .. .. § 561

(*c*) In the **functional** or **variable paraplegias** the paralysis is less pronounced and less persistent than in the foregoing ; it is apt to vary from day to day, and there are neither atrophy nor electrical changes .. .. § 564

CLINICAL INVESTIGATION.—Assuming that the case is really one of paraplegia, and not simply a stiffness of the joints due to gout, rheumatism, or old age, it is desirable (1) to investigate the history of the case, (2) to examine the spinal column, and (3) to ascertain whether the paralysis conforms to the upper neuron type, the lower neuron type, or the functional or variable type.

(1) The history of the case throws considerable light on the nature of the lesion, and particularly in regard to the mode of onset and evolution, the history of pain, and the age of the patient. The onset is very rapid in vascular lesions or those of an acute inflammatory type. The onset takes place more gradually in some forms of compression paraplegia, such as Pott's disease (§ 557), in multiple neuritis, chronic myelitis, and syringomyelia. The history of pain in the spine or legs is of importance, and some observers even go so far as to divide paraplegias into painful and painless. The presence of pain indicates an involvement of the nerve trunks or the posterior nerve roots, as in meningeal affections and tumours. Paraplegia in *childhood* in a large proportion of cases is due to Pott's disease or infantile palsy. *Adults* from twenty to forty are chiefly affected by paraplegia due to multiple neuritis, hysterical or syphilitic paraplegia. In *advanced* life the slow degenerative lesions are more often met with.

(2) Local examination of the spine should never be omitted. It will teach us at a glance whether or not angular curvature or malformations be present. Percussion down the spine may elicit tenderness, as in Pott's disease, tumour, or meningeal affections. Vertical pressure upon the head while the patient is sitting or standing will cause severe pain if vertebral caries or cancer be present, but not in hysterical or neurasthenic conditions. Rigidity or restricted movement accompanies all painful organic affections of the spine, as in meningeal diseases.

(*a*) The **upper neuron, spastic,** or **rigid paraplegias** form the largest of the three groups, and it contains a most important sub-group—viz., the compression paraplegias. The three features of this kind of paraplegia are those which belong to all paralysis due to lesions of the upper neuron—viz., rigidity, increased deep reflexes, and no marked muscular wasting. The members of this group differ from the functional paraplegias in their steady and usually progressive course. The following are the causes of the spastic paraplegias, placed more or less in order of frequency :

Compression Paraplegias.

I. Compression paraplegia due to Pott's disease.
II. Compression paraplegia due to tumours.
III. Injury.

Inflammation of the Cord or its Membranes.

IV. Myelitis and its varieties.
V. Hæmorrhage (spinal and meningeal).
VI. Embolism.
VII. Chronic spinal pachymeningitis.

Chronic System Lesions (Sclerosis).

VIII. Disseminated sclerosis, general paralysis of the insane, and tabes dorsalis —occasionally.
IX. Primary lateral sclerosis.
X. Ataxic paraplegia and Friedreich's disease (occasionally).
XI. Amyotrophic lateral sclerosis.
XII. Infantile cerebral and spinal paraplegia and other double cerebral or cerebellar lesions.
XIII. Toxic sclerosis.

Malformations.

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§ **557. Compression Paraplegia** is perhaps the commonest form of **spastic paraplegia,** and is due to compression of the spinal cord, either by spinal caries (Pott's disease), or other disease or injury of the vertebra, or a tumour pressing upon the spinal cord.

The *Symptoms* common to all forms of compression paraplegia, in addition to those just named belonging to upper neuron paraplegias, are (1) pain at some time from pressure upon the nerve roots, and, generally speaking, it may be said that when the pain precedes the paraplegia the lesion is extradural, when it is *vice versa* the lesion is probably intradural. (2) The symptoms are apt to be more marked in one leg than in the other (with the notable exception of Pott's disease).

I. **Vertebral Caries** (Pott's Disease, Tuberculosis of the Spine) may be regarded as the type of compression paraplegias. It used to be the commonest cause of paraplegia in young persons. It is the commonest of the compression paraplegias, injury coming next, and vertebral cancer next. The paraplegia in Pott's disease is due to the pressure of the inflammatory products which accumulate outside the dura mater and compress the cord. Its differential features are :

(1) In the incipient stage the child probably complains of "stomach-ache" or pains in the region of the umbilicus, the back, or the loins, due to pressure on the nerve roots. (2) The onset of the paralysis is usually gradual, and is rarely quite complete, but on rare occasions the onset is rapid. Both legs are affected equally. (3) There are marked rigidity and increase of the deep reflexes, often twitchings of the muscles. (4) In the early phases jarring by percussion on the head or jumping on the heels causes pain; there is generally pain and tenderness of the spine, and in course of time other indications appear at the seat of the mischief. (5) The subjects of the disease are mostly children or young persons who have a family or antecedent history of tuberculosis, and perhaps other manifestations of that disease. (6) The course is always protracted. Spinal caries, even without operation, tends in the long run to become quiescent, and if the patient is able to undergo prolonged rest, it is wonderful how the use of the legs may be restored, even after complete paralysis. I have seen cases recover after a complete paralysis lasting two or three years, and have made autopsies on patients who could walk whose cord at one spot was no larger than a quill. The *Diagnosis* is only difficult when there is no curvature or other local indication of caries. Of the other causes of extradural compression paraplegia, cancer and aneurysm are the commonest. They occur in older subjects, and are associated with great increase of the pain on movement. Exostoses can only be suspected when found also in other parts of the body. These and all the other causes of compression paraplegia are distinguished from Pott's paraplegia by (1) the unilateral predominance of the symptoms, and (2) pain being relatively a more prominent symptom. For the diagnosis of the position of the lesion, see below.

II. **Compression Paraplegia due to Spinal Tumour.**—Tumours of the spinal cord may be (α) extra-medullary (arising outside the spinal cord) or (β) intra-medullary (arising within the spinal cord). Extra-medullary tumours are the more common, and it is these which produce typical compression paraplegia.

α. Extra-medullary Spinal Tumours may be extra- or intra-thecal, but these two cannot be clinically distinguished. The following are the principal extra-medullary tumours—syphilitic gumma, sarcoma, carcinoma, tuberculous growths, myxoma, fibroma, meningeal hæmorrhage, hydatid cysts, chondroma, exostoses, lipoma (rare), or neurofibroma (springing from the nerve roots in the canal). Aneurysm, carcinoma, and sarcoma may arise outside the vertebral column, and invade the cord after eroding the vertebræ, but these three lesions generally have their own proper symptoms.

Symptoms of Extra-Medullary Spinal Tumours.—(1) Pain radiating in the distribution of the nerve roots pressed on is usually the earliest indication, and is rarely absent. Generally it is one-sided, round the chest or abdomen, or down the leg; sometimes there is pain at the site of the tumour. (2) Progressive paraplegia *starting and predominating in one leg*, and spreading upwards from the toes, more marked on the same side as the pain, and associated with (3) anæsthesia having the same features. Finally, both the paralysis and the anæsthesia become complete up to a definite level. The upper limit of the anæsthesia is the best guide as to the seat of the tumour, bearing in mind that the posterior roots enter the cord two or more inches (in the dorsal region) above their entrance into the spinal

canal (Fig. 157), and therefore the tumour must be cut down upon well above the anæsthetic limit. Of the two sub-groups of extra-medullary tumours, extra-thecal lesions are more often attended by pain, and intra-thecal lesions by cramps and contractures of the muscles at night, indicating compression myelitis. The superficial abdominal reflexes are sometimes abolished on the same side as the tumour—the supra-umbilical reflexes in tumours between the eighth and ninth, the infra-umbilical in tumours lying between the eleventh and twelfth dorsal vertebræ. Sometimes the umbilicus is drawn towards the healthy side. In meningeal hæmorrhage the symptoms of pain and paralysis are of sudden onset.

The *Diagnosis* of such cases rests on the presence and character of the pain, the progressive evolution of the symptoms, and their predominance on one side. Cervical meningitis "causes" pain on both sides. The *Prognosis* depends mainly on the nature and position of the tumour, and whether secondary myelitis has occurred. The *Treatment* (apart from anti-syphilitic remedies) is surgical.

β. Syringomyelia and Intra-medullary Tumours (§ 607) sometimes come under notice for paralysis of the legs, and this paraplegia may be very peculiar and contradictory—namely, an *atrophic* paralysis *associated with rigidity and increased knee-jerks.* The paraplegia is sometimes of an ordinary spastic type, but as a rule this only occurs late in the disease. The earlier symptoms of syringomyelia usually appear in the arms (sensory and trophic changes). But the symptoms of an intra-medullary tumour necessarily differ with the position of the tumour and the column chiefly involved.

III. **Injury to the Spinal Column** may cause displacement or damage to the vertebra (fracture-dislocation) or hæmorrhage, or may initiate acute myelitis or meningitis (*q.v.*). The symptoms following injury are those either of destruction (paralysis), or irritation (spasm), and the history of the injury generally enables us to make a diagnosis.

In cases of transverse myelitis after fracture-dislocation, it was formerly taught that the reflexes below the lesion were exaggerated unless and until the disease had, by descending, destroyed some part of the reflex arcs. But Dr. Charlton Bastian has shown that in cases of transverse myelitis the reflexes below the lesion may be absent, even without such extension downwards.

§ 558. The **Localisation of Spinal Lesions,** (*a*) as regards its level, is effected in three ways : (1) By the distribution of the motor weakness ; (2) by the alteration of the reflexes ; and (3) by the level of the upper limit of the pain and anæsthesia (see Head's Areas below). (1) and (2) are given in the table on pp. 810, 811.

(*b*) The particular column which is affected can be ascertained by studying the table in § 501.

Head's Areas of Analgesia and Hyperalgesia (pp. 812 and 813) may be of some use for the localisation of a spinal injury or lesion, but they are difficult to elicit and define. They have been elaborated by the successive researches of James Ross,[1] James Mackenzie,[2] William Thorburn,[3] and, more particularly, Henry Head.[4] These are areas of diminished or increased *sensibility to pain,* which correspond with certain spinal segments, as marked in the illustration. The reader is asked to remember in what follows that we are not now concerned with tactile sensation, but only with sensibility to pain (algesia). A destructive lesion of a spinal segment or nerve root is attended by analgesia of the corresponding area, an irritative lesion by hyperalgesia. The sensibility to pain of the skin below the spinal lesion

[1] *Brain,* January, 1888, part x., p. 333 ; and " Diseases of the Nervous System."
[2] *Brain,* 1893, part xvi., pp. 321 and 515 ; and *Med. Chronicle,* August, 1892.
[3] *Brain,* 1893, part xvi., p. 355.
[4] *Brain,* 1893, part xvi., p. 1 ; 1894, part xvii., p. 339 ; 1896, part xix., p. 153.

is, of course, disturbed, but to localise a spinal lesion you should carefully test the sensibility from above downwards with the point of a pin, and you will find an *abrupt margin of normal sensibility to pain* at the upper border of the affected area. The important question really, is to what level does the sensibility to pain remain normal. Head's areas were discovered mainly by the investigation (1) of cases of injury to the spinal nerve roots, (2) cases of visceral disease associated with soreness of the skin or reflex pain, and (3) cases of herpes.

Functions of the Spinal Segments.

Segment of Cord and Spinous Process.	*Muscles supplied.*	*Reflex.*	*Sensation.*
II. and III. C. Opposite 1st C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. C. Opposite 2nd C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus. Levator ang. scapulæ.	Pupil reflex = 4th to 7th cervical—*i.e.*, dilatation of the pupil produced by irritation of neck.	Neck. Shoulder. Outer arm.
V. C. Opposite 3rd C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus and brevis. Rhomboids and teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular reflex = 5th cervical to 1st thoracic—*i.e.*, irritation of the skin over the scapula produces contraction of the scapular muscles. Tapping tendon of supinator longus in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. C. Opposite 4th C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps reflex = 5th to 6th cervical — *i.e.*, tapping elbow tendon produces extension of forearm. Posterior wrist reflex = 6th to 8th cervical—*i.e.*, tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. C. Opposite 5th C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapularis. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist reflex = 7th to 8th cervical —*i.e.*, tapping anterior tendons causes flexion of wrist. Palmar = 7th cervical to 1st thoracic—*i.e.*, stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. C. Opposite 6th C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I. D. Opposite 7th C.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.

FUNCTIONS OF THE SPINAL SEGMENTS—*continued.*

Segment of Cord, and Spinous Process.	*Muscles supplied.*	*Reflex.*	*Sensation.*
II. to XII. D. Opposite 1st to 10th D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric reflex = 4th to 7th thoracic—*i.e.*, irritation of mammary region causes retraction of epigastrium. Abdominal = 7th to 11th thoracic — *i.e.*, stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.
I. L. Opposite 11th D.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric = 1st to 3rd lumbar — *i.e.*, stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L. Opposite 11th D.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Tapping patellar tendon causes extension of leg.	Outer side of thigh.
III. L. Opposite 12th D.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. L. Opposite 12th D.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal = 4th to 5th lumbar—*i.e.*, stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L. Opposite 12th D.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I. to II. S. Opposite 1st L.	Flexors of ankle, and toes. Peronæi.	Plantar reflex.	Back of thigh. Leg and foot, outer side.
III. to V. S. Opposite 1st L.	Perineal muscles.	Ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

These areas may also have two other possible uses. In certain *visceral diseases* groups of these areas are apt to be the seat of tenderness, and even pain, which is probably of a reflex nature ; single area tenderness is practically never met with. To test these areas the rounded head of a lady's hat-pin is best, its smooth surface evoking the sensation of soreness. The various organs stand in connection mainly with the areas as shown in table on p. 814, the areas being those indicated in the figures (Dr. Henry Head in " Quain's Dictionary of Medicine," third edition, p. 1138). There are also certain smaller *spots* of maximum tenderness to which a patient chiefly refers his pain, which remain tender longer than the rest of the skin around. The whole of this question is at the present time one of more scientific than practical interest.

Head's areas also correspond to the areas affected with attacks of *herpes* (zoster), which is now almost certainly proved to be due to some irritative lesion of the posterior root or root-ganglion, or of the corresponding spinal segment. It was

indeed, the careful study of a large number of cases of herpes and the careful examination of twenty-one fatal cases which enabled Dr. Henry Head to map out these algetic areas with the precision which he has achieved.

§ **559.** IV. **Acute Transverse Myelitis** is a transverse softening of the cord, due to inflammation, thrombosis, or some other similar vascular

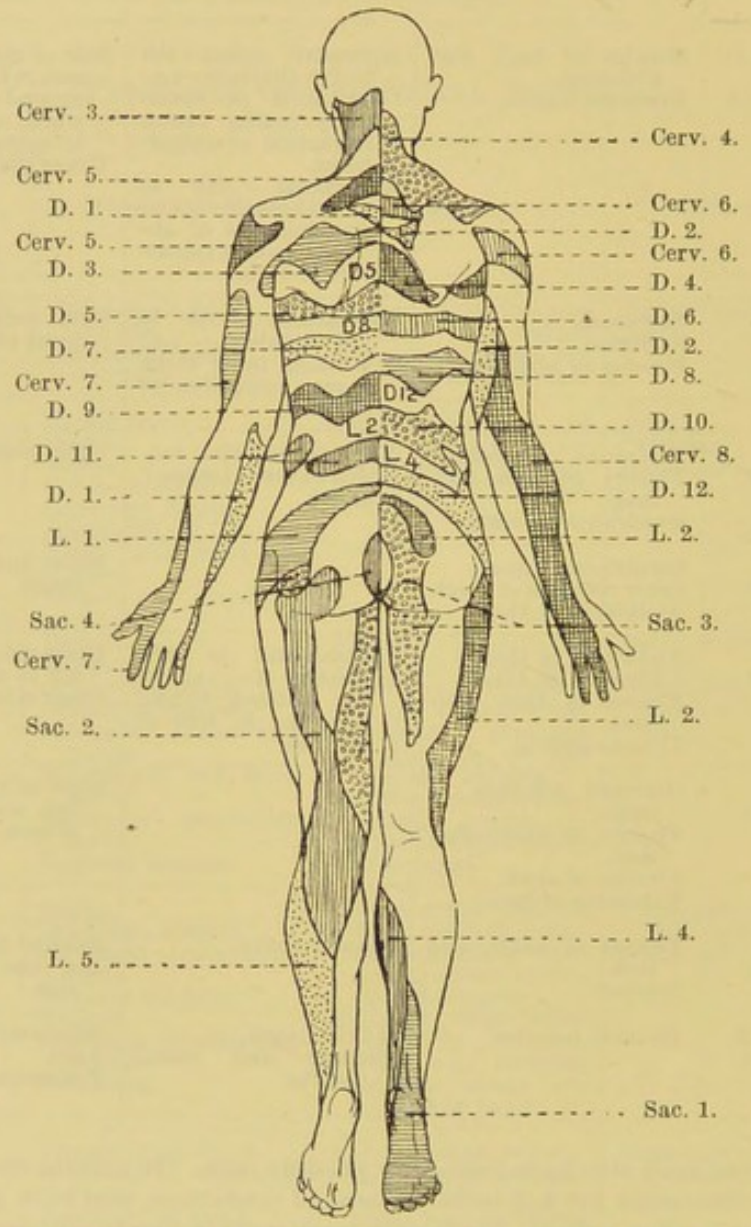

Fig. 157.—Segmental Algesic Areas of Head—*i.e.*, areas of increased or diminished cutaneous sensibility to pain when the corresponding spinal segment is irritated or destroyed respectively.

lesion, characterised in typical cases by the completeness of the loss of sensation and motion below the lesion, by the "girdle" pain, and by the tendency to bedsores and sphincter troubles. The dorsal region is the most usual position of the lesion. This paraplegia *at first* is flaccid.

Symptoms.—1. The advent may be as sudden as apoplexy, or it may occupy a few days. Generally it is attended by slight pyrexia.

According to the severity of the onset, cases are described as acute or subacute. 2. The "girdle" pain is a characteristic symptom, consisting of a feeling as of a constricting cord around the trunk opposite the upper limit of the lesion. There is also a band of hyperæsthesia and increased superficial reflexes in that position. Below this level the sensation and superficial reflexes are lost. 3. Paralysis and anæsthesia

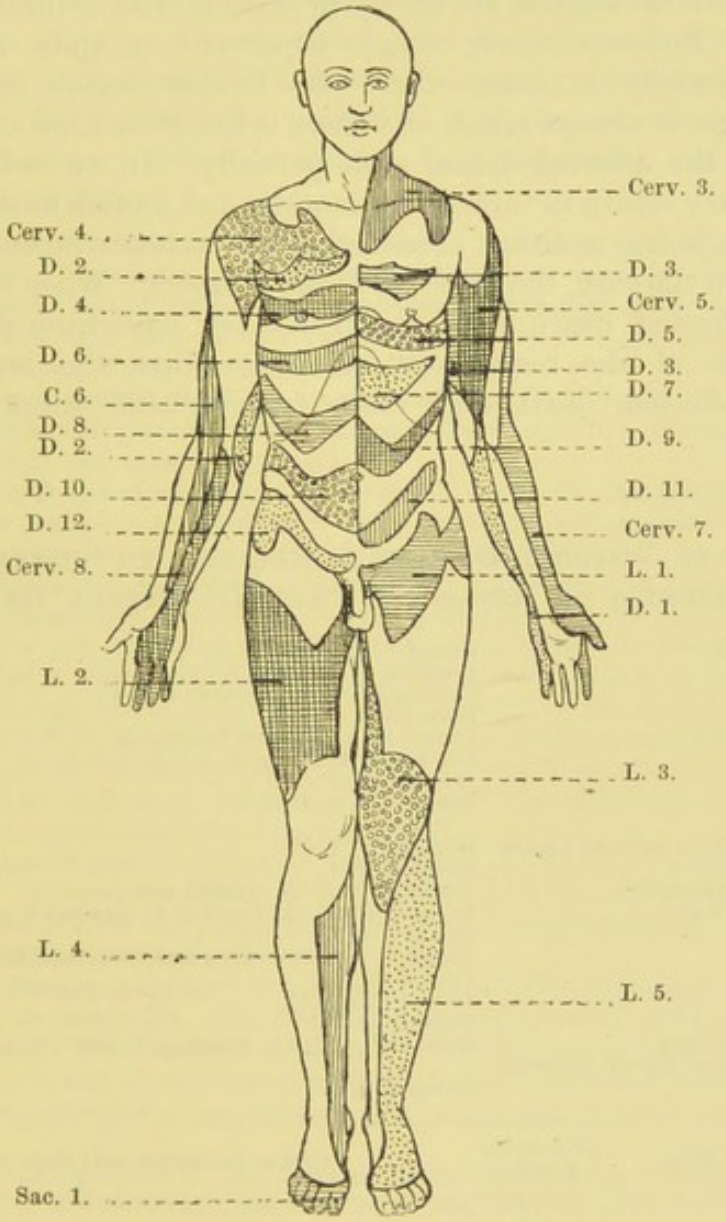

Fig. 158.—Front view of Fig. 157.

involve the whole of both legs. The paralysis is flaccid in the early stage, but the muscles gradually become stiff in the course of a few weeks owing to the descending sclerosis. There is no atrophy and no Erb's reaction, unless the cervical or lumbar enlargements are involved, when the paralysis is of an amyotrophic type (§ 600). The extent of the paralysis, and whether the trunk and arms are involved, depend

upon the situation and extent of the lesion. Involuntary startings of the legs are common. 4. The deep reflexes below the lesion are at first abolished, but they rapidly return (except when the lumbar enlargement is involved), then become exaggerated and accompanied by ankle-clonus. 5. Retention of urine and fæces is present at the onset; later on evacuation takes place without the patient being conscious of it. If the lumbar centres are involved, there is true incontinence from the onset. Bedsores nearly always supervene, in spite of the best nursing, especially in cases where the lumbar region is involved. 6. The course is always rapid, and there is but little hope of regaining the use of the affected limbs, even partially. In an ordinary case sensation may return in three to six months, and motion to some extent in six to eighteen months. The higher up or lower down the lesion is situated in the cord, the more grave the prospect. When the cervical region is affected, death often occurs in a few days from pneumonia, or paralysis of the respiratory muscles. When the lower dorsal region is affected, death may ensue from cystitis, surgical kidney, or bedsores.

Table of Visceral Disease in Relation to Superficial Tenderness or Reflected Pain (Dr. Henry Head).

Heart, Ventricle	Dorsal 1 (?), Dorsal 2, 3, 4, 5.
Auricle	Dorsal 5, 6, 7, 8, and (?) 9.
Aorta, Arch	Cervical 3 and 4, Dorsal 1, 2, 3, 4.
Dorsal Aorta	Dorsal 5, 6, 7, 8, 9.
Abdominal Aorta	Dorsal 10, 11, 12, and Lumbar 1.
Lungs	Cervical 3, 4, Dorsal 3, 4, 5, 6, 7, 8, 9.
Œsophagus	Dorsal 5, 6, 7.
Stomach	Dorsal 6, 7, 8, 9, 10.
Intestine—	
1. Duodenum to Sigmoid Flexure	Dorsal 10, 11, 12.
2. Rectum	Sacral 2, 3, 4.
Liver and Gall Bladder	Dorsal 7, 8, 9, 10. (Right side.)
Kidney and Ureter	Dorsal 10, 11, 12, Lumbar 1, Lumbar 2 and Occipital.
Bladder	Sacral 2, 3, 4.
Prostate	Sacral 2, 3, 4, Dorsal 10 and (?) 11 and Occipital.
Testicle	Dorsal 10.
Epididymis	Dorsal 11 and 12.
Ovary	Dorsal 10.
Uterine Appendages	Dorsal 10, 11, 12, Lumbar 1, and (?) Lumbar 2.
Cervix Uteri and Lower segment of Uterus	Sacral 2, 3, 4.

Area on the Scalp.	*Area on the Trunk.*	*Organs in relation with these Areas.*
Frontonasal.	Cervical 3. Cervical 4.	Lung (Apices), Stomach, Liver (Occipital).
Midorbital.	Dorsal 2. Dorsal 3. Dorsal 4.	Heart (Ventricle), Aortic Arch, Lungs.
Frontotemporal.	Dorsal 5.	Lung, Heart.
	Dorsal 6.	Lungs (lower lobes), Heart (Auricle).
Temporal.	Dorsal 7.	Lungs (lower lobes), Heart (Auricle), Stomach (Cardiac end), Liver (R. side).
Vertical.	Dorsal 8.	Stomach, Liver, and Gall Bladder, Lungs.
Parietal.	Dorsal 9.	Stomach (Pyloric end), Liver.
Occipital.	Dorsal 10.	Intestine, Liver, Ovary, Testis, Stomach (Occipital).

The *Diagnosis* in typical cases of myelitis is not, as a rule, difficult, on account of the girdle pain, rapid involvement of the bladder and rectum, and the completeness of the paralysis and anæsthesia. Compression paraplegia presents pain, is of more gradual onset, and there may be unilateral predominance. Acute myelitis may also have to be diagnosed from hæmorrhage or embolism of the cord (see below).

Causes.—Acute myelitis is more common in males between ten and forty years of age. Among the causes may be mentioned injury, hæmorrhage, extension of inflammation from the meninges, compression, over-exertion, exposure, suppression of the menses, and various toxic blood states. Some cases have been associated with syphilitic disease of the arteries.

Chronic Transverse Myelitis may supervene on the acute, or it may be chronic from the onset. It presents the same characteristics as acute myelitis in a lesser degree—complete loss of motion and sensation in both legs, girdle pain, involvement of bladder and rectum, and tendency to bedsores. At first, before the rigidity supervenes, it may resemble multiple neuritis.

Acute diffuse or **Central Myelitis** is a rare form of acute myelitis, accompanied by marked pyrexia, widespread paresis, and anæsthesia (§ 573). It rapidly spreads to the whole cord, and is generally fatal.

§ **560. Vascular and other Lesions** of the Spinal Cord.—V. **Hæmorrhage** into the spinal cord is said by most observers to be very rare, unless preceded by some congenital cystic defect or some degenerative or neoplastic lesion, such as glioma.[1] The exacerbations of symptoms in syringomyelia are believed to be due to hæmorrhage. Intra-medullary hæmorrhage is characterised by the sudden onset of complete motor and sensory paralysis, accompanied at first by loss of superficial and deep reflexes, which return in an exaggerated degree a few days later unless the lumbar enlargement is affected. It presents most of the symptoms of acute myelitis, from which it can only be differentiated by its instantaneous occurrence, attended sometimes by severe localised pain. Hæmorrhage into the spinal membranes has similar symptoms, accompanied by pain due to pressure on the nerve-roots, as in other extra-medullary tumours (§ 557).

VI. Embolism of the Cord is rare. The patient complains of a severe "shock in the spine," followed by sudden and complete paralysis within a definite area, which corresponds with the position of the lesion. Cardiac disease or the other causes of embolism are in operation.

VII. **Spinal Pachymeningitis** (Chronic Spinal Meningitis, Meningeal Thickening), may give rise to paraplegia, with stiffness of the legs, of very gradual onset and prolonged course. This morbid condition is said by some to be infrequent, and not to give rise to any obvious symptoms, but at the Paddington Infirmary I had no difficulty in collecting seven cases verified by autopsy in the course of two or three years.

1. The leading *Symptom* of this form of paraplegia is pain shooting down the nerves of the leg and elsewhere, aggravated by any movement of the back ; sometimes extremely severe, and accompanied by tenderness of the spine. 2. Stiffness and weakness of the legs, involuntary twitchings and increased reflexes were present in my cases as the disease progressed. 3. Patches of hyperæsthesia and anæsthesia were occasionally present, and in some there was a progressive atrophic weakness of various muscles due to the constriction of the nerve roots. 4. In certain cases the thecal mischief spreads to the spinal cord, and various symptoms may arise according to the column involved. When the posterior column is affected, as in the cases alluded to in § 579, the patient may present all the symp-

[1] See an interesting clinical lecture by Sir William Gowers in the *Lancet*, 1903, vol. ii., p. 993.

toms of tabes dorsalis. The course of my cases varied from two or three to about ten years, death supervening from some intercurrent malady.

The *Causation* is obscure. Syphilis was present in the history of some, but none of the cases seemed very amenable to anti-syphilitic treatment.

Cervical Pachymeningitis (Hypertrophic Cervical Meningitis) is the same anatomical condition as the preceding, limited to the cervical region. It was first described by Professor J. M. Charcot. Pain and atrophic paralysis affect the arms, and subsequently spastic paraplegia, due to descending lateral sclerosis.

VIII. **Chronic System Lesions.**—Three degenerative diseases of the central nervous system of fairly common occurrence may first come under notice for weakness of the legs—disseminated sclerosis, general paralysis of the insane, and occasionally tabes dorsalis. Each have other and more characteristic symptoms, and will be dealt with elsewhere. There are also five other rarer conditions, in which stiffness of the legs, due to lateral sclerosis, is a leading feature—primary lateral sclerosis (lateral sclerosis only) ataxic paraplegia (lateral and posterior sclerosis), amyotrophic lateral sclerosis (sclerosis of the lateral column and anterior horns), infantile cerebral and spinal paraplegia, and toxic sclerosis (see Fig. 150 in § 502).

IX. **Primary Spastic Paraplegia** (Synonyms : Primary Lateral Sclerosis, Primary Sclerosis of the Crossed Pyramidal Tracts, Erb's Paraplegia,[1] Tabes Dorsalis Spasmodique).—Lateral sclerosis on one or both sides is commonly met with as a spreading downwards from a localised disease in the brain or cord, but the primary spastic paraplegia consists of sclerosis of these columns without any primary disease above. Some deny the existence of a primary lateral sclerosis, and, personally, I regard the disease as extremely rare, though some cases of spastic paraplegia seem to baffle all inquiries as to the primary or initiating lesion. (1) The onset is extremely slow and insidious, and the course is slow and painless, extending over many years, twenty or thirty being said to be not uncommon. (2) Dull aching and stiffness rather than absolute weakness is the leading symptom, and this gradually results in a stilted walk, till finally the patient walks on tiptoe without bending the knees, which is very characteristic. In advanced cases there is cross-legged progression, because the adductors are specially involved in the rigidity. Both legs are involved, though one may be a little worse than the other. Increased knee-jerk and ankle-clonus are present, as in other cases of lateral sclerosis. (3) The other symptoms consist of a series of negative points—viz., no disturbance of sensation, no sphincter trouble, no bedsores, and no alteration in the electrical reactions are the rule. In the later stages of the case the arms may become stiff.

Etiology.—The patients are usually from thirty to forty years of age. Deficient blood supply has been suggested, and syphilitic toxæmia, leading to disease of the arteries, is a possible cause. Chronic alcoholism and other toxic agencies have been suggested. The author has seen cases indistinguishable from Erb's primary spastic paraplegia completely recover when the oral sepsis from which they suffered had been cured.

X. **Ataxic Paraplegia,** or postero-external sclerosis, is a rare disease, due to primary sclerosis in the posterior as well as the lateral columns. There is defective co-ordination of the movements and ataxy, but none of the other symptoms belonging to locomotor ataxy. The knee-jerks are increased, and there is stiffness of the legs. This disease is met with chiefly in men of middle age, but much the same spinal lesions occur in Friedreich's disease—*i.e.*, the hereditary ataxy of childhood.

XI. **Amyotrophic Lateral Sclerosis** (Charcot) is a rare condition, due to disease affecting both the anterior horns and the lateral columns. The symptoms are

[1] Erb has described under the term "syphilitic spinal paralysis" an insidious spastic paraplegia, which he regards as distinct from primary spastic paraplegia ; but in the author's view this is practically identical with the above. Trans. West Lond. Med. Chir. Soc., about 1902.

usually confined to the arms for a time, and the disease is therefore described under Brachiplegia, § 567.

XII. **Infantile Cerebral and Spinal Paralyses** (including birth palsy, spastic diplegia, Little's disease).—The cerebral leg centres lie on the mesial aspect just below the margin of each hemisphere, and during difficult or prolonged labour these are liable to damage by compression of the hemispheres and meningeal hæmorrhage. In other cases diffuse atrophy has been found, or porencephalus. When the condition occurs in prematurely born infants it is believed to be due to defective development of the pyramidal tracts. Consequent on any of these lesions degenerative sclerosis takes place down the crossed pyramidal tracts, and spastic paraplegia results. Nothing, perhaps, is noticed until the child begins, or ought to begin, to walk, which it is late in doing ; then the legs are stiff, and signs of lateral sclerosis are present. The arms also may be involved—spastic diplegia. A limited athetosis or a more generalised chorea spastica may ensue. In all these varieties there is very often a history of convulsions in infancy and a condition of mental deficiency. In other cases (hereditary spinal spastic paraplegia), however, the child is born with a stiffness of the legs, but without any mental symptoms or backwardness, and in these cases there has possibly been an injury to the cord at birth. Such patients may live bedridden to a considerable age—forty or fifty years. Some recognise a genuine hereditary form of the malady, which affects several members of a family, in whom some developmental defects may be assumed to exist.

XIII. **Toxic Combined Sclerosis.**—Of late years sclerosis of the cord has been associated by the labours of Taylor,[1] Russell, and others with various blood conditions, such as diabetes, profound anæmia, leukæmia and pellagra. The paralysis is never very great. The symptoms vary according to which of the columns is mainly affected, but paraplegia is generally incomplete in degree, and is sometimes associated with ataxy and numbness. The sclerosis affects chiefly the posterior and lateral columns of the cervical and thoracic regions. It may be recognised from similar spinal affections by the previous occurrence of one of the diseases named or some other toxic disorder, anæmia being, perhaps, the most typical instance.

Progressive stiffness of the legs, resulting in spastic paraplegia, is the leading symptom of **lathyrism** (lupinosis)—*i.e.*, poisoning by the use of meal derived from the seeds of the chick-pea (lathyrus sativus and l. cicera) mixed with other meal in the preparation of bread. The arms are very rarely affected. It is met with in India (chiefly), Algeria, and probably elsewhere. The anatomical condition is not known, but it is probably a toxic sclerosis.

Group B. **The Lower Neuron or Flaccid Paraplegias** constitute a more limited group than the upper neuron paraplegias, and are distinguished from them by the four following features : (1) The paralysis is flaccid—at any rate, for a considerable time ; (2) muscular wasting is a marked feature, and is attended by (3) characteristic electrical changes ; (4) the deep reflexes are absent. This group is distingushed from the functional paraplegias (Group C) by the steady and progressive course of the lower neuron paraplegias, by the muscular wasting, and the electrical changes. They are as follows :

I.	Multiple peripheral neuritis	§ 561
II.	Beri-beri (which is a form of the preceding)	§ 562
III.	Anterior poliomyelitis	§ 601
IV.	Acute transverse myelitis (at the outset)	§ 559
V.	Landry's paralysis	§ 576
VI.	Syringomyelia and intra-medullary tumours, intra-medullary hæmorrhage and embolism (sometimes)	§ 607

[1] James Taylor, Roy. Med. Chir. Soc. Trans., 1895 ; J. R. Russell, the *Lancet*, 1898.

§ **561. I. Multiple Peripheral Neuritis** (Synonyms: Multiple Neuritis; Polyneuritis) is a symmetrical inflammation of the peripheral nerve-trunks (due to a toxæmia) attended with pain and tenderness along their course, resulting in paresis and anæsthesia. This disease is more or less general in its distribution, but the paresis may predominate in the legs, and the patient frequently comes under our notice for paraplegia.

Symptoms.—The onset may be acute, subacute, or (more usually) chronic. Some observers make three varieties, according to whether the motor, sensory, or ataxic symptoms predominate. In acute cases pyrexia may be persent. (1) There is generally a premonitory stage in which there are numbness, tingling, cramps and twitchings in the legs or arms. Pain forms a prominent feature in all cases of neuritis, the degree varying according to the acuteness of the process. The pain shoots along the course of the nerves, is symmetrical in distribution, and increased by movement. (2) There is also deep-seated tenderness along the nerve-trunks and in the muscle substance, especially in the calves. These symptoms are soon followed by (3) flaccid paresis attended by more or less atrophy. The extensors are more affected than the flexors, and the patient has a characteristic foot-drop (or wrist-drop), and a difficulty in raising the legs in mounting stairs. (4) The electrical reactions vary, but there is in all cases a diminution of faradic reaction. In many cases there is also a diminution to both galvanic poles (a condition which one does not obtain in anterior poliomyelitis), while in a few cases there is also typical Erb's reaction (§ 516), and A.C.C. is greater than K.C.C. (5) The superficial and deep reflexes are diminished or disappear. (6) Anæsthetic and hyperæsthetic areas at the termination of the sensory nerves in the legs and arms may, with care, be revealed. Often there is a patch of anæsthesia surrounded by a zone of hyperæsthesia. Bladder and rectal troubles and bedsores are only met with at a very advanced stage, though in alcoholic cases with the dull mental state the excretions may be passed in bed. It is an interesting fact that the nerves involved vary somewhat with the toxic agent in operation, as though the latter had some selective proclivity. Thus, alcohol and beri-beri affect mainly the legs; lead, the arms; diphtheria, the throat and neck muscles; while arsenic appears in a few cases to affect mainly the trophic nerves of the skin. In lead paralysis the toxin selects the motor fibres of the musculo-spiral nerve, and produces double wrist-drop. Accompanying severe alocholic cases is loss of memory, apathy, and sometimes delirium.

Peripheral neuritis may have to be *diagnosed* from tabes and poliomyelitis. It is recognised from the former by the actual muscular weakness present, the high-stepping walk, and the pupils; and from the latter by the sensory changes and the age of the patient. The

neuralgic pains may have to be differentiated from other causes of "pains in the limbs" (§ 422); and, lastly, the disease will often need to be distinguished from the other flaccid lower neuron pareses in this group, and from syringomyelia (§ 607). The acute febrile cases are hard to distinguish from Landry's paralysis and poliomyelitis in adults (§ 576).

Prognosis.—Multiple neuritis is essentially a chronic affection, but has a distinct tendency to recover. The chief danger rests in the involvement of certain nerves, such as the pneumogastric in diphtheritic paralysis, when sudden death may ensue. The acute febrile cases may die in a week from involvement of the respiratory muscles or cardiac paralysis, but, in general terms, a large proportion of cases of multiple neuritis get well under appropriate treatment in the course of three to about twelve months, if the cause in operation be removed. Deformities from contractures may result in severe cases.

Causes.—Multiple neuritis may occur in either sex and at any age, but is most common in adults from twenty to fifty. Women are slightly more affected than men. There is always some toxæmia or blood condition in operation, and the commonest of these in adults is alcohol, in children, diphtheria. The other causes are: (*a*) Among the *heterotoxins*—lead, arsenic, silver, mercury, ether, bisulphide of carbon and naphtha; (*b*) among the *microbic toxins*—influenza, tuberculosis, leprosy, beri-beri, enteric fever, variola (?), rheumatic fever, scarlatina, and other infectious fevers; (*c*) peripheral neuritis may supervene in some autotoxic conditions—*e.g.*, gout, diabetes, cancer, oral sepsis, and severe anæmia. And an attack has sometimes followed overexertion or exposure. Traumatic cases come under the head of monoplegias.

Treatment.—The causes must be sought for and removed—*e.g.*, alcohol must be absolutely forbidden, and any gouty or other diathesis must be treated. Rest is the cardinal feature in all treatment, and merely remaining in bed will do much to cure. Iodide of potassium and quinine are undoubtedly of use for the elimination of toxins, whether it be alcohol, lead, gout, or other kind. When the acute and painful stage has subsided, galvanism is very valuable, and its systematic use, with or without faradism and massage, will hasten the restoration of the muscular weakness.

§ 562. II. **Beri-Beri** frequently comes under notice for paraplegia, sometimes for dropsy of the legs. The disease is endemic or epidemic in certain localities. The paraplegic form is now recognised as due to a peripheral neuritis.

The *Symptoms* of beri-beri belong to two classes: (*a*) Those referable to the neuro-muscular system (the paralytic type); and (*b*) those referable to the vaso-motor system (the œdematous type). Either may occur alone, but more often both are present, the paralysis predominating. The onset may be sudden or gradual, with prodromata of languor, pains, and slight dyspnœa.

In (*a*) there is no œdema. The patient may be so thin and emaciated that the condition is called "dry beri-beri." The first symptom is a difficulty in walking, with the characteristic high-stepping gait of foot-drop The knee-jerk is soon

lost, and the muscular paralysis may extend from the legs to the arms, diaphragm, intercostals, and larynx. There is cutaneous anæsthesia, occurring first in the legs, and sometimes not spreading further; at the same time there is great muscular tenderness, especially noticeable in the calves. The sphincters are not affected. The heart is dilated towards the right.

In (*b*) the characteristic symptoms are great dyspnœa and œdema, which is especially seen in the legs. Where dropsy is extreme the condition is called "wet beri-beri," the heart is dilated, especially to the right, with changing bruits from day to day. The digestion may remain unimpaired, and there is no fever.

Diagnosis.—Beri-beri has to be diagnosed from alcoholic and other forms of peripheral neuritis, and here the œdema, and the constant involvement of the heart in beri-beri are important. It may be diagnosed from locomotor ataxy by the absence of the characteristic ocular and other symptoms which occur with that affection.

Prognosis.—In an epidemic the mortality may rise to 50 per cent., but normally only 10 per cent. of the cases end fatally. Death occurs usually from cardiac failure or asphyxia, and is thus very frequently sudden and unexpected. A favourable case runs its course in a few weeks, but usually the disease lingers on for months, especially if the patient continue to live in an endemic area. Serious complications may arise in the form of œdema of the lungs or effusion into the serous cavities.

Causes.—Beri-beri occurs in Japan, China, the Malay Peninsula, parts of America, the West Indies, East Africa, and less often in India. It attacks all ages, but is less frequent in children and old people. Beri-beri occurs in outbreaks year after year in the same place. If it has once occurred in a ship, it is very difficult to disinfect that ship so that the disease will not arise in it again and again. If one case occurs in a crowded prison or town, the disease will rapidly spread over the whole community. Baron Takaki Kanihiro has traced the disease to rice badly cured or infected in some way. If this is so, the infection of the places where rice is stored would quite explain the spread of the disease.

Treatment.—When one case occurs in a crowded school or prison, everyone should be sent away immediately. If a case occurs on board ship, the patient must be brought on deck and kept in the fresh air, and on reaching land the ship must be thoroughly disinfected. When a patient acquires the disease on land, he must be removed immediately to another locality and isolated. As regards diet, all bulky food (and therefore rice) must be avoided. Other treatment is symptomatic, and if symptoms of great venous obstruction occur with intense dyspnœa, bleeding must be at once resorted to.

§ **563. Other Causes of Flaccid Paraplegia.**—III. **Acute Anterior Poliomyelitis** at the onset may take the form of a paraplegia, but it more often affects one of the legs or one of the arms. In the course of a week or two it always settles down into one limb, one segment of a limb, or one set of muscles. It is almost entirely confined to children. It is described under Amyotrophy (§ 601).

IV. **Acute Transverse Myelitis** (§ 559) is at the outset a flaccid paraplegia, but the paralysis is not atrophic excepting in the unusual event of the lesion occupying the cervical or the lumbar enlargement. The paralysed limbs become rigid, and the reflexes return in the course of a few weeks.

V. **Landry's Paralysis** is also a flaccid paralysis (§ 576). It is a rare disease, resembling acute myelitis in some respects, only no gross lesions after death have been found. There is a sudden onset of flaccid paralysis of the legs, followed by rapid extension to the muscles of the trunk and arms, and generally a rapidly fatal termination. A microbe having the form of a tetracoccus has been described in connection with the disease (E. F. Buzzard), but its etiological significance has not yet been established.

VI. **Syringomyelia** and **Intra-medullary Tumours** and **Hæmorrhage** may come under notice as paraplegia with flaccidity, and very often with a contradictory increase in the knee-jerks; but on further examination this will be found to be preceded by the arm symptoms (§ 607).

Group C. **Functional or Variable Paraplegias.**—When an organic lesion is present, whether it be in the upper or lower neuron, we get a continuous, or rather an unvarying, paralysis which progresses steadily for better or worse. But the leading feature of functional paraplegia is (1) that it varies in intensity and sometimes in its other features from day to day. (2) The paralysis is less marked, it is a paresis rather than a paralysis, and it does not clearly conform either to the upper or lower neuron type ; (3) it is accompanied by other evidences of the causal condition.

The principal varieties of variable paraplegia are—

I. Hysterical paraplegia.
II. Railway spine.
III. Paraplegia after fevers.
IV. Diver's paralysis.
V. Reflex paralysis.

§ **564. Hysterical Paraplegia** is undoubtedly the commonest, and may be taken as the type, of functional paraplegias. The nature of the lesion we do not know, and so variable are its clinical features that we have to rely mainly upon the fact of its occurrence in a female who is the subject of other hysterical manifestations. (1) The paraplegia is generally of sudden onset, and not infrequently dates from some hysterical seizure. I once saw a healthy girl coming out of church in front of me drop with the onset of the disorder. It is rarely complete, and varies in intensity from day to day. Sometimes it is rigid, but almost as often flaccid, and the patient drags her feet along in a characteristic way, resembling peripheral neuritis in the presence of foot-drop. (2) The paralysis and the other symptoms are inconsistent and evanescent. Thus, there may be hyperæsthesia in one leg, anæsthesia in the other, and the two may change places or disappear from day to day. All the symptoms may disappear rapidly, though I have observed rare cases of several years' duration.[1] (3) The muscles do not waste nor lose their electrical contractility. The deep reflexes may be unchanged, but I have generally found them exaggerated. The plantar reflex gives a normal flexor response showing the absence of organic disease of the spinal cord (Babinski). (4) Incontinence of urine or fæces is very rare, and bedsores never occur with good nursing. (5) Other evidences of the hysterical diathesis are present.

In the *Diagnosis* it is well to remember that the patient is practically always a female, though male cases have been recorded.[2] In myelitis there are trophic changes, girdle pain, and incontinence—features which are wanting in the functional variety. Other organic paraplegias are identified by their continuous and progressive character, and by the presence of ankle-clonus and Babinski's sign. These latter are regarded as absolutely conclusive of organic disease, but there is a kind of spurious

[1] Trans. Clin. Soc. Lond., vol. xxii., and the *Lancet*, 1901, vol. i.
[2] The *Lancet*, 1889, vol. i. and vol. ii., p. 792.

ankle-clonus in many cases of hysteria which is only distinguished with difficulty from the ankle-clonus of organic disease. The *Causes* and *Treatment* are dealt with elsewhere (§ 524).

§ **565.** II. **Railway Spine** and other **Functional Paraplegias.**—A severe shock or injury to the spine, such as occurs in railway accidents, may cause (1) a direct injury to the cord, such as hæmorrhage, laceration, or concussion ; (2) the shock may determine the occurrence of some degenerative lesion which does not come on perhaps for some months, such as disseminated sclerosis, tabes, or spastic paraplegia ; or (3) a form of painful paraplegia may ensue, not coming on until some days, weeks, or even months after the injury. It is with this last we are now concerned. Some, like Erichsen and Gowers, regard it an incipient myelitis ; others, like Herbert Page, maintain that it is due to a functional change (? vascular). The symptoms consist of (1) severe spinal pain and tenderness (spinal neuralgia) and obscure peripheral sensations, such as tinglings, twitchings, or numbness in the legs. (2) A paresis or pseudo-paralysis, which Page believes may be due to the fear of pain produced by movement. (3) Bladder symptoms are sometimes present, such as frequency of micturition or a dribbling at the end, or a difficulty in defæcation. These cases are very difficult to treat successfully. Rest, with change of scene and occupation, are the main elements.

III. **Paraplegia after Fevers.**—Anæmia and congestion of the cord are said by some to give rise to no symptoms, but by others are said to cause tingling and cramps, especially at night, and a variable degree of weakness of the legs, chiefly on exertion. General anæmia of a severe type has been shown to cause actual sclerosis of the cord (see Toxic Sclerosis). The paraplegia following beri-beri and most fevers is due to peripheral neuritis, sclerosis, or myelitis, but that which is especially liable to occur after jungle fever is held by Indian authorities to be due to anæmia of the cord. All these forms are characterised mainly by (1) the circumstances under which it occurs, (2) the paralysis being incomplete and usually transient.

IV. **Diver's Paralysis** (Synonyms : Caisson Disease, Compressed Air Illness) is a paraplegia which occurs in men who work under water or tunnel under the ground at high atmospheric pressures. It is due undoubtedly not so much to the pressure they are subjected to, as to the sudden release of that pressure, which produces congestion of the cord, with liberation of gas from the blood. In fatal cases hæmorrhage and myelitis have been found. This was well illustrated in a case shown by Dr. Robert Maguire at the Medical Society.[1] The patient had had six or seven previous attacks, and the last was brought on entirely by his sudden rush to the surface from a depth of 150 feet—that is to say, from a pressure of 90 pounds to the square inch to one of 15 pounds to the square inch. The paresis is never quite complete, but is of sudden onset when the patient returns to the normal atmospheric pressure. It usually affects the legs, rarely the arms to any extent. Anæsthesia, severe pain, and sphincter paralysis only occur in the graver cases. Auditory vertigo, hæmorrhage from the nose, lungs, and other parts sometimes occur. The prognosis is favourable in most cases ; pain and paresis pass off in a few days to six weeks. A few cases have died.

Treatment.—Curative measures consist simply of rest and the avoidance of alcohol—a failing to which most of these workmen are addicted. Ergot has been recommended. Preventive treatment consists in following out precautions for gradual decompression. Men employed in diving or in tunnel working under pressure should be compelled to undergo a very gradual process of decompression. Dr. Snell recommends[2] ten minutes' decompression for each atmosphere of pressure.

V. **Reflex Paraplegia** is a variety which some do not admit, but there appears to be certain very rare cases associated with gastro-intestinal, uterine, or other

[1] The *Lancet*, April 14, 1900.

[2] "Monograph on Caisson Disease," by Dr. E. H. Snell. H. K. Lewis, London, 1896.

irritation. It was first described by Trousseau. After operations on the anus there may be weakness of the legs and inability to pass urine for days. The loss of power appears to be never quite complete. The only means of identification consists in the presence of a reflex cause and the disappearance of the paralysis when this is removed. Some cases supposed to be of reflex origin have been found to be due to neuritis ascending to the cord.

§ **566.** The **Prognosis** and **Treatment** of Paraplegia.—*General Remarks on the Prognosis of Paraplegia.*—Paraplegia is always a serious symptom, not only because it prevents locomotion, but because it indicates structural or functional disease of that important structure the spinal cord. In general terms (1) all functional disorders are more favourable than organic; (2) flaccid paraplegias tend to run a quicker course either towards recovery or death than rigid paraplegias; (3) the most unfavourable signs are bedsores, and the implication of the bladder and rectum. The different forms of paraplegia may be grouped for the purposes of prognosis into four groups (see table below). The presence of complications adds to the gravity of any case. Many cases die of cystitis and pyelitis or the chronic septicæmia which results from bedsores; others from pneumonia or other complications. Finally, efficient or inefficient nursing is an extremely important factor in the prognosis, for these cases constitute the test and trial of all that makes for efficiency in nursing.

PROGNOSIS OF THE DIFFERENT FORMS OF PARAPLEGIA.

Those which generally recover completely, either spontaneously or under adequate treatment.	*Those which generally terminate in* PARTIAL *recovery, either spontaneously or under adequate treatment.*	*Those which tend to become chronic and and incurable, but not fatal.*	*Those which tend to a fatal issue.*
Hysterical and the other varieties of functional paraplegia. Peripheral neuritis. Some cases of Pott's disease. Non-malignant extramedullary tumours. Paraplegia after fevers. Toxic sclerosis.	Most cases of Pott's disease. Anterior poliomyelitis.	Dorsal myelitis. Embolism. Some spinal injuries. Chronic spinal meningitis. Hæmorrhage. Primary lateral sclerosis. Ataxic paraplegia. Amyotrophic lateral sclerosis. Infantile cerebral and spinal paraplegia.	Malignant and inaccessible tumours. Acute cervical or lumbar myelitis. Some cases of hæmorrhage. Malformation.

The *Treatment of paraplegia*, excepting in "functional" cases, is not very hopeful, for a seriously damaged portion of the cord can never be fully restored. There are three indications: To remove the cause, to prevent complications, and to restore the functions. (*a*) To remove the cause: (1) Anti-syphilitic treatment, and particularly iodide of potassium, should be employed at once in all possibly syphilitic cases; 60 to 180 grains of the iodide should be given daily. It will probably not restore a damaged segment of cord, though Brown-Séquard advocated its use in what were called cases of atrophic softening. In some chronic cases it certainly seems to promote the absorption of inflammatory products. (2) If the existence of inflammation or congestion of the cord be suspected, belladonna and ergot may be given; experimentally these remedies produce contraction of the vessels of the pia mater. Rest and the prone position, the patient lying on the stomach, are also of advantage. In cases of congestion or myelitis nux vomica, opiates, and galvanism should be avoided. Where hæmorrhage is suspected, prolonged absolute rest in the prone position is necessary. (3) Counter-irritation, I believe, might with advantage be used more often than it is, especially in the upper neuron group—blisters to the spine, frequent cupping, hot douches

(98° to 101° F.), frictions, mustard or ung. hyd. iod. rubbed into the spine. In chronic cases the best treatment, in my experience, is the " coup de fer " of the French—that is to say, dotting the point of a Paquelin thermo-cautery down the spine every other day. Several cases of sclerosis under my care have derived advantage from these methods. (4) Surgery comes to our aid in cases of Pott's disease, injury, malformations, and some tumours. Certain of the causes require special treatment. In *Pott's disease* rest and extension in a prone or supine position afford a good chance of complete recovery (head and arms fixed to the wall, counter-extension by weight). In older people laminectomy may be done at once because the disease tends in them to progress. Various methods of forcibly reducing dislocation are now adopted. Sayre's jacket and various kinds of apparatus are used ; one of the best of these, perhaps, is one designed by Mr. Jackson Clarke.[1] In *acute myelitis* an ice-bag to the spine and the administration of atropine subcutaneously and locally have been advocated, though but little can be done beyond rest and the prevention of bedsores. For *chronic meningitis* (pachymeningitis) and the six upper neuron scleroses (see § 556 to § 560), the iodides or atropine may be tried, but counter-irritation is the best. The treatment of *hysterical paraplegia* is given elsewhere, but the value of faradic and static electricity is undoubted.

(*b*) To prevent complications should be our endeavour in all cases. A catheter carefully *aspecticised* should be passed three or four times in the twenty-four hours in cases of partial or total retention, and with considerable care, for the parts are often anæsthetic. The patient should be placed upon a water bed, the bowels kept gently acting with laxatives or enemata ; and the formation of bedsores prevented by cleanliness, dryness, and the relief of pressure at all prominent points. The question of nursing is of the highest importance in all of these cases, especially in cases of myelitis.

(*c*) The restoration of the function of the muscles may be promoted in due course by the application of galvanism, faradism (particularly the combined current), massage, and passive movements. These means are more useful in flaccid paraplegias, and are not suitable in recent or irritable cases or in the earlier stages of acute myelitis. Nervine tonics, strychnine, phosphorus, arsenic, cod-liver oil, iron, quinine, are all of value to promote nutrition. Bearing in mind the view of modern pathologists, that in cases of sclerosis of the cord the primary lesion is a malnutrition of the nerve-cells and fibres, rest and a liberal dietary, especially one containing abundance of cream and fats, may be of use, and in actual practice I have found it so.

The patient complains of weakness or **paralysis** *of* BOTH ARMS. The case is one of BRACHIPLEGIA (*i.e.*, Brachial Diplegia).

§ **567. Brachiplegia** is paralysis of both arms without paralysis of the legs. It is not a very common condition, and must not be confused with diplegia, which is double hemiplegia, nor with brachial monoplegia. It is met with occasionally in the following diseases. In the *Diagnosis of the cause* you should first ascertain whether you have to do with a *lower* neuron lesion such as I., II., III. ; or *upper* neuron lesion as in VI. and VII.

I. Double *musculo-spiral paralysis* (p. 830) is the commonest cause of brachiplegia, as met with in lead poisoning and sometimes other toxic conditions, such as poisoning by arsenic or silver.

II. In *syringomyelia* (§ 607) wasting and weakness of one or both arms may be the earliest feature. The paralysis is usually flaccid, but may be rigid, and is accompanied by loss of temperature sense and other sensory changes.

[1] See discussion Clin. Soc., the *Lancet*, March, 1900.

III. In a few cases of *acute anterior poliomyelitis* (§ 601) both arms may be paralysed at the same time.

IV. In certain cases of *idiopathic muscular atrophy* (*primitive myopathy*), weakness of the muscles of the arms and shoulder girdle may be the earliest and for a long while, the only symptom.

V. *Injury to the cord* in the cervical region may result in atrophic paralysis of the arms.

VI. In *hypertrophic cervical meningitis* and in *extra-medullary tumour* in the region of the cervical enlargement there is acute pain in the arms, and the " claw hand " usually develops.

VII. **Amyotrophic lateral sclerosis** (Charcot) is a rare disease, due to disease of the anterior horns and also of the lateral columns. In the first stage, lasting usually from four to twelve months, the symptoms are mostly confined to the upper extremities, which are feeble, and gradually *undergo atrophy*. Combined with this is rigidity and increase of the deep reflexes of the arms, which gradually assume a characteristic position owing to the contracture of the muscles. Both arms are usually involved, but not infrequently one side predominates. In the second stage the lower extremities become invaded by the symptoms already described under Primary Lateral Sclerosis. The disease differs from progressive muscular atrophy, of which some regard it as a variety, in the presence of the stiffness both of the arms and of the legs, and also in its relatively rapid course, for death generally takes place in from one to three years. It more closely resembles cervical pachymeningitis, excepting in the absence of pain.

The patient complains of weakness or **paralysis** *in* ONE ARM *or* ONE LEG. The case is one of MONOPLEGIA.

§ **568. Monoplegia** is loss of power in one limb. Monoplegia brachialis is paralysis of one arm ; monoplegia cruralis, of one leg. Its causes are as follows :

a. Single Nerve and Plexus Paralysis (§ 569).

I. Single nerve paralysis.
II. Plexus paralysis.
III. Occupation neuroses.
IV. Arthritic atrophy.

b. Spinal Monoplegias (§ 571).

I. Acute anterior poliomyelitis.
II. Chronic anterior poliomyelitis.
III. Spinal tumours, Pott's disease, and other causes of paraplegia (occasionally).
IV. Syringomyelia.
V. Amyotrophic lateral sclerosis, cervical pachymeningitis and other causes of brachiplegia (occasionally).

c. Cerebral Monoplegias (§ 572).

I. Focal cortical lesions.
II. Hysterical monoplegia.

d. Certain Primitive Myopathies (§ 603).

The chief POINTS TO INVESTIGATE are, first, precisely which of the muscles is affected and the character of the paralysis ; secondly, the electrical reaction ; thirdly, the sensation of the affected part ; and fourthly, the presence of any cause of pressure existing along the course of the nerve trunks, plexuses, or roots. It is often difficult to decide whether an alleged weakness in the forearm is not in reality due to disease in one of the smaller joints. In any case the joints should be examined, because even after slight injury an arthritic amyotrophy (see below) occasionally ensues, which may cause weakness in the limb.

Monoplegia may be due to a lesion situated (*a*) in the peripheral nerves, plexuses, or roots; (*b*) spinal affections involving the anterior horns or anterior roots; or very rarely (*c*) localised cerebral lesions; the first two being paralysis of the lower neuron, the third those of the upper neuron type.

§ **569. Single Nerve Paralysis.**—Peripheral nerve and nerve-plexus lesions give rise to a monoplegia which, like all lower motor neuron lesions, is *flaccid, accompanied by R. D.*, and is followed by *atrophy* of the affected muscles. The key to the detection of the nerve involved consists in the identification of the precise muscles affected. The lesion may be an injury, pressure, or inflammation, but the symptoms are much the same in kind, though they differ somewhat in degree. A divided nerve may be taken as the type.

Symptoms.—Division of a *motor* nerve (or its severe contusion or compression) gives rise at once to (1) flaccid paralysis of the muscles supplied, and alterations in the electrical reactions; (2) abolition of the tendon reflexes in the muscular region involved, followed in the course of the ensuing week by (3) muscular atrophy; and (4) total loss of contractility to faradism. Most nerves are *mixed* and pain and various kinds of paræsthesia are very constant; indeed, pain is the most prominent symptom in acute inflammation or severe injury. Anæsthesia is much less constant. Sensation may remain intact, even after complete section, owing, it is said, to the extensive anastomoses of the nerves. Sensation, when lost, is sooner recovered than motion. Persistent and extensive anæsthesia indicates destruction or section of more than one nerve-trunk or of a whole plexus. Vaso-motor and trophic disorders often result—redness, hyperidrosis, œdema, wasting of the skin (glossy skin), subcutaneous tissue and bones, and vesicles followed by badly healing sores.[1]

The *Diagnosis* may always be accomplished by the electrical reaction of degeneration. Thus are we enabled to distinguish a nerve injury from (1) a direct muscular injury; (2) arthritic amytrophy following a slight contusion of the joint; and (3) spinal monoplegias which are distinguished by the different course they run, and the associated symptoms (§ 571).

The *Prognosis* depends to some extent upon the degree and cause of the injury on compression. Complete R. D. indicates complete severance or destruction of a nerve. But it is wonderful how a nerve will repair with rest, as Mr. John Hilton[2] pointed out long ago.

The *Causes* of peripheral nerve and plexus lesions are manifold, but may be grouped under two headings, as exemplified mainly in the musculo-spiral nerve. The special causes affecting the several nerves will be considered afterwards.

(*a*) *Injury or Pressure.*—Pressure during sleep (especially after alcoholic intoxication), ligatures, the use of crutches (crutch-palsy of the musculo-spiral), luxation of the humerus or other bones, bullet wounds,

[1] See a case reported by the author in *Brain*, part lxiii., 1893.
[2] Hilton's "Rest and Pain," edited by Jacobson; London, Bell and Sons, 1887.

stabs, and direct blows on a nerve or nerve-trunk (*e.g.*, brachial plexus above the clavicle), or fractures, may injure the nerve ; later, callus (recognised as a cause by the pain coming on some time after the injury) may involve or compress a nerve, or the nerve may be included in a cicatrix (internal or superficial). Sudden extension of the arm upwards may lead to severe damage or laceration of the brachial plexus ; damage during parturition may act on these or other nerves and severe muscular action (*e.g.*, contraction of the triceps on the musculo-spiral) may act (as Sir William Gowers has shown) by compression. Diseases of the bones beside a nerve, or through which a nerve passes (*e.g.*, caries or syphilitic disease), or enlarged glands or other tumours may lead to compression, and extensive pleurisy at one apex may have the same effect. A small injury of the thumb may lead to ascending neuritis (Kausch, quoted by Oppenheim).[1] Forced functioning leads to occupation neurosis, which may result in spasm (*q.v.*), paralysis, or tremor.

(*β*) A *Toxic Agent* (as already mentioned in Multiple Neuritis) sometimes acts as a predisposing factor, sometimes as the sole cause ; and the toxic agent often seems to have an unexplained proclivity for certain nerves, as in the case of lead for the musculo-spiral. Alcohol generally produces multiple neuritis, but may act as a contributory factor to slight injury or local compression. Arsenic, and occasionally mercury and silver, have been known to produce musculo-spiral paralysis. Diphtheria and influenza are well-known toxic causes of neuritis, and the other infectious fevers act occasionally. Diabetes and enteric fever have been accompanied by paralysis of the circumflex nerve. Paralysis of the musculo-spiral has been observed in enteric fever, articular rheumatism, and pregnancy ; and rheumatism and gout are believed to operate similarly.

In the *Treatment* of injury or compression of a mixed nerve the first thing is to ascertain and remove the cause. Iodide may be given if periostitis is suspected, or operation for bone disease. Rest, as just mentioned, is of paramount importance. Weak galvanism (6 to 8 m.a.) regularly applied, especially in the form of a limb bath, is of the greatest value, and later this may be combined with faradism and massage. If at the end of many months' perseverance the R. D. is still present, surgery may be summoned to our aid. Surgical treatment followed by the above will sometimes completely restore muscles that have been paralysed and with the R. D. for many years. The distal end of an injured or diseased nerve has been successfully spliced on to another healthy nerve (Purves Stewart and Ballance, Wilfred Harris,[2] Low and others). The treatment of single nerve lesions is also referred to under Peripheral Neuritis (§ 561) and Neuralgia (§ 604).

The Symptoms and Causes of Paralysis of Individual Nerves will now be considered. For sensory symptoms, see Figs. 159 to 162.

[1] "Diseases of the Nervous System." Lippincott, London, 1904.
[2] Report of the Clin. Soc. Lond., the *Lancet*, October, 1904.

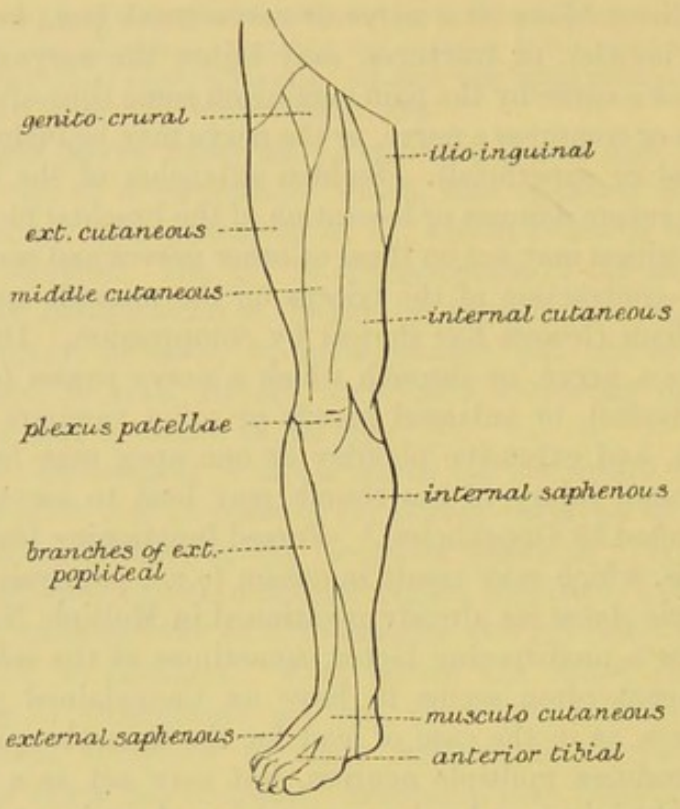

Fig. 159.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

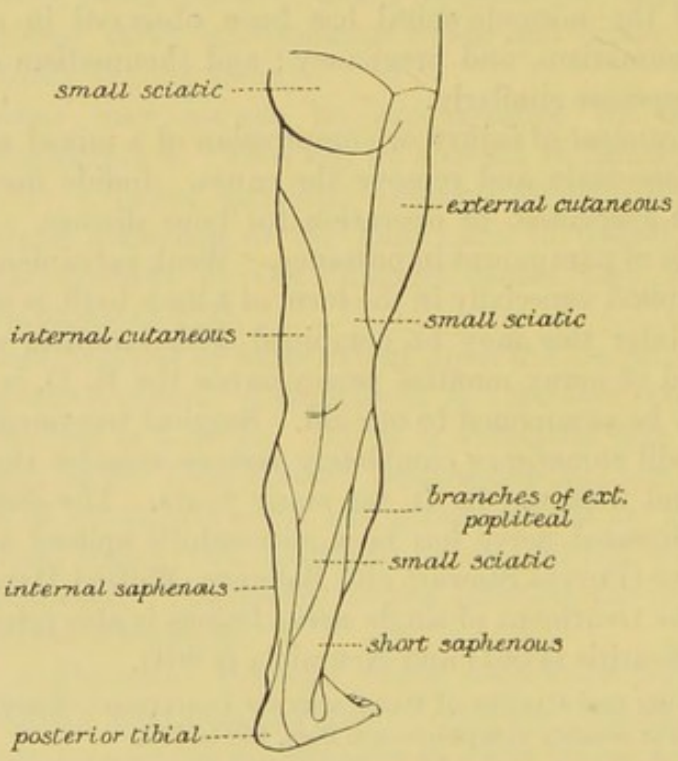

Fig. 160.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

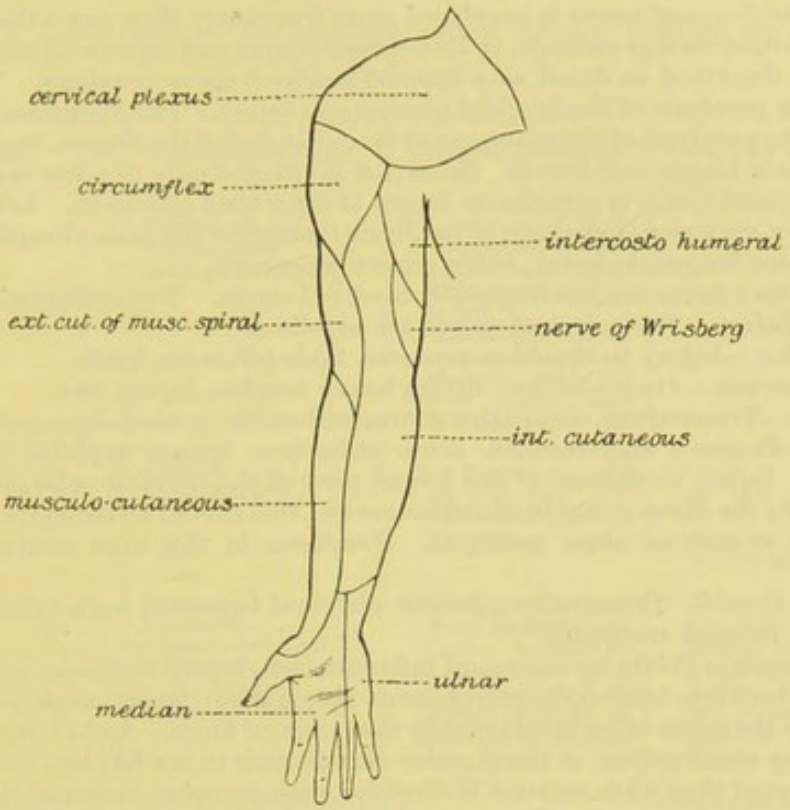

Fig. 161.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

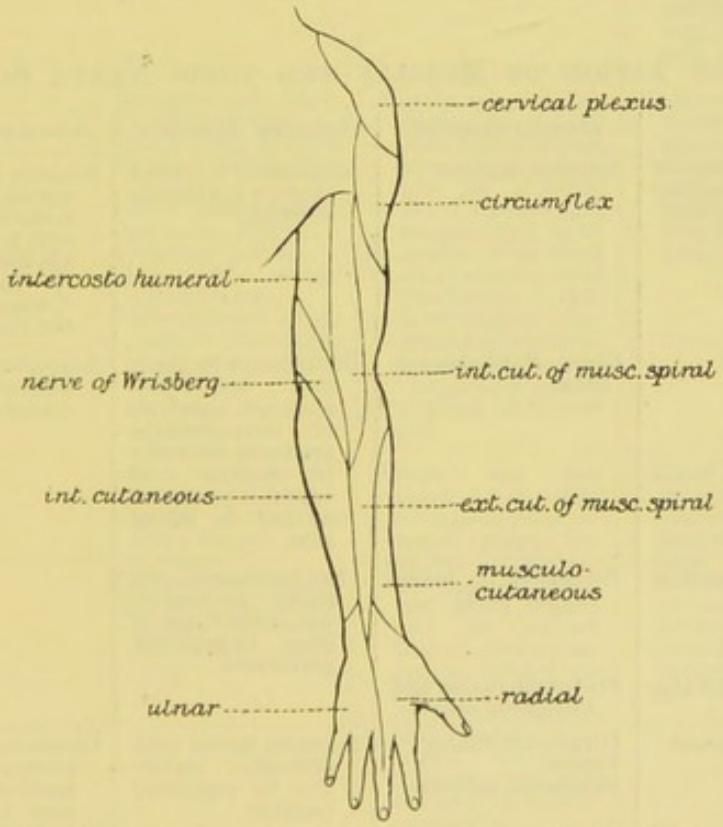

Fig. 162.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

The *musculo-spiral* nerve is paralysed more frequently than any other nerve of the extremities, owing, perhaps, to its peculiar course and superficial position, and it may be described in detail as a type of localised nerve paralysis. It may be involved in paralysis of the brachial plexus (*vide infra*). The *Symptoms* are wrist-drop, due to paralysis of the extensors of the wrist and of the fingers, together with the supinator longus and brevis. Sensation is affected only in a few severe cases. The commonest *Cause* is pressure or injury of some kind (group *a*). Lead poisoning is also a very common cause of paralysis limited to the musculo-spiral nerve; the supinator longus, however, being generally exempt.

The OTHER NERVES are less frequently involved singly. The symptoms are given in a table below. The principal causes are as follows:

Circumflex.—Injury to shoulder, pressure, toxic (diabetes, lead).

Long Thoracic.—Over-exertion, lifting heavy weights, injury, etc.

Median.—Traumatism, occupation neuroses (dentists, joiners, cigar-makers, etc.).

Ulnar.—Pressure and trauma, acute infectious fevers, syphilis, occupation neuroses. Injury or disease of the lowest part of the cervical enlargement may involve only the fibres going to the ulnar nerve; this should be remembered before diagnosing a case as ulnar paralysis. Numbness in this area occurs in tabes dorsalis.

Anterior Crural.—Pressure by pelvic or vertebral tumours; toxic (gout, alcohol, diabetes); femoral aneurysm.

Great Sciatic.—Pelvic tumours and inflammation, injury to femur, compression during parturition, toxic (gonorrhœa, gout, rheumatism, fevers, nephritis). Pressure within the pelvis often involves only the peroneal fibres. An habitually loaded rectum may cause paresis of the muscles of, and pain in the left leg, but it should be remembered that when sciatica is attended with atrophy, cancer of the sigmoid flexure, or rectum, or some other pelvic tumour should be considered.

Phrenic Nerve.—Diphtheria, injury or disease of the third and fourth cervical roots. The symptoms of paralysis of the phrenic nerve are (1) dyspnœa on exertion; (2) during deep inspiration the abdomen does not protrude, owing to paralysis of the diaphragm.

TABLE OF ACTION OF MUSCLES AND THEIR NERVE SUPPLY.

Nerve.	*Muscles Supplied.*	*Defective Movement.*	*Deformity Produced.*
Posterior thoracic or external respiratory nerve of Bell.	Serratus magnus.	Arm cannot be raised above a horizontal position.	Scapula higher than normal. On attempting to stretch arm forward scapula lifts itself with its inner border "wing-like" from the thorax.
Supra-scapular.	Supra- and Infra-spinatus.	Disturbance in function not pronounced, paralysis of infra-spinatus produces difficulty in writing; of supra-spinatus, fatigue in lifting arm.	Scapular spine prominent when infra-spinatus atrophied
External anterior thoracic.	Pectoralis major (upper part)	No movement entirely prevented, but adduction of arm imperfectly performed.	
Internal anterior thoracic.	Pectoralis major and Pectoralis minor.		
Musculo-cutaneous.	Coraco-brachialis. Biceps. Brachialis anticus.	Forearm flexed with difficulty, especially in supinated position.	Characteristic depression on outer surface of upper arm between insertion of deltoid and origin of supinator longus.

Table of Actions of Muscles and their Nerve Supply —*continued.*

Nerve.	*Muscles Supplied.*	*Defective Movement.*	*Deformity Produced.*
Subscapular.	Subscapularis. Teres major. Latissimus dorsi.	In paralysis of latissimus dorsi forcible backward depression of raised arm is lost. In paralysis of teres major elevation of shoulder with the arm against side is lost.	
Circumflex.	Deltoid. Teres minor.	Arm cannot be abducted nor elevated backward or forward.	Change in the shape of the shoulder; relaxation of shoulder-joint develops later.
Musculo-spiral.	Triceps. Anconeus. Supinator longus. Extensor carpi radialis longior. Brachialis anticus.	Elbow, wrist, and basal phalanges of fingers cannot be extended; grip weakened; impaired flexion of forearm if supinator longus is involved.	"**Wrist-drop**," fingers flexed in metacarpo-phalangeal joints; thumb opposed to fingers and somewhat depressed downwards.
Posterior interosseous branch.	All the supinators and extensors of carpus and fingers except supinator longus and extensor carpi radialis longior.		Supinator longus paralysis detected by placing forearm midway between pronation and supination, when flexion against resistance does not bring muscle belly into view.
Median.	Pronator radii teres. Palmaris longus. Flexor carpi radialis. Flexor sublimis digitorum. Flexor longus pollicis. Opponens pollicis. Abductor pollicis. First and second lumbricales. Parts of flexor brevis pollicis and flexor profundus digitorum.	Flexion of hand with slight force and with ulnar deviation. Fingers cannot be properly flexed at first phalangeal joint, while flexion of terminal phalanges only practicable in last three fingers. Pronation of arm lost. Opposition and flexion of terminal phalanx of thumb lost.	Position of hand not markedly altered, generally turned towards ulna and held slightly supinated. Wasting of thenar muscles conspicuous.
Ulnar.	Flexor carpi ulnaris. Adductor pollicis. Muscles of ball of little finger. Interossei. Last two lumbricales. Part of flexor brevis pollicis and flexor profundus digitorum.	Patient can flex hand, but only with adduction towards radius. Inability to flex terminal phalanges of last three fingers and to adduct thumb. Basal phalanges cannot be satisfactorily flexed nor middle and distal phalanges extended. Abduction and adduction of fingers impossible.	"**Claw hand**" most pronounced in fourth and fifth fingers. First phalanx in extreme extension and second and third held firmly flexed; atrophy of hypothenar eminence and of interossei.

TABLE OF ACTIONS OF MUSCLES AND THEIR NERVE SUPPLY —*continued.*

Nerve.	*Muscles Supplied.*	*Defective Movement.*	*Deformity Produced.*
Intercostals.	Intercostals. Rectus abdominis. External oblique. Internal oblique. Transversalis.	In paralysis of abdominal muscles forced expiration interfered with; only possible to rise from a reclining position by using arm as a support.	Lordosis; pelvis strongly bent forward; abdomen and nates show up prominently.
Lumbar.	Erector spinæ. Quadratus lumborum.	The back in walking and standing thrown backward; on sitting spinal column is arched convexly backward.	Slight lordosis which disappears on reclining; pelvis raised.
Anterior crural.	Sartorius. Pectineus. Quadratus femoris.	Inability to extend lower leg. Absence of knee reflex. Paralysis of iliopsoas evidenced by inability to flex hip.	Gait disturbed. Patient stepping carefully, avoiding flexion of knee.
Obturator.	Gracilis. Obturator externus. Adductor longus. Adductor brevis. Adductor magnus.	Adduction and to a slight extent external and internal rotation impaired.	
Inferior gluteal. Superior gluteal.	Gluteus maximus. Gluteus medius. Gluteus minimus. Tensor vaginæ femoris.	Abduction and particularly extension at hip joint hampered. Loss of abduction and circumduction of thigh.	In walking, leg swings too far inwards, also excessive lifting and sinking of pelvis—waddling gait.
Internal popliteal.	Gastrocnemius. Soleus. Tibialis posticus. Fexor communis digitorum. Flexor longus hallucis.	Loss of plantar flexion of foot and toes. Patient unable to lift himself upon tips of his toes. Walking difficult.	Claw position of toe (pied en griffe) pes calcaneus or valgus.
External popliteal.	Tibialis anticus. Extensor proprius hallucis. Extensor longus digitorum. Peronei. Extensor brevis digitorum.	Foot falls from its own weight, and cannot be raised, nor can first phalanx be extended. Walking difficult, toes scrape the floor.	"**Foot-drop.**" Foot remains in equino-varus position.

§ 570. II. **Plexus Paralysis,** paralysis due to a lesion involving a nerve plexus, is another cause of monoplegia. It is recognised by the number and extent of the muscles involved. It is not always possible to distinguish nerve root from nerve trunk involvement.

I. There are three forms of **brachial plexus paralysis :**

(1) *Upper brachial plexus paralysis* of the shoulder and arm (Duchenne-Erb) involves the muscles of the upper arm—namely, deltoid, biceps, brachialis anticus

and supinators, sometimes infra-spinatus and sub-scapularis. It results from tumours or lesions involving the fifth and sixth cervical roots, or by injuries pressing the clavicle against the first rib or spinal column.[1]

(2) *Lower brachial plexus paralysis* of the forearm is much rarer. It involves the muscles of the hand and the flexors of the forearm, sensation being impaired in the region of the ulnar nerve, the inner surface of forearm and upper arm.

(3) *Total brachial plexus paralysis* is very rare, and always traumatic.

Brachial neuritis, a toxic, ill-defined affection of several of the nerves of the arm, or of the whole of the brachial plexus, is described under Pain (§ 604), which is its leading symptom.

Obstetrical paralysis, due to injury during parturition, very often takes the form of upper brachial plexus paralysis ; sometimes the facial nerve is injured by the forceps. Recovery generally takes place in course of time. Other forms of birth palsies are referred to under Hemiplegia.

II. **Lumbar** and **sacral plexus paralysis** is much rarer than brachial, and is due to tumours or disease adjacent to the lumbar or sacral vertebræ. The sigmoid flexure is a frequent primary seat of malignant disease, and may make itself manifest by sacral or sciatic paralysis. The anterior crural nerve (see table) is generally involved.

III. **Occupation neuroses,** such as writer's cramp, telegraphist's palsy, etc., may be attended by paresis, but they more often consist of a muscle spasm, under which they are described (§ 584).

IV. **Arthritic amyotrophy** is described under Amyotrophy (§ 603). It is due to wasting of some of the extensor muscles following contusion or disease of the joint, sometimes of quite a trivial kind.

§ **571. Spinal Monoplegias.**—Affections of the spinal cord may give rise to monoplegia when the lesion involves the anterior horns or anterior roots. We are therefore in presence of a *lower motor neuron paralysis*, which differs from the last group only in the circumstances under which the paralysis occurs, and in the distribution of the muscles involved.

I. **Acute Anterior Poliomyelitis** (Infantile Paralysis) is described under Muscular Atrophy (§ 601). It is the commonest cause of monoplegia in childhood, and though it may affect more than one limb at the outset, 55 per cent. of all cases settle down into a crural monoplegia. In 23 per cent. of the cases one arm is affected.

II. **Chronic Anterior Poliomyelitis** (Progressive Muscular Atrophy) is met with chiefly in adults as a slow progressive amyotrophy starting at the ends of the limbs, generally in the hands, sometimes in one hand.

III. **Spinal Tumours,** or **Pott's Disease,** especially when pressing on the nerve roots, in the region of the brachial or lumbar plexus, may commence with monoplegia, and are recognised by lancinating pains shooting from the back down the limb, the gradual advent and prolonged course of the paralysis, and the concomitant symptoms due to pressure upon the cord (§ 557).

IV. **Syringomyelia** frequently starts as an atrophic paralysis of one, sometimes of both arms, associated with various sensory and trophic symptoms (§ 607).

V. **Amyotrophic Lateral Sclerosis** and **Cervical Pachymeningitis** commence as an atrophic paralysis of one or both hands. They are relatively rare diseases.

§ **572. Cerebral Monoplegia.**—Cerebral lesions generally produce hemiplegia ; only very rarely a monoplegia. The clinical features here are those of an upper

[1] It was this paralysis that Harris and Low successfully treated by cross union of the nerve roots.—*Brit. Med. Journ.*, October 24, 1903.

motor neuron lesion, which would at once distinguish it from the two preceding groups.

I. **Focal cortical lesions** producing monoplegias are comparatively rare. Embolism or thrombosis of the anterior cerebral artery (not a common position) produces a crural monoplegia. Occasionally vascular lesions in the aged involve the arm and face centres only. Tumours limited to one of these centres are practically unknown.[1]

II. **Hysterical monoplegia** (arm or leg) is less rare than organic cerebral monoplegia. Hysterical monoplegia generally dates from an accident or nerve storm or attack of some kind, and it is usually attended by segmental anæsthesia—*i.e.*, the area of loss of sensation is bounded by a circular line drawn round the limb, usually at a joint.[2]

The patient complains of a **widespread paralysis** *or muscular weakness.* The case is one of GENERALISED PARALYSIS (general debility being excluded).

§ **573. Generalised Paralysis** is met with towards the end of quite a number of nervous diseases, but only a few begin with an involvement of all the limbs. We must be careful to exclude the causes of general debility (Chapter XVI.). General paralysis accompanied by stiffness is described under Spasm (§ 582).

Toxic or Functional.

1. Multiple peripheral neuritis due to alcoholism, diphtheria, syphilis, etc.
2. Hysteria.
3. Paralysis agitans.
4. Myasthenia gravis.

Intracranial Lesions.

5. General paralysis of the insane.
6. Diffuse basal tumours.
7. Cerebellar tumours.
8. Cerebral pachymeningitis.
9. Infantile diplegia cerebralis.
10. Encephalitis.

Spinal and Bulbo-Spinal Lesions.

11. Disease or injury high up in the spinal cord.
12. Cervical pachymeningitis.
13. Disseminated sclerosis and other chronic degenerative conditions.
14. Bulbar paralysis.
15. Landry's paralysis.
16. Diffuse myelitis.
17. Acute anterior poliomyelitis.

Amyotrophy.

18. Idiopathic muscular atrophy.

In seeking to DIAGNOSE THE CAUSE of a case of generalised paralysis of this kind one naturally turns *first* to some possible *toxæmic* or *functional* condition such as alcoholism and hysteria.

Secondly, the possibility of some *intracranial disease*, such as a basal cerebral or cerebellar tumour, or general paralysis of the insane would next engage our attention, and we should seek for the associated symptoms referable to the cranial nerves or to the mind.

[1] One case has been recorded by Professor J. M. Charcot and one case by Dr. A. Hughes Bennett.

[2] A very typical case forms the subject of a clinical lecture by the author in the *Clinical Journal*, May, 1904.

Thirdly, we might suspect some *spinal* or *bulbo-spinal* disease when the peripheral symptoms, or symptoms referable to the cranial nerves, would be more prominent than the cerebral or mental symptoms.

Among the Toxic and Functional causes, **chronic alcoholism** is not infrequently followed by a generalised weakness passing on to a generalised paralysis. It is probably due to peripheral neuritis or a generalised toxic degeneration of the nervous system.

Other causes of peripheral neuritis, such as **diphtheria,**[1] may also produce general paralysis. The **syphilitic** toxin may produce peripheral neuritis, but it more often produces General Paralysis of the Insane. In the last stages of **hydrophobia** general paralysis ensues.

In **Hysteria** I have occasionally met with a flaccid paralysis coming on suddenly, and affecting all the limbs.

Paralysis Agitans (§ 590) is from the outset attended by weakness of all the limbs. This gradually increases until the patient becomes bedridden, and all the limbs become stiff and powerless.

§ **574. Myasthenia Gravis** (Asthenic Bulbar Paralysis) is a rare condition, possibly toxic in origin, consisting of progressive weakness of all the cerebro-spinal muscles. It was first described by Willis in "The London Practice of Physic" in 1685, and was studied again by Sir Samuel Wilks in 1877, and Erb in 1878. About sixty cases had been recorded up to 1900.[2]

Symptoms.—1. The patient easily becomes tired, and the muscular weakness is always worse in the evening. All the voluntary muscles of the body are affected, but especially those of the face, eyes, and neck. Bilateral ptosis is present in about 80 per cent. of the cases, and all the ocular muscles are weak (ophthalmoplegia externa). The face is expressionless, and there is often a difficulty of swallowing and of articulation. A nasal speech after talking awhile and a difficulty of mastication after starting to eat are other examples of the rapid fatigue of the muscles. Paroxysmal dyspnœa may supervene. All the symptoms vary in intensity from time to time, but the patient gets gradually weaker. 2. The reaction to faradism is characteristic, for the muscles become exhausted in a very short time. Although the muscles will contract at the moment when the terminal is first applied, they soon become completely flaccid again, and after several applications may fail altogether to respond. This, the myasthenic reaction, is quite peculiar to the disease. 3. There are no fibrillation, no obvious muscular atrophy (till late in the disease), no reaction of degeneration, and no sensory changes.

Diagnosis.—Cases may be mistaken for bulbar paralysis, in which, however, the ocular symptoms are wanting. Diphtheritic paralysis is like myasthenia, but the myasthenic reaction is wanting; so also in neurasthenia, hysteria, and the early phases of chronic degenerative lesions. In Addison's disease there are pigmentation, emaciation, and other symptoms.

The *Prognosis* is grave, the disease is insidious, and may last for several years, but in the end is fatal, either from asphyxia or some intercurrent affection.

The *Etiology* is uncertain. Young persons are mostly affected. The generalised character of the symptoms and the resemblance of myasthenia to diphtheritic paralysis suggest a toxic origin. The thymus is often found to be persistent and enlarged, and small masses of lymphoid tissue may be found in the muscles. Dr. Farquhar Buzzard has described minute lymphorrhages in the muscular tissue as a constant and characteristic lesion.

For the *Treatment* rest, massage, full doses of strychnine, iodide of potassium and mercury may be tried.

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[1] Dr. F. E. Batten (*Brit. Med. Journ.*, November 19, 1898) has shown that very few of the nerves really escape in diphtheritic paralysis.

[2] Dr. Harry Campbell and Dr. Edwin Bramwell, *Brain*, 1901.

Intracranial Lesions mostly produce hemiplegia, but there are three conditions which may produce generalised paralysis.

General Paralysis of the Insane (§ 541), which is a chronic degenerative change in the cerebral cortex, gradually passes on to a generalised paralysis.

Diffuse basal tumours or gummatous meningitis involving the pons or the peduncles may cause generalised paralysis, but in that case the cranial nerves are also involved.

Cerebellar tumours may produce general paralysis, associated with nystagmus and a characteristic gait (§ 577).

Cerebral pachymeningitis produces a vague generalised paresis and mental weakness.

Hæmorrhage or **injury** at birth may cause paralysis of both arms and legs (infantile spastic diplegia).

§ **575. Encephalitis** (Polio-encephalitis) is a morbid condition to which attention has lately been directed by Dr. F. E. Batten and others (Dr. F. E. Batten, "Acute Polio-myelitis and Encephalitis," the *Lancet*, December 20, 1902; and "Polio-encephalitis Inferior," Trans. Path. Soc., London, vol. liv., part iii., 1903), in which miliary foci of thrombosis affect the cortical cells in the same way as anterior polio-myelitis affects the anterior horns. The subjects are mostly children. The symptoms necessarily differ with the position of the mischief, but a generalised weakness is not uncommon, and this may be associated with symptoms of insular sclerosis or ataxy. I have occasionally seen what appears to be a diffuse inflammation of the brain substance (encephalitis). I remember a case of recent syphilis in a man of about thirty-one, who was admitted into the infirmary with symptoms of cerebral irritation—restlessness, muttering delirium, general weakness commencing optic neuritis, who died a few days later comatose. After death the organs of the body and the meninges were healthy, excepting for congestion, but the brain was deeply injected, and its whole substance extremely soft, so that it could be washed away under the tap, and was of a brownish tint. Under the microscope the cortical cells were swollen, blurred, and granular. The cerebral arteries appeared to be quite healthy.

Among spinal and bulbo-spinal lesions **Pott's disease** and **spinal tumours** high up in the cord may involve all four limbs, the mind remaining clear. Injury at birth affecting the upper part of the cord causes **infantile spastic paralysis** (§ 560, xii.), though it is more usually a paraplegia only.

Cervical pachymeningitis, amyotrophic lateral sclerosis, and progressive muscular atrophy may produce a like effect.

Disseminated sclerosis, tabes, and other **degenerative spinal lesions** may terminate in generalised paralysis.

Bulbar paralysis (glosso-labio-laryngeal palsy) may affect all the limbs when it comes on acutely. Chronic cases are only affected by generalised paralysis in their final stages.

§ **576. Landry's Paralysis** is a rare condition of ascending flaccid paralysis, of which the pathological explanation is obscure.

The *Symptoms* start with flaccid paralysis of the legs, rapidly extending to the trunk, arms, and neck, and cranial nerves in some cases. It usually terminates fatally from involvement of the respiratory muscles, in two days to two weeks. There are no (or very slight) sensory changes, no muscular atrophy, no trophic or electrical changes, and no loss of sphincter control. The spleen has been enlarged in some cases.

Etiology.—It occurs chiefly in males between twenty and thirty. It bears considerable resemblance to a rapid peripheral neuritis, and it has been suggested that it is due to an acute toxæmia of the lower motor neuron. A tetracoccus has been found in the spinal fluid by lumbar puncture (Farquhar Buzzard).

Acute diffuse myelitis may rapidly ascend the spinal cord, and cause an acute generalised paralysis. There is anæsthesia and loss of sphincter control, but no muscular atrophy (§ 559).

Acute spinal paralysis of adults is really an **acute anterior poliomyelitis** similar to that affecting children (§ 601). Wasting is rapid, with R. D., and recovery is never complete. A subacute form is also described.

General paralysis associated with extreme wasting of the muscles is seen in the later stages of the **idiopathic muscular atrophies,** and the peroneal type of myopathy (§ 603). The gradual onset in youth, the progressive course of the disease, and the hereditary history aid the diagnosis.

β. Inco-ordination and Disordered Gait.

§ **577.** The **Gait** of all patients suffering from motor defect should be carefully studied ; it will often teach us a great deal about the malady. The defect may be due to muscular weakness, to muscular rigidity, to clonic spasm, to true inco-ordination, or to want of balancing power. The most characteristic disorder of the walk occurs in the *inco-ordination* of tabes dorsalis and the sinuous or *reeling gait* due to want of balancing power in cerebellar lesions. The anatomical remarks on this subject (§ 503) may help to explain what follows.

The various diseases in which disordered gait is met with may be grouped as follows :

a. Inco-ordination (or ataxy) is met with most characteristically in (1) *locomotor ataxy* (tabes dorsalis), in which there is an exaggerated movement, a stamping gait, with the feet wide apart, lifted high, and brought down forcibly—*i.e.*, the patient being unable to co-ordinate his movements. It is met with in other diseases affecting the posterior columns—namely (2) *spinal tumour* affecting the posterior columns, especially in the lumbar region ; (3) *chronic posterior spinal pachymeningitis ;* (4) *Friedreich's hereditary ataxy* (rare) ; (5) *ataxic paraplegia* (rare) ; and (6) other conditions where the *posterior columns* are involved primarily or secondarily.

b. Reeling Gait, or swaying like a drunken man, is met with most typically in (7) *cerebellar disease.* It also occurs in patients who are the subjects of (8) frequent or continuous *vertigo,* either from circulatory or other causes.

c. The Spastic Gait is a stiff gait, owing to the stiffness and rigidity of the legs, due to lateral sclerosis. By degrees the patient takes to walking on tiptoe, the toes turn in, and are scraped along the ground. Later still we get what is called cross-legged progression owing to the predominance in the contracture in the adductor muscles. This may occur on one or both sides. On *both sides* it is met with in the various (9) rigid or *spastic paraplegias* and in ataxic paraplegia. On *one side* it occurs in (10) organic *hemiplegia,* owing to descending sclerosis in which the spastic character of the gait induces a circumduction of the rigid limb as it is brought forward in walking, which, as Sir Benjamin Brodie pointed out many years ago, is in strong contrast with the flail-like flaccidity of many functional hemiplegias. It must be remembered, however, that this distinction only applies to hysterical hemiplegia when the paralysis is flaccid. Some cases are attended with rigidity, and in these the distinction does not obtain.

d. Festination is the gait in which the patient bends forward as he walks and shuffles along the floor, his face looking fixedly forwards. It is met with in (11) *paralysis agitans,* and to some extent in old age.

e. In the High-Stepping Gait the patient raises his knees too high. It is met with typically in well-marked (12) *peripheral neuritis* and other amyotrophic (lower neuron) paralyses attended by *foot-drop,* on account of the predominance of the

paralysis in the extensor muscles, the patient, so to speak, flinging the foot upwards instead of raising it. It is also met with in (13) *pseudo-hypertrophic paralysis.*

f. There is a peculiar "JAUNTY" or dancing walk in (14) *chorea*, associated with excessive arm movement, which is very characteristic. Other tremors (§ 589) may render the walk peculiar.

g. There is a very characteristic ATTITUDE and gait, with the head and arms hanging forward owing to the weakness of the neck muscles, in (15) *post-diphtheritic paralysis* of childhood. It can be recognised as the child walks into the out-patient room.

h. The WADDLING gait is met with in (16) *congenital hip dislocation*, advanced *rickets*, *achondroplasia*, all conditions of *dwarfism* and in *coxa vara*. This last named is a peculiar congenital condition, in which the neck of the femur forms a less obtuse angle than is usual, and causes the patient to sway from side to side as he walks.

i. The LIMPING GAIT is met with as a result of (17) *infantile paralysis* and in any (18) injury or *joint affection* confined to one side.

§ **578. Tabes Dorsalis** (Locomotor Ataxy) may be defined clinically as a very chronic disease commencing with disturbances of the muscle sense and various other derangements of sensation and of the cranial nerves, and terminating in total abolition of the faculty of co-ordinating the voluntary movements; muscular power usually remaining intact until near the end. It is essentially a sensory, sensorial and muscle-sense disorder, syphilitic in origin.[1]

To understand this disease, it is necessary to refer to its *Histo-pathology.* It is interesting to notice that in the course of years our knowledge of tabes has, as it were, shifted clinically backwards, so that what were formerly regarded as the essential symptoms of the disease are now looked upon as complications or sequelæ. At first the disease was classed as a paraplegia; now we know that paralysis of the legs only occurs at the termination, and not always then. Next, it was regarded as an ataxy. Finally, we are coming to regard even this ataxy as an advanced symptom, and at the present day we concentrate our attention on what is called the "pre-ataxic stage," which consists of muscle-sense and other sensitive and sensorial changes. Until recently tabes dorsalis was described as a primary sclerosis of Burdach's columns, chiefly in the lumbar region, combined with a similar process involving more or less of the posterior spinal roots, the spinal ganglia, and sometimes the posterior horns, and giving rise to an ascending sclerosis in Goll's columns, the antero-lateral tracts, and occasionally the cerebellar tracts above. Other parts are also sometimes involved; thus, Charcot mentions that the cells in the anterior horns are affected in cases accompanied by joint disease. This, ever since the days of Todd, was regarded as the essential lesion of the disease, but, unfortunately for this view, there have been at different times cases recorded[2] in which, although the patient revealed all the most typical symptoms of the disease during life, there were no such lesions found after death. During the past few years valuable contributions have been made to our knowledge concerning the structures which are subservient to muscular sense, and about the real lesion in tabes. A little fusiform structure called the muscle spindle has been found by physiologists for half a century in all the muscles of the body excepting the diaphragm, the intrinsic muscles of the tongue, and (?) those of the eyes. Each consists of a laminated sheath of white fibrous tissue, containing a lymph space, in which lie striated fibres resembling muscle fibres, only one-third of their diameter. Nerve fibres in great profusion enter the spindle, and Professor W. S. Sherrington[3] has shown not only that these nerve fibres are

[1] Dr. Ford Robertson regards a diphtheroid bacillus (*b. xerosis*) which he has found in the cerebro-spinal fluid as the cause of Tabes and G. P. I.

[2] *E.g.*, Dr. A. Hughes Bennett, Clin. Soc. Trans., vol. xviii.; Déjérine, and others.

[3] *Journal of Physiology*, 1896-1897.

connected with the posterior roots, but that they convey afferent impulses ; and there is no doubt, as Kerschner and others maintained, that these are really the end organs of the muscle-sense nerves. Dr. F. E. Batten[1] has shown that these muscle spindles are diseased in tabes, the intra-fusal nerve endings and contiguous muscle fibres undergoing degeneration, and that the same changes are absent in primitive myopathies, progressive muscular atrophy, and infantile paralysis. Now, whenever the nutrition of a neuron is impaired, the peripheral endings of its processes are the first to undergo degeneration. Applying this principle to the pathology of tabes in the light of Sherrington's and Batten's researches, the condition of affairs may be depicted in Fig. 163. The cell body of the muscle-sense

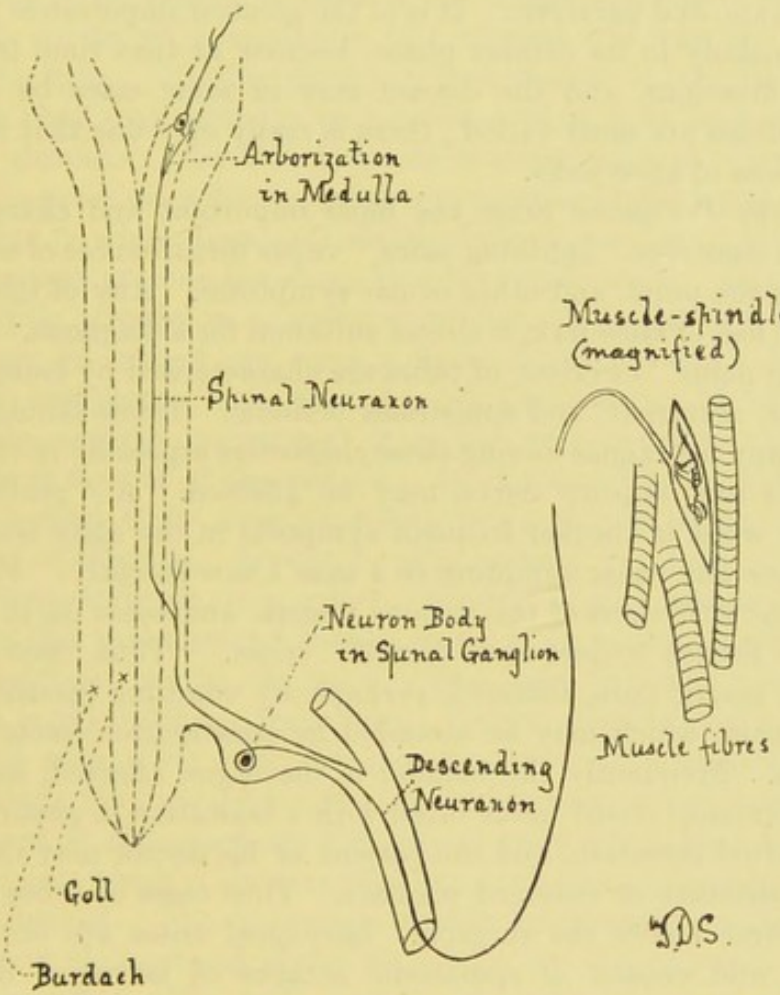

Fig. 163.—Diagram of the spinal cord and muscle-sense neuron to show the PATHOLOGY OF TABES DORSALIS.—The sites of initial degeneration are at the ends of the ascending (spinal) neuraxon—viz., its arborisation in the medulla—and the descending neuraxon — viz., its termination in the muscle-spindle.

neuron is found in the posterior ganglion, and it has an upper or spinal neuraxon, and a lower or peripheral neuraxon ; and in tabes we now know that the degeneration starts at the peripheral end of the latter. We have good reason to believe, though the grounds are not quite so sure, that the same degenerative process attacks the termination of the upper neuraxon, which is in association with the arborisations of the cerebro-bulbar neurons. In view of these valuable researches we can now understand why in some of the cases of tabes no sclerosis of the spinal cord is found, by supposing that such cases died before the degeneration had spread to this position. It also helps to explain why in this essentially spinal affection,

[1] *Brain*, Autumn number, 1898.

which starts with ataxy of the limbs (owing to degeneration of the lower neuraxon), we are liable to have so many cranial manifestations (owing to degeneration of the upper neuraxon). And, finally, it also helps us to explain why, in the long pre-ataxic stage, the chief manifestations are associated with sensory disturbances, because we may fairly assume that the other sense neurons of the lower level are similarly affected by the slow degenerative process. The presence of sclerosis in the spinal cord in such cases is explicable on the modern pathological view of interstitial fibrosis, which is secondary to the nerve degeneration.

The *Symptoms* of tabes run a prolonged course of ten, twenty, or even thirty years, and they may be divided into three stages—pre-ataxic, ataxic, and paralytic. It is of the greatest importance to recognise the malady in its earliest phase, because at that time treatment is more efficacious, and the disease may in some cases be arrested. The symptoms are most varied; there is really only one that is always present—loss of knee-jerks.

(*a*) In the *Pre-ataxic Stage* the most important and characteristic symptoms consist of "lightning pains," vague disturbances of sensation, changes in the pupil, and other ocular symptoms. Any of these, combined with loss of knee-jerk, is almost sufficient for a diagnosis. (1) The "lightning pains," or crises, of tabes are characterised by being erratic, evanescent, recurrent, and sometimes periodic. Tabes should be suspected in any neuralgias having these characters especially in the sciatic nerve, but any sensory nerve may be affected. A "girdle pain" round the waist is another frequent symptom in the early stages—six years before any other symptom in a case I saw recently. Pains also occur in the situations of the various viscera, and come on in attacks, which the French writers have named "crises." Thus, there may be attacks of gastric pain, followed, perhaps, by vomiting (gastric crisis); or rectal pain, which may be attended by constipation (rectal crisis); and so on. Frequently the bladder is the organ affected, sometimes with pain (vesical crisis), more often with a hesitation in passing water, or with actual retention, and the patient or his doctor may think that there is a stricture or enlarged prostate. Thus cases of tabes may frequently come before the surgeon. Laryngeal crises are occasionally observed, and consist of spasmodic attacks of laryngeal dyspnœa, often coming on at night. These may be mistaken for asthmatic attacks, and are very alarming, but the patient recovers in an hour or so. All these pains are alike in being erratic, evanescent, and recurrent. (2) Numerous and remarkable are the many different anæsthetic and paræsthetic symptoms observed in tabes. One of the commonest consists of scattered patches of anæsthesia, especially along the ulnar borders of the hands, and on the soles of the feet, giving rise to a sensation of "treading on cotton wool." Sometimes there is analgesia without anæsthesia of the whole or part of the body. Sometimes there is thermal anæsthesia; sometimes delayed sensation; sometimes misplaced sensation (allocheiria); but the strangest of all, perhaps, is what Duchenne described as the "masque tabetique," in which the

patient has anæsthesia of the nose, lips, or face, which gives him the impression that he has no face. Perversions or abolitions of taste or smell are not uncommon, and diminution or increase of the sexual power. (3) The ocular symptoms are very important, and the most frequent of these is transient diplopia, due, apparently, to a transient weakness of one or other of the ocular muscles. My experience bears out the statement that quite half the cases of tabes at one time or another suffer from diplopia. The pupils present very characteristic signs. The most typical of these is the Argyll-Robertson pupil ; *i.e.*, the pupils act to accommodation (the student will note the two a's), but not to light. Another very common change is inequality of the pupils, or they may both be very contracted or both immobile to all forms of stimulus. Another frequent manifestation is primary optic atrophy, coming on with a greyish appearance of the optic disc, and terminating in white discs with clearly-defined margins. Any of these symptoms may precede the ataxy by ten or more years. In my experience, if optic atrophy is an early symptom, the ataxy is very late in developing ; in one of my cases it never developed at all, though all the other symptoms were present. (4) The absence of knee-jerk is among the earliest symptoms ; it is the only constant and almost invariable symptom of the malady. (5) Various trophic disturbances are now known to be associated with tabes, thanks chiefly to the observations of Charcot. It was he who pointed out that a particular kind of osteo-arthritis, now called " Charcot's joint disease," may be present in this malady, and it may occur quite early, at a time when other symptoms are in abeyance. The joint becomes swollen, but usually it is pale, and free from pain. Indeed, there is such a complete absence of pain, heat, and redness, the three classical symptoms of inflammation, that the joint—the hip for instance—may become quite disorganised or dislocated ere the patient pays much heed to it.[1] In the knee-joint, which is the favourite situation, the swelling is less likely to be overlooked. The occurrence of such an arthritis, combined with the pupillary changes and absent knee-jerk, are alone enough to establish the diagnosis. The nails may be affected with a sort of painless ulceration ; the teeth may be similarly affected and drop out ; or perforating ulcers of the foot may occur. All these lesions have the same slow chronic characters. It will be observed that the pre-ataxic symptoms fall into two categories—central or cranial, and peripheral—and this is now explained by the researches of Sherrington and Batten (Fig. 163). As to their relative value, if the patient be a male, of an age between thirty and fifty, and present symptoms taken from any

[1] In an interesting case narrated by Charcot, the patient who was doing his military service, casually found that he could not march as well as the others, and on examination it was ascertained that the two hips had gone on to dislocation. " Nouvelle Iconographie de la Salpêtrière," tome v.

two of the above groups, I believe one is justified in diagnosing the disease.

(*b*) The *Ataxic Stage* consists of the above symptoms, which gradually become emphasised in the course of years, combined with (1) the characteristic walk described above; and (2) loss of equilibrium on standing. The normal power of the muscles remains, and their nutrition is often remarkably good, but the patient cannot control them. The typical ataxic gait has been described, and is easily recognisable when once seen (§ 577). These patients find a difficulty in starting to walk, but, once started, they may improve as they go on, up to a certain point. They also have great difficulty in turning round, and the late Dr. Hilton Fagge mentioned an amusing incident of a patient who once explained his unpunctual arrival at the hospital by his having started in the wrong direction, and being compelled to continue until he met someone who could help him to turn round and start the other way. The arms are much less affected than the legs—at any rate, until quite late. The patient can write or pick up a pin, but there is often a difficulty in touching the nose when the eyes are closed. The loss of equilibrium on attempting to stand with the eyes shut and the heels together is known as Romberg's sign, and is very characteristic of this disease. The explanation of this sign is given in § 503.

(*c*) The *Terminal Paralytic Stage* is really the stage of *complications*, and it may be many years before this stage sets in. The mind remains clear in most cases until quite the end, and the patient may be able to conduct his business for ten, twenty years or more after his locomotion has become imperfect. By degrees the inco-ordination becomes extreme, and by-and-by paralysis supervenes, generally, though not always, of an atrophic form. Bladder complications are frequent, and various other visceral conditions supervene; death results from these or bedsores, not from the disease. Perhaps the most frequent cause of death is pneumonia. Bulbar paralysis may supervene, but one of the most frequent of the nerve complications of tabes is general paralysis of the insane (paralytic dementia); and in visiting an asylum, it is remarkable to learn what a large number of general paralytics have commenced as cases of tabes. Many varieties and transitional forms are met with in these two diseases among out-patients, and give rise to much difficulty unless you remember this frequent association.

Causes.—(1) *Age.*—Like general paralysis of the insane, tabes is almost entirely confined to adults between the ages of twenty-five and forty, and (2) it is almost entirely confined to the male sex—certainly over 90 per cent.[1] (3) Syphilis can very generally be traced in the

[1] The fact of tabes predominating so markedly in the male sex is interesting in connection with the theory of "forced functioning," which may be briefly stated thus: Prolonged forced functioning of any nerve structure (especially if combined with malnutrition) results, in course of time, in atrophy and overpowering of this structure by the surrounding tissues, and its consequent degeneration. Males use their co-ordinating and muscle-sense apparatus much more than females.

history of cases of tabes, and in spite of the fact that iodides and mercury fail so completely in the treatment of tabes, there is very little doubt that tabes is really a slow parasyphilitic degeneration of the muscle-sense neurons. (4) A history of neuropathic antecedents, and especially alcoholism in the parents, is often present. Professor J. M. Charcot[1] insisted very much on the potency of alcoholism in the father as a cause of this and many other nerve affections. Sometimes a family history of other nervous maladies can be traced. (5) Prolonged bodily fatigue, especially if combined with exposure to cold and malnutrition, act both as predisposing and exciting causes. The disease is frequent in postmen, commercial travellers, and others who lead an active life. This is interesting in connection with the theory of origin in forced functioning just referred to. Among the rarer causes, venereal excess, prolonged grief, anxiety, or mental strain may be mentioned.

The *Diagnosis* of tabes in typical cases—and tabes is one of the few diseases of the nervous system which usually conforms to a type—is not difficult excepting in the very earliest (pre-ataxic) stage. The age, sex, and syphilitic history are very characteristic, and the only disease apt to be confused with it at this time is general paralysis of the insane, which, however, usually presents the mental peculiarities and muscular tremor that are wanting in tabes ; compound cases occur. In the ataxic stage it may have to be differentiated from lumbar tumour, pachymeningitis, ataxic paraplegia, and other diseases which will be mentioned below. Lumbar puncture (p. 925) may aid in diagnosis.

The *Prognosis* of tabes has hitherto been regarded as hopeless—at any rate, for cure—though the disease occasionally lasts throughout a patient's lifetime without materially shortening it. But by degrees, by careful attention to clinical detail, we have learned to recognise it in its earlier stages, and much may certainly be done at that time to arrest or delay the degenerative process. The prognosis rests chiefly on three points—first, upon the rapidity of development of the symptoms ; secondly, on the causes in operation, especially when some of them are removable ; and thirdly (the most important point) the stage at which the disease is recognised.

Treatment.—(*a*) For the *curative treatment* all antisyphilitic treatment is almost useless, though mercurial inunctions and injections may be tried in the early stages ; iodides are certainly useless. The leading principles which I have found most useful are the cessation of function of the deranged structures, a liberal dietary, combined with galvanism. Perfect rest in bed should be enjoined, the patient not being allowed even to stand for an instant. The dietary should contain a large proportion of fatty food ; tonics may be administered to promote the assimilation, especially cod-liver oil, as being, in my opinion, the remedy

[1] " Chaque goutte de liqueur séminale d'un alcoolique contient, en germe, la famille neuropathique tout entière."—" Nouvelle Iconographie de la Salpêtrière."

which is the best "nerve food." Electricity requires great judgment, and should not be applied to cases which are irritable or rapidly advancing. The best way, I have found, is to apply an ascending current to the spinal cord, the negative pole being fixed on one side of the back of the neck, and the positive pole being shifted from place to place down the back of the spine on the other side, resting half to one minute at each place. The strength of the current should be very moderate. Long sittings and strong currents should be avoided, but a sitting should be held every day. The system of exercises elaborated by Dr. Frænkel has for its object the education of the lost power of muscular co-ordination. I have applied this method of treatment in early and suitable cases with marked benefit. Belladonna and ergot may be given in cases where congestion of the cord is suspected from the presence of spinal pain and rapidly developing symptoms. Arsenic, silver, and other metals I believe to be injurious; at least, I have known patients go back under their use. Didymin and cerebrin are two remedies which are still on their trial. Patients should certainly avoid anything like fatigue of mind or body. Sexual excess, exposure to cold, alcohol, and tobacco should be avoided. (*b*) In regard to *Symptomatic Treatment*, the most important symptoms calling for treatment are the lightning pains. Morphia must be forbidden, for with a recurrent pain like this the habit is bound to be developed. On the other hand, the same treatment which cures or modifies the disease also relieves the pains. Phenacetin, antipyrine, hyoscyamus, belladonna, physostigmine, Indian hemp, and other analgesics, combined with rest and warm baths, are often successful.

Whenever the **posterior columns of the spinal cord are affected,** whether primarily or secondarily, an ataxic gait and other symptoms resembling tabes may arise. Severe anæmias and other toxic debilitating conditions may be complicated, as Dr. James Taylor and others have shown, by posterior sclerosis and ataxic gait. In alcoholic subjects there is at first a more or less transient ataxy, which later on, as posterior sclerosis ensues, becomes permanent. There are four named DISEASES OF THE SPINAL CORD, all of which, compared with tabes dorsalis, are relatively rare, but give rise to inco-ordination of the gait resembling tabes—viz., spinal pachymeningitis, spinal tumour involving the posterior columns, Friedreich's disease, and ataxic paraplegia.

§ **579. Spinal Pachymeningitis,** or chronic thickening of the meninges, is apt sometimes to be confined to, or at any rate to predominate in, the posterior region of the theca vertebralis, and in some cases I have seen gave rise to disordered gait and other symptoms which it was impossible to distinguish from those of tabes dorsalis. I believe that many cases of so-called tabes cured by iodide were possibly instances of this affection of syphilitic origin (cp. § 560). One is assisted in recognising this condition if there is the long history of *continuous* pain and tenderness in the spinal column, and continuous pain shooting down the spinal nerves. These considerations have led me to describe as a separate condition—

Posterior Spinal Pachymeningitis.—It is extremely chronic, and not infrequently the symptoms are few and ill marked, on which account such cases very rarely find their way into hospitals. They get into the infirmaries, however, and my experience at the Paddington Infirmary led me to the conclusion that the disease is fairly common. At a meeting of the Neurological Society of London held at

the Paddington Infirmary in 1890, I was able to show the cords of five fatal cases and three examples of the affection during life. Here is a fairly typical case : A woman, æt. seventy-two, was admitted into the infirmary in January, 1890, with all the classical symptoms of tabes preceded and accompanied by very severe lightning pains. The history, briefly, was that for three years prior to admission she had suffered from paroxysmal pain shooting down the legs and various other places, and that she had had other attacks which resembled gastric crises. For two years prior to admission she had suffered from ataxy which became progressively worse, and at last so marked that she was unable to stand. Many careful observers saw the case ; all had no doubt that it was an example of locomotor ataxy occurring in a woman. She died of pneumonia in June, 1891, and at the autopsy, though the pia mater and arachnoid on the anterior surface of the cord was normal, that on the posterior surface was thickened and opaque. This thickening was irregularly distributed from end to end, and was much more marked in certain patches of 3 inches in length in the mid-dorsal and in the lumbar regions, in which positions it was about the thickness of a piece of washleather, and showed commencing calcareous plates. These patches were firmly adherent to the posterior aspect of the cord, and the posterior columns beneath were sclerosed as in cases of advanced tabes.

Spinal Tumour (intra- or extra-rachidian) pressing on the back of the cord, especially in the *lumbar region*, is often attended by ataxic symptoms closely resembling those of tabes. It may be distinguished from tabes (i.) by the absence of the pre-ataxic symptoms above mentioned ; (ii.) by the fact that it is preceded by severe, constant, neuralgic pains in the spine, spreading upwards from the lumbar enlargement ; (iii.) as in other cases of spinal tumour, all the symptoms—which, be it noted, include actual weakness and often stiffness of the legs—have a tendency to be unilateral—*i.e.*, predominate in one or other leg.

§ **580. Friedreich's Disease** (Hereditary Ataxia), is a rare hereditary condition occurring in children, often sisters and brothers of the same family. These cases have five differential characters. (i.) The ataxy is often most marked in the arms. There is inability to stand with the eyes shut, sometimes loss of patellar reflexes, and other symptoms characteristic of tabes. The disease, in short, presents a mixture of the symptoms of disseminated sclerosis and tabes ; and pathologically it has been shown to be a postero-lateral sclerosis. The knee-jerks progressively diminish as the disease advances. There are, however, no lightning pains, no crises, and no Argyll-Robertson pupil. (ii.) Tremors of an ataxic nature come on later in the arms, so that the hand in approaching the mouth does not reach its goal, and is accompanied by jerky, irregular movements of the head and neck. (iii.) The speech is impaired in the same manner as in disseminated sclerosis. (iv.) Nystagmus is present in most cases. (v.) There is usually no mental change. The muscles become weakened, and deformities such as scoliosis and talipes ensue.

Ataxic Paraplegia is another rare affection, which corresponds, in my belief, to the spasmodic tabes dorsalis of some authors. It is due to sclerosis affecting the posterior as well as the lateral columns, and it results in symptoms of the two lesions. It is differentiated from tabes by the spastic rigidity, increased knee-jerks, and ankle-clonus. The Argyll-Robertson pupil and lightning pains are absent. As time goes on, the rigidity becomes more marked.

§ **581.** In **Cerebellar Lesions** the gait is sometimes ataxic but more often staggering. These lesions may be obscure, but they may be suspected in presence of a group of four symptoms—viz., a reeling *gait*, *headache*, *vertigo*, and *nystagmus*—especially when those symptoms occur in childhood. The walk is fairly characteristic, having a reeling character, and the patient tends to sway from side of side like one intoxicated. But sometimes, especially when one lateral lobe is affected, the patient tends to fall over towards the side of the lesion.

There is frequently weakness, without actual paralysis, sometimes hemiplegic, but more frequently paraplegic or general in its distribution. The knee-jerk does not help us, for it may be absent, increased, or normal. The cranial nerves mostly affected are the fifth, sixth, and the optic nerve. A symptom occasionally present is forced muscular movements of the neck, pulling down the head towards the side on which the lesion is situated, in tumour of the lateral lobe. Other occasional symptoms are attacks of syncope, palpitation, and dyspnœa, which are of considerable gravity, as they indicate pressure on, or spread to, the medulla, though in one instance of this kind the patient (on whom I made the autopsy) lived for one year and ten months after the onset of these symptoms. Tenderness or (later) bulging may be detected in the affected region.

The *Prognosis* and *Treatment* of cerebellar tumour is that of other intracranial tumours. Its deep-seated situation and the proximity of the medulla render it one of the most serious of intracranial tumours. In a few cases the tumour has been successfully removed.

(γ) Increased Muscular Action.

Involuntarily increased muscular action may be—

Continuous, when it is known as rigidity or *tonic spasm* . § 582

Intermittent, when it is known as *tremor* if the movements are small and vibratile, and *clonic spasm* if the movements are large . § 589

Attacks of violent muscular movements, associated with more or less disturbance of consciousness, are known as *convulsions* . § 597

The patient presents a continuous stiffness or **rigidity** *in the affected muscles*—Tonic Spasm.

§ **582. Tonic Muscular Spasm** or rigidity is indicative of an irritative lesion, functional or organic in some part of the motor tract, or a morbid irritation in the muscular substance. It occurs in the following conditions :

I. Paralytic rigidity (early and late).
II. Hysterical rigidity.
III. Occupation cramp.
IV. Cramp.
V. Tetanus.
VI. Tetany.
VII. Hydrophobia.
VIII. Thomsen's disease.
IX. Arthritic rigidity.

The first of these causes is of organic origin ; the second, third, and fourth are functional ; the fifth, sixth, and seventh are toxic ; the eighth is due probably to some hereditary defect ; and the ninth is probably reflex.

§ **583. Paralytic Rigidities of Organic Origin** are associated with paralysis, and are of three kinds, as exemplified in hemiplegia.

1. *Early Rigidity* of the muscles is that which comes on with the paralysis in cases of hemiplegia due to hæmorrhage, or within the

next few days in association with irritative lesions such as pressure on the anterior spinal roots. The rigidity of hæmorrhage comes on suddenly, and passes off gradually in the course of a week or so. The rigidity of other irritative lesions only passes off when the irritation is removed or the nerve-tracts destroyed.

2. *Late or Spastic Rigidity* of the muscles is that which comes on gradually in the course of a month or so in all cases of paralysis due to lesions of the upper motor neuron. It is met with typically in hemiplegia, spastic paraplegia, and all lesions followed by *descending lateral sclerosis* in the spinal cord. It is always associated with increased knee-jerks, ankle-clonus, and Babinski's sign ; tremors or clonic muscular spasms or athetosis may also be present. It comes on gradually, and increases progressively. It diminishes during sleep. Chloral hydrate relieves this kind of rigidity, which is sometimes very troublesome and painful in hemiplegic cases.

3. *Organic Contracture* ensues in all paralysed muscles in the course of years, whether of the upper of lower neuron type (such as hemiplegia and infantile paralysis respectively). It is due to an atrophic fibrosis of the substance of the paralysed muscles.

Hysterical Rigidity may easily be confounded with 1 and 2 above, unless the history is accessible. It generally comes on quite suddenly after an emotional storm. It is often of limited extent, involving perhaps only the wrist-joint. It may pass off as suddenly as it came. It rarely lasts long, but may occasionally persist for years. This rigidity is said to persist during sleep. The patient, who is a female, presents other signs of hysteria.

§ **584. Occupation Cramp** and other **Occupation Neuroses.**—We have already seen that exhaustion produces muscle-cramp. The continual overuse of a certain group of muscles is apt to produce five symptoms, at first localised to that part, but tending ultimately to spread to other muscles, if the cause continue in operation. In order of frequency, they are (1) tonic spasm, (2) paresis, (3) pain, (4) tremor, and (5) in some cases either atrophy or hypertrophy follows.

Writer's Cramp is the most frequent example, and may be taken as a type, but what follows will apply almost equally to other occupation neuroses. (i.) *Tonic Spasm.*—In scriveners' palsy, after writing for some time, the fingers get so stiff that the patient cannot write. Sometimes the spasm is very painful, and occasionally there are twitchings. The character of the writing alters, and in the course of weeks or months the slightest attempt at writing produces a tonic spasm in the muscles used for that purpose. For a long time *tonic spasm appears only on attempting to write*, the part in the intervals being quite free from symptoms, but in the later stages the spasm may become persistent or start spontaneously. In the later stages also the spasm is apt to spread from the hand to the arm, shoulder, neck, and other parts. In the

early stage the general uses of the limb are unimpaired, and a patient affected with writer's cramp may paint, or play the flute, but in most cases other delicate manipulative procedures cannot be performed with the same precision as formerly. (ii.) The *Power* of the grasp as tested by the dynamometer, is said to be normal, but this is not a delicate test of the small muscles in writing. By careful examination, I have very rarely failed to detect some loss of power. Poore[1] also insists that definite slight weakness of certain muscles of the hand is not uncommon. (iii.) Some discomfort is always experienced and this sometimes amounts to actual pain. This pain, moreover, has a great tendency to spread. In some cases the pain is the most pronounced feature of the case, and is accompanied by tenderness of the nerve-trunks, and tender points elsewhere (occupation neuralgia). "Pins and needles" and other subjective symptoms are by no means uncommon, but I am not aware that anæsthesia is ever observed. (iv.) Atrophy is said to be rare, but a careful comparison of the interossei and other muscles of both hands in several cases has convinced me that it does sometimes occur. Hypertrophy is much more frequent; it usually follows cases in which spasm is the leading feature. (v.) Electric irritability is slightly increased in the earlier stages, and slightly diminished in the latter to both faradism and galvanism, in the muscles and the nerves (Gowers). (vi.) Twitchings and clonic spasms are only occasionally observed, but tremor and unsteadiness are common.

Course and Prognosis.—The advent is very gradual, and the course prolonged over many months or years depending on the continuance of the cause. The prognosis depends on (i.) the duration of the affection and stage when the treatment was commenced; (ii.) the means of the patient to cease the occupation; and (iii.) his intelligence in grasping ing the principles of treatment.

The *Diagnosis*, by reason of its association with the occupation, is not usually difficult, but there are several organic and functional disorders which may be mistaken for writer's cramp at their outset. Brachial neuritis (*q.v.*), lead palsy, and even disseminated sclerosis may be mistaken for it, but in these careful inquiry shows that even from the first the symptoms were not solely determined by the act of writing.

Treatment.—The disease is more frequent in nervous, sensitive persons especially when suffering from a general "lowered tone," or malnutrition, anxiety, or grief. There is also a distinct tendency in persons of a neurotic family history. The fact that there is but one determining cause should render the treatment comparatively simple, but, as a matter of fact, it is very difficult because the malady is chiefly found in those whose daily bread depends upon the performance of a certain

[1] Vivian Poore, The *Practitioner*, 1878, and Med. Chi. Trans., vol. 61.

muscular movement. Careful re-education can, however, accomplish a great deal, and particularly the adoption of a freer and larger style of writing, *by holding the pen more loosely and in a different way.* The patient should learn to hold the pen between the first and second fingers with the *back* of the hand against the paper, and practise writing a series of large sloping M's. A certain amount of rest—at any rate, at the outset—is indispensable. Meantime the patient should begin learning to write with his left hand. Sedatives are of service, especially in neuralgic forms, such as chloral, bromide, small doses of morphia, physostigma, Indian hemp, belladonna, or atropine ($\frac{1}{100}$ grain hypodermically two or three times a week). Nervine tonics are recommended, and even strychnine, but I have often observed this remedy do more harm than good. Faradism also does harm, but voltaic electricity has often seemed to me particularly beneficial, especially when combined with massage. Gymnastic exercises are strongly advocated by some. One remedy—cod-liver oil—has rendered great service, and some of the most successful cases I have seen, have been treated by a combined method of moderating the amount and improving the style of the writing, and the administration of small doses of bromide and cod-liver oil. In many cases dyspepsia or a rheumatic or gouty taint is present, which should be met by appropriate measures.

Other Occupation Neuroses resembling the foregoing are found among telegraphists, drapers (in using scissors), cigarette rollers, violin-players, piano-players, typewriters—any occupation, in short, which necessitates the constant repetition of one particular movement.

Cramp is a tonic muscular spasm occurring in one or more muscles of a limb. It is a troublesome symptom of not infrequent occurrence in persons apparently in good health. It is most apt to come on at night. A muscular cramp of great severity may seize one or all the limbs, and even the respiratory and trunk muscles of a swimmer, and prove very deadly unless aid is close at hand. It is apt to come on in those whose muscles are exhausted, but a low temperature of the water plays some part. Some persons seem prone to be affected by cramp throughout life on slight causes, such as lying with a limb in a strained position. Others only suffer from it when their digestion or general health is out of order. The best remedy is to get out of bed and gently move and rub the limb. Bromide and chloral hydrate will generally prevent its occurrence, and any lithæmia, gouty or rheumatic tendencies should be corrected. Cramp is a frequent premonitory symptom in peripheral neuritis and phlebitis. It is also met with in subjects of Bright's disease and gout, and cramp of the legs is a painful symptom in the first stage of cholera.

§ 585. **Tetanus** is a severe disease characterised by paroxysms of tonic and sometimes clonic spasms, due to the inoculation into a scratch or wound of a specific microbe whose chief habitat is in the earth.

Symptoms.—(1) Within a few days after the injury the patient complains of stiffness of the jaw and back of the neck. (2) Very soon these muscles become rigid. The condition of the jaw is known as trismus, or lock-jaw, in which the jaws cannot be separated. This *tonic* rigidity affects all the muscles of the trunk, and in a less degree of the extremities. The back is rigid, sometimes arched in the position of *opisthotonos*, in which only the head and buttocks rest on the bed.

Or there may be flexion to one side—*pleurosthotonos*, or bending forward of the body—*emprosthotonos*. The angles of the mouth are drawn down and the eyebrows are elevated—*risus sardonicus*. (3) Clonic spasms supervene from time to time, in which the already rigid muscles become more contracted, with agonising pain. The slightest touch may cause clonic spasms. In severe cases these spasms become more frequent, leading to death from involvement of the glottis or respiratory muscles. (4) The temperature may be normal or slightly raised throughout, and may rise to 108° F. just before death. There is often retention of urine. The mind is clear till quite the end. A *variety* due to head wounds is described, with paralysis of the facial muscles and difficulty in swallowing.

Diagnosis.—In hydrophobia the spasm is at first clonic. It affects chiefly the muscles of respiration and deglutition, and there is more mental agitation than in tetanus. In strychnia poisoning in the intervals between the spasms the muscles relax, and the spasms involve the extremities to a greater degree. In spinal meningitis there is a temperature, and there is no trismus. Tetany does not reremble tetanus. In hysterical opisthotonos there are other evidences of hysteria. Trismus is caused also by disease of the pons and acute bulbar paralysis, and in association with periostitis of the jaw, disease of the temporo-maxillary joint, or other local irritation; but the course of the disease serves to differentiate these from tetanus.

Prognosis.—Death occurs in acute cases in one to twelve days from exhaustion or involvement of the glottis or respiratory muscles. In more chronic cases the spasms become less frequent, and recovery ensues in two or more months. The mortality is very high—about 90 per cent.

Etiology.—Tetanus is caused by a wound, however trivial, into which the tetanus bacillus has entered. The bacillus has its habitat in the earth.[1] Tetanus of the new-born is due to want of aseptic precautions in treating the navel.

Local *Treatment* of the wound is necessary by cautery or antisepsis. For the spasms, chloroform, bromides, chloral hydrate, and other drugs are given, and recoveries have been reported after their use. The tetanus antitoxin has given good results in the subacute cases. For acute cases, unless given very promptly it is not so useful. Roux has shown that it is best introduced through a trephine hole in the skull; but it may also be given by lumbar puncture (p. 925).

§ **586. Tetany** is a muscular stiffness occurring in paroxysms, affecting mainly the ends of the four extremities.

The *Symptoms* come on mostly in infancy in the form of a muscular spasm, or, to be more correct, a paroxysmal stiffness affecting both the forearms, hands, and feet. The attitude of the fingers compressed into a cone (the accoucheur's hand) has been emphasised, but it is unessential; it is the attitude assumed by athetosis and many other tonic spasms. The paroxysms last from a few seconds to an hour or so, and in severe cases there is no intervening relaxation. In severe cases, moreover, all the muscles of the body are affected, and there may even be opisthotonos. There is a good deal of neuro-muscular irritability to compression and to both forms of electricity, and if the nail be drawn down the face, a wave of muscular contractility follows it. Many degrees of severity are seen, and the disease may only last two days or two or more months, recovery being the general but not invariable rule. The *Diagnosis* is not difficult. In tetanus the spasm mainly affects the jaw, and there is a history of injury.

Etiology.—Tetany is most frequent in children the subject of rickets or diarrhœa, or other gastro-intestinal disturbance, but it is not solely confined to childhood. In adults it is particularly associated with dilatation of the stomach[2] or any other gastro-intestinal condition attended with fermentation. Cases have been noted in association with pregnancy, albuminuria, and after removal of the thyroid. The disease is almost certainly due to a muscular toxin.

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[1] The *Lancet*, November 24, 1888.

[2] For a carefully studied fatal case of tetany following dilatation of the stomach, see J. S. McKendrick, the *Lancet*, September 24, 1898.

The *Treatment* should be directed to the causal condition. Bromide, chloral hydrate, and immersion in cold water (unlike Thomsen's disease) will relieve the spasm.

§ 587. **Hydrophobia** is a contagious disease characterised by spasms of the muscles of deglutition and respiration, and due to inoculation by the saliva of an animal suffering from rabies.

Symptoms.—(1) After an incubation stage, during which the patient presents no symptoms, which is generally about six weeks, never less than twelve days, and may even last as long as twelve to eighteen months or more, there is an insidious onset of malaise, with perhaps slight fever, and sometimes tingling in the wound. (2) With or without premonitory symptoms paroxysms of painful spasms of the pharynx supervene, coming on at first with a slight stiffness, and brought on by any attempt to swallow. (3) These spasms, at first clonic, become tonic, lasting a quarter to half an hour at a time, and spread to the muscles of respiration and of the neck. The attacks produce excruciating pain and agony of mind. The mind is quite clear, but in the intervals there are prostration and general hyperæsthesia. (4) Paralysis ensues in three or four days' time, first of the muscles of the lower jaw, and death follows within a week from the onset.

Treatment.—To destroy the virus at the seat of entrance suction immediately after the bite is heroic, but efficacious. Cauterising the wound may be employed. The immunisation treatment of Pasteur is dealt with in § 386. Narcotics, chloroform inhalation, and chloral may be employed.

§ 588. **Thomsen's Disease** (Congenital Myotonia) is a rare and obscure condition of universal muscular stiffness of indefinite duration, aggravated by rest. Tension and stiffness of the limbs are experienced on first attempting to rise after resting, but they relax to some extent after continued movement. The muscles of the lower extremities are affected most, but those of the face, tongue, and eyes may be similarly involved. The stiffness is increased by cold. Sometimes there appears to be slight hypertrophy, never atrophy. The galvanic reactions vary, but Faradism is never lost, and this leads to the idea that the disease is in the muscles, not in the nerves. The malady is noticed for the first time in youth, and is very probably congenital. Several members of a family may be affected. The disease does not appear to shorten life. Warmth and continued activity relax the spasm to some extent.

Arthritic rigidity is known by its being associated with some joint lesion, though the latter may be very slight. It affects both the extensors and flexors of the joint, though chiefly the flexors. I have usually noticed that it is increased during sleep.

There are **irregular movements** *or shaking of the affected muscles, the range of the movements being either small* (Tremor), *or large* (Clonic Spasm).

§ **589. Tremors** and **Clonic Muscular Spasms** form a very frequent and pronounced symptom in many different nerve lesions. For clinical purposes abnormal muscular movements may be divided into tremors or movements of small size, and clonic spasms or movements of larger range; paralysis agitans may be regarded as a type of the tremors, chorea as a type of the clonic spasms. The symptoms we are now considering must not be confused with generalised convulsions or fits (§ 597), nor with the uncertain movements of paralysed limbs or tabes dorsalis.

Classification.—It will be convenient to consider first the diseases in which tremors occur (such as paralysis agitans), and later the causes of clonic spasms (such as chorea). But this division must not be taken

too absolutely for those which are commonly small are occasionally apt to be large, and *vice versa*.

Tremors.	Clonic Spasms.
Partial Degenerations.	*Generalised.*
I. Paralysis agitans.	I. Chorea.
II. Senile tremor.	II. Hysterical spasms.
III. Disseminated sclerosis.	III. Myoclonus multiplex.
Functional.	*Localised.*
IV. Hysterical trembling.	IV. Habit spasm.
Toxæmic.	V. Facial spasm.
V. General paralysis of the insane.	VI. Spasmodic torticollis.
VI. Alcoholic tremor.	VII. Other localised spasms.
VII. Metallic tremor.	*Organic.*
VIII. Other toxic conditions.	VIII. Post-paralytic clonic spasms.
IX. Nervous or neurasthenic tremor, and Graves' disease.	
Organic.	
X. Organic diseases in which tremor is not always a prominent feature : (1) Post-paralytic tremor. (2) Amyotrophic paralysis. (3) Lateral sclerosis, Friedreich's disease, and other degenerative conditions.	

Clinical Investigation.—The more important points to be observed about cases of tremor or clonic spasm (in addition to their size or range, as just mentioned) are : 1. Their *distribution*, which may be localised to one limb, as in hysteria, or generalised, as in alcoholic tremors.

2. The *rhythm* or regularity of the tremor should be noted ; for example, it is regular in paralysis agitans, senile tremor, disseminated sclerosis, Graves' disease, small hysterical tremors, alcoholic and metallic tremors ; whereas it is irregular in the various forms of chorea and in some functional conditions.

3. It should always be ascertained whether the tremor is present when the limb is *resting* on the bed or table, or only when the muscles are in action. Some kinds of tremor come on only during muscular action, as in disseminated sclerosis. This is known as *intention* tremor. Other kinds of tremor, such as paralysis agitans, persist during muscular rest—*e.g.*, when the limb is laid on a table. It should, however, be borne in mind that all kinds of tremor tend to be increased when the muscles are thrown into action, or when the patient's attention is directed to them, or during emotional states.

4. The *mode of advent* does not afford much valuable information. It is apt to be sudden in all forms of hysterical and functional spasm, but in most of the remainder it is gradual.

5. The *age* of the patient often gives us an important clue, for among the various causes paralysis agitans and senile tremor are uniformly found among the aged ; whereas alcoholic tremor, metallic tremor, and paralytic dementia invariably affect persons in the middle decades of life ; and, finally, hysterical tremor, chorea, disseminated sclerosis, and habit spasm are mostly met with in the comparatively young.

6. *Causation and Pathology.*—All tremors and clonic spasms may arise under one of three pathological conditions : (1) Some organic lesion of the nervous system,

such as disseminated sclerosis ; (2) idiopathic or toxæmic causes, such as general debility, anæmia, alcohol, hysteria, syphilis, rheumatism, or other toxic conditions ; or (3) reflex irritation, such as may be seen in the case of facial spasm or habit spasm, when the tremor may disappear on the removal of a diseased tooth or correction of the refraction of the eye, or, as in a case I have seen, remedy of disease of the pharynx.

A study of the organic cases throws much light on the pathology of tremors and clonic spasm, for we find that they are always due either to partial destruction or imperfect recovery of some part of the motor tract. The greater frequency of post-hemiplegic clonic spasms (1) after embolic lesions, which are less destructive than hæmorrhage ; (2) after hemiplegia associated with hemianæsthesia which indicates a lesion far back in the internal capsule ; and (3) after the hemiplegia of children, in whom the recuperative power is so much greater—all point to this conclusion. Turning to other diseases, we see that insular or disseminated sclerosis is attended by an incomplete destruction of the motor tract. Paralysis agitans, again, is evidently one of the senile degenerations, which is very gradual, and therefore for a long while incomplete. Later on in this disease, when the motor fibres are more completely destroyed, the tremors cease, and paralysis, with tonic rigidity, ensues. Finally, it may be seen what a very large proportion of the causes of tremor and clonic spasm are toxæmic, idiopathic, or functional origin, and it is in just such functional causes where one would find only partial destruction (or partial recovery) of the motor tract.

§ **590. I. Paralysis Agitans** (Synonyms : Shaking Palsy, Parkinson's Disease).—The onset is extremely gradual, and the three cardinal symptoms are (i.) tremor, which is moderate in size, rhythmical, and of general distribution, excepting the head and neck. It continues when the limbs are supported. Not infrequently it predominates on one side of the body, or in the arms and legs, and the onset is often accompanied by "rheumatic" pains—facts which point to a degenerative lesion in the peripheral nerves.[1] (ii.) The muscles gradually become stiff, and the attitude and aspect of the patient (Fig. 4, p. 24) are very characteristic. The head appears to be fixed, and is bent a little forward ; and in walking the patient has a tendency to walk forward (festination). The patient's face appears like an expressionless mask with the eyes always looking forwards. (iii.) The patient is nearly always over forty years of age, and, in 65 per cent. of the cases, of the male sex. (iv.) The other and less important points for differential purposes are progressive weakness, which gradually involves all the limbs ; the speech, which is drawling, indistinct, monotonous, and slow ; and various subjective sensations, such as restlessness and a continuous desire to be moved, if in bed. The intellect is preserved, and though the disease lasts for many years, it does not usually shorten life. No complaint is made of vertigo or nystagmus. Towards the end of life the tremor disappears, and is replaced by paralysis, rigidity, and contractions.

The pathology of paralysis agitans has not been completely settled, but some years ago Dr. Robert Maguire and the author, by examining the nerves after death, found varying degrees of degeneration in the

[1] In one case Dr. Robert Maguire and the author found after death degenerative lesions in all the chief peripheral nerves.

different peripheral nerves and other parts of the nervous system. In view of the remarks in § 589, this would fully explain the symptoms. It seems probable that the degeneration is a prematurely senile one—just as premature senility may affect the arteries.

A **senile tremor** is described by some, but in the course of a large infirmary experience I was rarely able satisfactorily to differentiate paralysis agitans from senile tremor. However, in the latter the tremor always involves the head and neck, being, indeed, sometimes most marked in or limited to this situation, and rigidity and paresis are less marked.

§ 591. **Disseminated Sclerosis.**—Islets of sclerosis in the spinal cord were described by Cruveilhier as an anatomical condition, but the clinical symptoms attaching to the condition were not described until Professor J. M. Charcot studied this condition clinically, and gave to it the above appropriate name. There are three types of the disease recognised, the spinal, cerebral, and cerebro-spinal, according to the part chiefly involved. The three characteristic symptoms of this disease are (i.) the tremor, which is very rhythmical, rather larger than paralysis agitans, and occurs only when the muscles are in action, ceasing when the limb is supported. This is, indeed, the most typical of "intention tremors." It involves all the muscles, including those of the head and the tongue. The intention character differentiates the tremor from paralysis agitans, and it may be distinguished from chorea by the fact that the arm goes straight to its goal, as, for instance, in feeding, whereas in chorea it takes a zig-zag course. (ii.) The patient is usually under thirty (the earliest case I have observed was sixteen years old). It rarely commences in persons over forty-five or fifty years of age. The sexes are equally affected. (iii.) The speech is characteristic, being slow and syllabic—*i.e.*, pausing between the syllables of a word and clipping the labial consonants. There is a torpid condition of the intellect, especially in those cases where the brain is also involved. (iv.) Vertigo is also an important and early symptom, occurring in something like 70 per cent. of the cases. (v.) Nystagmus is another constant symptom, and it is often accompanied by primary optic atrophy (white atrophy). (vi.) Various other symptoms may arise, according to the position of the patches of sclerosis. Thus, if the lateral columns be much involved, we get spastic symptoms; if the posterior columns, tabetic, and so on. In an analysis of fifty cases, Dr. F. S. Palmer[1] found that weakness, especially of one limb only, was the first symptom noticed in the disease in fifteen cases. The patellar reflexes are usually increased, but they may be normal or diminished. Their absence is serious, as denoting involvement of the grey matter. Various sensory disturbances may also be observed. (vii.) The course of disseminated sclerosis is prolonged; it may last from two to ten years, being longest in the purely spinal form, shortest in the cerebro-spinal form.

Hysterical Tremor is a very frequent manifestation of that diathesis. It may be small and regular or large and choreïform (see below).

General Paralysis of the Insane has been fully described in Mental Disorders. Here the tremor is very fine, regular, and vibratile, and it tends to affect the lips and tongue more than any other parts; indeed, at first these alone may be involved. It is accompanied by more or less general weakness and by characteristic mental alteration.

Alcoholic Tremor is one of the most constant evidences of chronic alcoholism. It is specially noticeable in the hands, is small and vibratile, rhythmical, dependent on muscular action (intention tremor), and worst in the morning. There is also a history of dyspepsia with morning vomiting, insomnia, and other evidences of chronic alcoholism.

Metallic Tremor.—Mercury, lead, and zinc, especially when introduced into the system in the form of vapour, or in small doses for a considerable length of time,

[1] "Early Manifestations of Insular Sclerosis," *Med. Press and Circular*, September 7, 1904.

not infrequently produce tremor. It is small, rhythmical, and only apparent when the muscles are in action. It is also characterised by the other signs of poisoning by the respective metals. Mercurial tremors occur, for the most part amongst thermometer-makers, and water-gilders in the manufacture of mirrors. Lead tremors occur largely amongst the Cornish and Cumberland lead-miners, and zinc poisoning amongst brass-founders, who are exposed to the fumes of oxide or oxychloride of zinc.

Other **toxæmic conditions** are also attended by tremor, such, for instance, as diabetes, ague, influenza, and pyrexial states. Here the tremors are small and rhythmical, and a history of the cause is easily made out. **Graves' disease** is mostly attended by a fibrillary tremor, and in such cases inquiry should be made for thyroid enlargement, ocular prominence, cardio-vascular phenomena, and other neurasthenic symptoms, the tremor being really one of these.

Nervous Tremor arises from either debility of the muscular or nervous system, and is seen in its most typical form in cases of *neurasthenia* or *general debility* from any cause, under which headings the other characters of the various causal conditions are described. Tremor may also be present in *occupation neuroses*.

There are **organic diseases** in which tremors occur, but these are usually accompanied by other more prominent symptoms, as in *amyotrophic paralysis*.

Tumour of the brain may be accompanied by muscular movements, especially (i.) when the tumour presses on, without actually destroying, some part of the Motor Tract—*e.g.*, in children who are the subjects of a tuberculous tumour near the crura or the posterior part of the internal capsule—there is tremor exactly like that of disseminated sclerosis. (ii.) If it be situated in the Parietal Region or near the Optic Thalamus, the tremor usually takes the form of spastic athetoid movements. (iii.) Cerebellar lesions may be associated with spasmodic movements of the neck muscles on one or other side. (iv.) Some lesions in the Frontal Region may be attended by a fine tremor of the hand on the same side (T. Grainger Stewart). (v.) Lesions of the Red Nucleus or its connections with the Cerebellar Dentate nucleus of the opposite side or with the anterior horns below are attended by tremor.

Lateral sclerosis often gives rise to tremor of the legs on walking or any muscular movement.

Friedreich's disease is characterised by tremor or by disorderly movements.

§ **592. Chorea** (St. Vitus' Dance ; Sydenham's Chorea) is a disorder of the nervous system generally occurring in childhood, characterised by irregular awkward movements of the limbs and a tendency to cardiac valvular disease, running a more or less definite course usually towards spontaneous recovery. It was first described by Sydenham in the seventeenth century.

(1) The movements usually partake more of the character of gesticulations or exaggerations of normal movements than of tremors or even clonic spasms. In carrying a spoon to the mouth, the hand does not reach its goal, as it does in disseminated sclerosis. The movements may involve all the muscles of the body, even those of deglutition, the face, the tongue, and respiration, including the diaphragm. They are sometimes hemiplegic in distribution, and may predominate in the upper extremity or the face. The movements cease during sleep. The deep reflexes vary. (2) A certain amount of paresis may accompany or alternate with the movements, and the chief symptom for which many patients are brought is because they drop things or fall down. Sometimes they are brought for restlessness at school. Very severe cases may develop maniacal symptoms. Eruptions on the skin (ery-

thema, herpes zoster, purpura, and subcutaneous nodules) have been observed. (3) It is also accompanied, in a large proportion of recurrent cases, by valvular disease, which is indistinguishable from that of acute articular rheumatism.

The *Diagnosis* from habit spasm is occasionally difficult, but the latter is more persistent in duration, more limited in distribution, and the involuntary movements always affect the same set of muscles. It may also have to be diagnosed from various other clonic spasms mentioned below.

The *Prognosis* is usually favourable, and tends to spontaneous cure in the course of one to three months, though in about one-third of the cases permanent cardiac disease remains, and 20 per cent. of all cases of chorea get rheumatism within six years.[1] The severity of the attack depends a good deal upon the age of the patient. Over the age of puberty the disease assumes a much graver aspect, and is very apt to be recurrent; and when it complicates pregnancy in young women the mortality, according to Dr. Robert Barnes,[2] amounts to 30 per cent. Great severity of movement, recurrence of attack, and maniacal symptoms (chorea insaniens) are always grave.

Etiology.—The disease is essentially one of childhood, and is three times as frequent in the female. There is a special liability for chorea to follow articular rheumatism, quinsy, scarlatina, and to a less extent other infective disorders. For many years I have taught that chorea was microbic in origin, and pointed out in proof (1) the practical limitation of the disease to childhood; (2) its more or less definite course and tendency to spontaneous recovery; (3) the marked tendency to endocarditis, and (4) the spread of the disease to other children by so-called "imitation." In 1903 Dr. J. F. Poynton revealed the rheumatic diplococcus in cases of chorea, and the disease is now coming to be regarded as microbic. As in rheumatism, there is a marked tendency to recurrence, and it is predisposed to by many of the same conditions as the acute specific fevers. Fright or other sudden emotion may determine an attack.

Treatment.—Salicylates as first introduced by Dr. David B. Lees, administered in the same way as in rheumatism, are almost as successful in chorea. Aspirin and quinine have been tried. The patients must be taken from school, and do far better in bed even in slight cases. The movements, if violent, may need bromides and large doses of chloral (10 grains every two hours if awake) or trional; chloretone is valuable. A water-bed is desirable. Arsenic in gradually increasing doses is the best tonic, and may alone be sufficient to cure mild cases. A wet pack and other methods for the application of heat or cold are excellent means of treating severe cases (see § 596).

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[1] Dr. F. E. Batten, the *Lancet*, November 5, 1898.
[2] St. Thomas's Hospital Reports, about 1870.

Huntingdon's Chorea (Chronic Chorea, Hereditary Chorea) is characterised by irregular gesticulatory movements, coming on gradually between the thirtieth and fortieth year, and lasting for the rest of life. The gait is irregular and swaying, with sudden stoppages, the speech is affected, and there is mental impairment leading to dementia. This disease runs in families. A similar affection coming on in the aged, without hereditary cause, is known as *senile chorea*. Rhythmical and other forms of chorea are mentioned below. It is unfortunate that the term " chorea " has been applied to these various conditions, which are totally distinct from Sydenham's chorea. *Henoch's chorea electrica* is probably identical with paramyoclonus multiplex (below).

Hysterical tremor and spasms are of different kinds. They may be generalised, like chorea, or localised to one situation ; and they are aggravated by, but not dependent on, voluntary muscular action. They are characterised by (1) their sudden access, generally after some emotional shock ; (2) they vary in size, rhythm, and even in position from hour to hour and day to day ; (3) they have a tendency to predominate in the head, neck, or arm ; and (4) they occur in a characteristic age and sex. A typical case of this sort was that of a young lady, æt. twenty-four, whom I saw lately with Dr. Alfred Masters, who had suddenly developed choreïform movements in the right upper extremity while under the unusual influences of a long stay at her fiancé's home. The disorder consisted of a continuous regular tremor of the arm, interspersed with attacks of large irregular clonic spasms. The knee-jerks were so much exaggerated that the patient almost jumped out of the chair. Another class of hysterical spasm has been appropriately named chorée rhythmée by Charcot, in which condition the movements are distinctly choreïform, more or less generalised, and differing from ordinary chorea, first, by their coming on in attacks, or, at any rate, being liable to severe exacerbations ; and secondly, by having a tendency to a certain degree of regularity. In one case of this kind which I observed[1] these attacks could be started by pressure on the mamma, and they could be stopped by steady pressure in the ovarian region. In addition to these two forms, which might be called localised hysterical spasms and chorée rhythmée respectively, there are a large number of other rarer kinds, which may be provisionally placed in the hysterical group, though their precise relation to hysteria has not been made out. For instance, *saltatory spasm* (jumping chorea) is a rare condition of clonic spasms, affecting principally the lower extremities, which are usually in a state of more or less rigidity, and subject to violent extensor spasms whenever the soles of the feet are touched, or when the patient is placed upon the feet. The hands and arms are usually free. Hammering and dancing chorea have also been described by Charcot[2] and others.

Myoclonus Multiplex is a very rare condition described by Friedreich.[3] Judging by the few cases that have been recorded, this condition may be provisionally described as a disease consisting of attacks of sudden shock-like clonic muscular spasms, not usually sufficiently prolonged to produce tremor of the limbs, bilateral, affecting mainly the proximal segments of the four extremities ; occurring mostly in males, and running a chronic course. Spasms of larger range may occur. It is probably due to some congenital or hereditary defect, though it may not be revealed for several years after birth. The most curious part of this strange disorder is the exemption of the wrists, ankles, hands, and feet. It is usually relieved by fairly strong galvanic currents, but is apt to relapse. The only case that I have seen had lasted for nearly thirty years. Much confusion exists as to the identity of the disease.

§ **593. Spasmodic Tic** is a recurrent, involuntary, clonic spasm affecting certain groups of the voluntary muscles, and producing

[1] *Clinical Journal*, October 19, 1898.
[2] " Leçons Cliniques sur les Maladies du Système Nerveux."
[3] Virchow's Archiv, bd. lxxxvi., p. 421, 1881.

twitchings which at first are limited to one place, but apt to spread. Many names have been applied to the condition—Habit Spasm, Convulsive Tic, Tic Non-douloureux, Impulsive Tic (Gilles de la Tourette), Spasmodic Torticollis—but they are essentially all the same. In the author's view their pathology is similar, and depends on two factors : (*a*) An instability, natural or acquired, of the subconscious and reflex centres ; (*b*) a repetition of a certain movement or trick until it becomes first involuntary, then automatic, and finally exaggerated. They also depend on the principle that nervous impulses travel more easily along a track they have travelled before—*e.g.*, by repetition. In the treatment of some of these cases the author has been very successful with systematised muscular exercises and deep breathing.

Tic or **Habit Spasm** is a first cousin of chorea. It is, in fact, a localised choreiform movement, and it is often difficult to distinguish between the two diseases. It practically always starts in childhood, mostly in girls between seven and fourteen, and is, indeed, an exaggeration of the normal restlessness of this age. It consists of sudden quick muscular twitchings, say of the eyelids, face (causing grimaces), shoulder, or arm, differing from the chorea in (i.) being always limited to one place, (ii.) performing the same action, and (iii.) running an indefinite course. The face and arms are the most frequent situations, but the muscles of respiration or any other part may be involved, and respiratory sniffs or grunts are not infrequent. It is always worst when the child is nervous, or attempts to restrain the movements, and parents should be cautioned not to scold the child for the habit. There is frequently a history of neurosis in the family, and habit spasm may certainly arise by the " imitation " of other children. Arsenic is of considerable value, but other medicines are not of much use. Removal from the surroundings under which the disease arose is the most efficient remedy.

A violent form of **Impulsive Tic** allied to habit spasm, and, like it, mostly arising for the first time in children, though not confined to them, consists of explosive muscular movements, usually of the face or arms, but in violent cases all over the body. Explosive sounds accompany these movements, either barking or inarticulate grunts, or the constant repetition of one word (echolalia), or some obscene or swearing word (coprolalia). The condition often occurs in association with some mental deficiency.

Localised Clonic Spasm may affect almost any voluntary muscle or group of muscles in the body. The case should be investigated on the lines below indicated under Facial Spasm. A large proportion are hysterical. In clonic spasm of the *diaphragm* very curious respiratory spasms and grunts occur. Hysteria and possible reflex causes of irritation in the stomach should be remembered. It is also seen with organic cerebral disease. Clonic *masticatory* spasms may arise in paralysis agitans and old age, and sometimes in hysteria. *Nutatory* or nodding spasms occur in children at the time of dentition or as a form of epilepsy, sometimes, associated with nystagmus.

§ **594. Clonic Facial Spasm** (Synonyms : Spasmodic Tic, Convulsive Tic, Tic Non-Douloureux, Mimic Spasm) is the term employed for a condition of persistent clonic spasm of the muscles of the face. Clonic facial spasm may arise under three different conditions : Organic lesion, reflex irritation and idiopathic or constitutional causes. The favourite age for facial spasm is between thirty and sixty.

An *organic lesion* may give rise to facial spasm in the same way as it gives rise to facial paralysis, and this cause is known either by the presence or a past history of paralysis. However, if spasm be present, it is an indication either that the destruction of the facial nerve tracts was incomplete, or else that partial recovery has taken place (compare end of § 589). In this way tumours, such as sarcoma or

aneurysm of the vertebral artery, pressing on without entirely destroying the facial nerve, give rise to facial spasm. A fall on the head or a cortical injury at birth may act similarly. Diseases of the bones (syphilitic or tuberculous) through which the nerve passes may also be suspected ; and all the structures beside which the nerve passes should be carefully and thoroughly investigated. If the lesion be cerebral the spasm is more general, and is apt to involve muscles physiologically associated, perhaps on opposite sides. Thus, if both corrugators and the muscles on one cheek only be involved, a cortical lesion is indicated. If it be the nucleus or the nerve trunk that is affected, we then find that there are, or have been, the paresis and electrical changes characteristic of this condition.

Reflex irritation, especially of the fifth nerve, may be in operation. The teeth should be examined, a history of injury to the fifth nerve inquired for, pharyngeal adenoids removed, and the refraction should be tested.

Idiopathic Causes, such as grief or other emotion, debility, anæmia, the climacteric, hysteria, gout, rheumatism, syphilis, or tubercle, may act as contributory causes. It is this group that belongs to the class Spasmodic Tic (*vide supra*).

Facial spasm of *organic* origin may be *diagnosed* from habit spasm by its persistence, the age of the patient, the surrounding circumstances, and electrical changes (if any). But I know of no means of distinguishing *non-organic* facial spasm from habit spasm in the face, though all the books describe them separately.

Course and Treatment.—Facial spasm, as above described, is generally a very chronic and progressive condition, resisting all our efforts excepting in those cases where the cause is removable, and the causes should be thoroughly investigated on the lines above given. Weak galvanism of the affected nerve may be tried. To palliate the spasm bromides, gelsemium, cimicifuga, antipyrin, chloral, and in extreme cases morphia and other nerve sedatives may be employed.

§ 595. **Torticollis** (Wry-Neck) is a spasm of the muscles on one side of the neck. Wry-neck is of two kinds—the congenital, due, probably, to injury at birth ; and the acquired, which may appear at any age. In the former condition the spasm is tonic ; in the latter the spasm is chiefly clonic, but in long-standing cases there may be some tonic spasm as well. (*a*) *Congenital* wry-neck is due to a contraction of the sterno-mastoid of one side, rarely both. The chin is projected upwards and to the opposite side. It may not be noticed by the parents till the child is several years old. It is generally associated with facial asymmetry, as pointed out by Sir Samuel Wilks. The only remedy is tenotomy. (*b*) *Acquired* torticollis consists of a slow clonic spasm, recurring every few minutes, associated in long-standing cases with a certain amount of tonic spasm which, when the sterno-mastoid is involved (as is usual), draws the head to the opposite side. In about half the cases the trapezius is associated in the spasm ; in other cases the splenius, scalenus, and platysma may also be involved. The condition is very intractable.

Causes (compare also the remarks on Facial Spasm, which apply here) and *Treatment.*—The causes of torticollis are often obscure. The acquired form very commonly appears to be of hysterical origin. I have met with cases coming on after injury to the back of the head, and in some cases relief has been obtained by remedying a gouty or rheumatic habit of body. Like facial spasm (*q.v.*) wry-neck probably arises under three different conditions—organic lesion, reflex irritation and idiopathic causes—and treatment should be directed to these. Many drugs have been tried, without much benefit. Galvanism of the spinal accessory nerve regularly applied twice daily has in one instance produced permanent relief. Surgical means (stretching, division or excision of the nerve, or division of the muscle and resection of the posterior branches of the upper cervical nerves) have been tried, but as far as I am aware, without much success.

Muscular twitchings or startings may arise in muscles which are over-fatigued or in the half-waking state in various conditions attended by constitutional debility (*q.v.*). They are an occasional symptom of neurasthenia, and twitchings of the same kind affecting the legs are also one of the earlier symptoms of peripheral neuritis.

§ 596. Post-Paralytic Tremors and Spasms.—The fact that hemiplegia and paraplegia of organic origin is gradually succeeded by tonic rigidity and other evidences of lateral sclerosis has already been referred to, but occasionally we also get clonic movements of various kinds supervening sooner or later, especially when the paralysis has occurred in early life.[1] There are many different kinds of clonic spasm, but practically they come under three types, which, in order of frequency, are (*a*) a slow mobile spasm called *athetosis* ; (*b*) movements which are more or less *rhythmical ;* and (*c*) irregular choreiform movements not inappropriately called *post-hemiplegic chorea.* They are all characterised by (1) having the same distribution (generally hemiplegic) as that of the preceding paralysis, though in cases of hemiplegia the arm is always more affected than the leg. (2) The presence of the other symptoms of lateral sclerosis. All of these movements are relatively rare in adults, but in such cases they are more frequent after hemiplegia due to an embolic lesion than after a hæmorrhagic one, and also after lesions which are associated with hemianæsthesia. The jerkings of the legs quite early in cases of paraplegia due to a complete transverse lesion with a fairly extensive area of healthy cord below, are caused, not by descending sclerosis, but, as most believe, by the cutting off of inhibitory influences from the brain.

The *Prognosis of Tremors and Clonic Spasms* in general terms is much more favourable than that of hemi- or para-plegia—a fact which is in keeping with the remarks on pathology in § 589. The prognosis of some has been dealt with in detail. (1) In none of the above conditions is there, as a rule, any immediate danger to life, excepting in certain cases of chorea and paralytic dementia. (2) There are but three of the above maladies which tend progressively to a lethal termination—viz., disseminated sclerosis, paralytic dementia, and progressive muscular atrophy. Disseminated sclerosis lasts for an average of five or six years, being shortest in the cerebro-spinal form and longest in the spinal form, the cerebral form occupying an intermediate position. In all four of these the course rarely lasts longer than ten years, and in acute cases only about a year or so. (3) Another group of the above diseases endure for a lifetime, though without materially shortening it—viz., paralysis agitans, senile tremor, lateral sclerosis, many cases of facial and neck spasm, and some of the rarer group allied to hysteria. Nevertheless, some of these can be ameliorated. (4) Many of them progress towards spontaneous recovery—*e.g.*, chorea, which is perhaps the most frequent clonic spasm met with, hysterical and nervous tremors, habit spasm, and many cases of facial spasm. (5) Many of the above are curable, chiefly by removing the cause—for example, in alcoholic, metallic, and other toxic tremors, Graves' disease and the great majority of those which depend on reflex and idiopathic causes.

Indications for the *Treatment of Muscular Tremor and Spasm.*—For the rational treatment the reader should refer to the brief reference to the pathology (§ 589 and § 593), giving the three pathological causes on which the cases may depend. The indications for treatment are fourfold. (1) To restore the partially damaged motor fibres or otherwise remove the cause. Iodide of potassium is of use, not only in cases of known syphilitic origin, to promote absorption, but also to eliminate toxic agents, such as lead, mercury, etc. (2) Careful investigation should always be made for any reflex irritation, such, for instance, as any uterine, stomach, and other visceral trouble in cases of hysterical spasm. (3) The third indication is to remedy any constitutional or general defect, such as anæmia, rheumatism, debility over-work, or over-worry. (4) If the cause cannot be ascertained or eradicated, we can, nevertheless, in many cases alleviate the tremor or spasm by appropriate means. Thus, hyoscyamus and its alkaloid (hyoscine hydrobromate, gr. $\frac{1}{200}$ to $\frac{1}{50}$ ter die) are very valuable in this respect, and many instances could be quoted of considerable relief in cases of paralysis agitans, senile tremor, disseminated sclerosis, etc. Cannabis Indica, physostigma, conium (succus ℥ss. increased to ℥ss.) may also be tried. Opium and morphia are permissible in some severe cases,

[1] This is one of the facts supporting the belief that tremors and clonic spasms of organic origin are due to partial destruction or partial recovery of a motor nerve tract. It is the recuperative power of childhood which leads to partial recovery.

but, as a rule, the relief is only temporary. Bromides are undoubtedly of great value in neurasthenic and other nervous tremors, hysterical tremors and spasms, paralytic dementia, and chorea. Warm baths, Turkish baths, and the application of heat is an agent for the relief of tremor and spasm which is too often neglected. Hot water internally and externally is the best remedy I know of for the relief of spasm of unstriped muscular fibres (such as spasmodic dysmenorrhœa) and the hot pack[1] or hot bath, as mentioned before, acts wonderfully in cases of chorea. The very simplicity of this remedy—the application of heat—renders it all the more valuable. In cases where the paralysis is a leading feature strychnine may be given, though, as a rule, this remedy is contra-indicated in nearly all clonic spasms. Tremors and spasms of hysterical origin must be treated on general lines, as in other cases. Cold douches are useful for functional spasms. Localised muscular spasms are also treated by massage and electricity, the anodal pole being placed on the affected side.

The patient has **attacks** *of* **clonic and tonic spasms** *associated with more or less* DISTURBANCE OF CONSCIOUSNESS. The case is one of CONVULSIONS.

§ **597. Convulsions** are sudden, violent, clonic, and sometimes tonic spasms affecting the greater part or the whole of the body, usually accompanied by some disturbance of consciousness.

The most common cause of convulsions is idiopathic epilepsy, and convulsions arising from other causes may so closely resemble epilepsy that they are often described as epileptiform convulsions. Eclampsia is an old term used for epileptiform convulsions. The following are the causes of convulsions :

Functional.

I. Idiopathic epilepsy.
II. Hysterical convulsions.

Organic Lesions.

III. Intracranial syphilis.
IV. Gross lesions of an irritative nature, such as (1) intracranial tumour (Jacksonian epilepsy) ; (2) hæmorrhage and embolism ; (3) chronic degenerations of the nervous system ; (4) acute meningitis.

Toxic Causes.

V. Uræmia, diabetes, cholæmia.
VI. Puerperal eclampsia.
VII. Various other toxæmic conditions, such as (1) alcohol ; (2) lead ; (3) drugs ; (4) tetanus (tonic spasm), diabetes, malaria, acute specific fevers (in children).

Circulatory.

VIII. Cardio-vascular disorders, including chronic Bright's disease.

Reflex.

IX. Reflex causes.

Convulsions in Infancy and Childhood.

[1] A thick blanket should be thoroughly wetted in a pail of boiling water, wrung out, and rolled up tight, and then the patient rolled in it, afterwards in another dry one, and left for half an hour to perspire. It is often advisable to promote diaphoresis by a dose of liquor ammoniæ acetatis, for the benefit derivable is much less unless perspiration ensues.

The chief points in the CLINICAL INVESTIGATION of a convulsive attack are the age of the patient, the character of the attack, the state immediately preceding and following it, and the attendant conditions. Ask *first* the *age*, and *secondly* whether the patient ever had an attack before. If the patient be under twelve months old, turn to Infantile Convulsions (§ 599). Between one and ten years of age embolism, hæmorrhage, and many constitutional derangements may give rise to convulsions. Between ten and twenty is the commonest time for idiopathic epilepsy to commence. If the patient be over thirty, and has never had a fit before, syphilis should be suspected. Hysterical convulsions mostly affect the female sex between fifteen and twenty-five and at the climacteric. If the patient be over fifty, uræmia and apoplexy should be suspected. *Thirdly*, the *characters of the fit* afford considerable aid in diagnosis. Thus (i.) the convulsions of major epilepsy and major hysteria are always generalised; partial convulsions indicate usually a cortical lesion (Jacksonian epilepsy). (ii.) Unconsciousness is an invariable accompaniment of epilepsy, and is very usual with uræmia, cerebral hæmorrhage, and syphilis. On the other hand, consciousness is not completely obliterated in by far the greater number of cases of hysterical convulsions, and in a considerable number of limited cortical lesions. *Fourthly*, inquiry should be made as to whether the fit was preceded by an aura (epilepsy), and what is the condition immediately afterwards. *Fifthly*, the history and attendant symptoms should be examined in the usual way.

§ **598. Idiopathic Epilepsy** (Synonym: Falling Sickness) is a disease which consists of sudden attacks of loss of consciousness, with or without convulsions, without any discoverable lesion in the blood or in the brain. It occurs in two clinical forms, *minor* epilepsy (petit mal), which consists simply of a transitory disturbance of consciousness, and is therefore referred to in § 529, and *major* epilepsy (haut mal, grand mal) which consists of a convulsive seizure with loss of consciousness.

Symptoms.—A complete epileptic fit has the following characters, though they are rarely all present in their entirety: (1) In some cases, during the previous twelve to twenty-four hours, there may be *prodromata*, consisting of headache, giddiness, malaise, or alteration of character or mood. In more than half the cases this stage is absent. (2) The fit in many cases is immediately preceded by an *aura* or warning—*i.e.*, a sensation lasting at most only a few seconds, which is of value as indicating the point of the cortex whence the cortical nerve-storm starts. Of these auræ there are four groups. *Sensory auræ* are most common—*e.g.*, "a wave passing over the body," numbness, flashes of light or of colour, or singing in the ears; *motor auræ—e.g.*, twitching of a muscle or a limb, rarely of the trunk, and in rare cases there is a "procursive aura," in which the patient runs forward or turns round and round; *psychical auræ—e.g.*, various strange thoughts or perhaps illusions; and, *somatic auræ—e.g.*, gastric discomfort, nausea, or fluttering in the stomach. Some form of aura is present, in my experience, in about three quarters of the cases. (3) *Loss of consciousness* is the pathognomonic and indispensable feature of *all forms* of idiopathic epilepsy. It succeeds the aura so quickly that the patient may not have time to place himself out of danger before loss of consciousness is complete. (4) *Convulsions* supervene almost

at the same moment as the unconsciousness. They are often ushered in with a scream, and in the classical form consist of a short stage of tonic convulsions lasting about forty seconds, followed by a stage of clonic convulsions lasting one to three minutes. In the tonic stage the breath is held, the hands clenched, the back rigid, the legs extended, the pulse quick and may be imperceptible. During the tonic stage respiration is stopped, and the patient becomes blue in the face (a diagnostic point of value). The clonic movements soon involve the whole body, and are sometimes of great violence, consisting of rapid extension and flexion of the limbs, opening and shutting of eyes and jaws. The tongue is often bitten—a danger to avoid by thrusting a piece of wood, the handle of a pocket-knife, or something of the kind between the jaws. The pupils are dilated and the conjunctivæ insensitive. As the convulsions pass off the respiration becomes stertorous or snoring. Urine, fæces, and even semen may be voided. The saliva issues from the mouth as a frothy foam, sometimes blood-stained from injury of the tongue. (5) A stage of *stupor* or *drowsiness* succeeds the convulsions, and may last for some hours. This stage passes gradually into a deep sleep. The temperature directly after the convulsions is said to be raised, sometimes as much as 4° or 5° F. (6) In the *post-epileptic state*, after recovery, there may be aphasia, or transient paresis, or the patient may perform automatic, irresponsible acts, dressing or undressing himself, or putting the property of others into his own pocket. Occasionally hallucinations, delusions, or active mania ensue, or the patient makes obscene remarks or commits acts of violence. Some patients are distinctly homicidal, rarely suicidal. One fact of considerable forensic import must be mentioned—namely, the phenomena of the post-epileptic state are by no means proportionate to the severity of the seizure (major or minor) which they follow—often quite the reverse. After severe fits the patient is exhausted, after slighter attacks his faculties are often stimulated, and he may perform acts of which afterwards he has no recollection.

The *intervals* between the fits vary considerably; any time from a few days to many years. In the intervals the patient usually remains in fairly good health of mind and body until the next attack; indeed, it frequently happens that he professes himself in better health after an attack than before. *Epilepsy* has a close association with *insanity*. In a certain proportion of cases the fits are followed by weakmindedness, whether the case has been treated with bromides or not; occasionally by other forms of insanity. It appears to be the frequency, rather than the severity, of the fits, the age at which they commenced and heredity that determine the occurrence of mental alteration. Thus, petit mal quite as often leads to mental trouble as does epilepsy major; some say more often. Again, epileptic fits may be followed (and sometimes even replaced) by mania of a most dangerous nature,

in which the patient may (quite unconsciously) commit acts of the most brutal kind. Epilepsy and insanity appear to be closely related to each other by their hereditary origin ; one child may be epileptic, another insane.

Varieties.—The above is the classical form of epilepsy major, but every degree of severity may be found between this and epilepsy minor. Frequently one or the other, or several of the above features may be wanting, but unconsciousness is the one constant feature, and in epilepsy minor it may be the only symptom present. Sometimes fits of various kinds may alternate in the same patient ; but generally each patient has fits which conform to one, or at most to two, varieties. The *status epilepticus* is a rare condition in which the patient has a series of fits occurring in very rapid succession for several hours or even days, consciousness not being regained in the intervals ; the temperature may rise to 107° F., and the condition may be fatal.

The *Diagnosis* of epilepsy minor will be found in § 529. Epilepsy major may have to be diagnosed from any of the causes of convulsions (see list, § 597 ; see also table below). Convulsions coming on for the first time after thirty are more probably *syphilitic* than idiopathic. In *feigned epilepsy* the pupils are not dilated, and they react normally to light, the conjunctivæ are sensitive, and the application of strong ammonia to the nostrils generally reveals the fraud ; the absence of the cyanotic condition may also aid. The alleged fact that the patient will not hurt himself is not of very great assistance, for I have often seen patients do themselves serious injury if the motive for the fraud is sufficiently powerful.

Course and Prognosis.—If unchecked by treatment the fits recur throughout life, though with widely varying frequency. The frequency of the fits is the leading factor in the prognosis. Those in whom fits occur with moderate severity a few times per annum may remain well and clever, but when more frequent mental deterioration results, not, be it remembered, as a consequence of the administration of bromide, but as part of the disease. Indeed, in petit mal, on which bromide has least influence, mental symptoms ensue more often than in grand mal. A combination of the two forms is worse than either singly. Death may occur from an accident during the fit, but rarely or never from the disease.

Etiology.—Both sexes are about equally affected. About 75 per cent. start the fits before the age of fourteen ; idiopathic epilepsy very rarely begins after twenty. Heredity is a potent factor, and in a large proportion there is a family history of epilepsy or other nervous ailments. Among the determining causes of a fit may be mentioned fright, excitement, head injuries, the menstrual period (many females have fits only at that time), alcoholic and sexual excess. Epileptic fits have

been known to be associated reflexly with such causes as the irritation of a scar, disease of the eyeball, disease of the nasal mucous membrane, on the removal of which the fits ceased. My colleague, Mr. Work Dodd,[1] has shown, moreover, that in certain epileptics having a high degree of asthenopia, the fits become much less frequent or cease altogether when the error of refraction is accurately corrected and the asthenopia relieved.

TABLE OF DIAGNOSIS OF EPILEPSY.

	Major Epilepsy.	*Hysterical Convulsions.*	*Feigned Epilepsy.*
Preceded by	Characteristic aura.	Globus, or choking, epigastric sinking or emotional disturbance.	A motive for fraud.
Mode of onset.	Sudden.	Sometimes gradual, perhaps with screams.	*Carefully planned.*
Characters of convulsions.	March definite and noiseless. Tonic brief, followed by clonic movements. Cyanosis during tonic stage; tongue may be bitten; urine and fæces may be voided.	Progress irregular, with screaming or crying. Tonic rigidity often prolonged and recurrent. Clonic movements often purposive and irregular; *tongue not bitten;* urine and fæces rarely voided; usually no cyanosis.	Not following the usual march; *absence of cyanosis and asphyxia;* urine and fæces not voided; tongue not bitten.
Consciousness.	Always lost.	Never quite lost.	Retained.
Eye symptoms.	Pupils dilated and irresponsive to light; conjunctivæ insensitive.	Pupils responsive to light; conjunctivæ sensitive.	*Conjunctivæ sensitive; pupils responsive to light.*
Duration.	Rarely exceeds a few minutes.	Generally exceeds 4 or 5 minutes, may last half an hour, and liable to recur.	Indefinite.
Termination.	Stupor and drowsiness.	Sobbing, crying, laughter, or prostration.	Not followed by profound stupor.

It is worthy of note that Hughlings Jackson[2] believes that every epileptic patient has an epileptiginous zone or area, corresponding on the surface of the body to the centre in the brain whence the cortical discharge starts; but in actual practice it is rarely possible to discover this zone.[3]

Three cases are recorded[4] in whom, in addition to fits occurring spontaneously in the usual way, slight cutaneous irritation in a definite area produced an epileptic seizure. In Dr. Jackson's case a boy aged eight, a flick with a silk handkerchief on the back of the head from behind, so that he received no warning, would result in his immediately falling down in a fit. In 1897, at the age of nineteen, this boy was under my care, and he presented hemiplegia and unmistakable signs of descending sclerosis and commencing athetosis. It is extremely interesting to notice

[1] *Brain*, 1895. [2] Med. Soc. Trans.
[3] Gowers, "Diseases of the Nervous System."
[4] James Dunsmure, *Edinburgh Med. Journ.*, October, 1874; Hughlings Jackson, *Brit. Med. Journ.*, 1886, ii., p. 962; Wilfred J. Harris, *Lancet*, 1897, ii., p. 537.

that all these cases presented important features in common. All were children—two boys and one girl—and in all the fits began early in life. In all the epileptiginous zone was situated in the head, and it was only when the patients were unaware that they were going to be touched that fits were produced. In falling, it was noticed that these patients generally struck their heads with great violence, suggesting that the clonic convulsions began in the neck muscles, pulling down the head in one direction. Finally, there were evidences in all pointing to a gross lesion of the brain as a cause of the fits. The two boys were hemiplegic, and this symptom was increased after the fits. The girl was not hemiplegic, but in her the history of a series of convulsions at the age of three, followed by two years' interval, points in the same direction (see § 593 below).

Treatment.—Bromides constitute the sheet anchor in the treatment of idiopathic epilepsy. Personally I prefer the ammonium salt in doses of 20 grains twice daily, gradually increased to 60 grains until the fits cease to occur. The administration should continue for two full years after the cessation of the fits. When thoroughly under the influence of bromide the palate reflex is lost. For nocturnal epilepsy add digitalis to a single dose of bromide. If bromides fail, borax, belladonna, picrotoxin (or a combination of this with bromide, known as Gelinot's dragées), amyl nitrite or liquor trinitrini three or four times a day may ward off an attack of E. major, but the two last aggravate E. minor. Morphia has been successful where other things failed. A regular life aids; and excess of animal food (of which these patients are very fond) and of salt, should be avoided. Regular purgation is of distinct advantage in many cases. Possible causes of reflex irritation should be removed. The treatment during an attack consists simply in placing something between the teeth and preventing injury. The *status epilepticus* may be ameliorated by amyl nitrite, chloroform, chloral hydrate by rectum, morphia, and ice to the spine.

Hysterical Convulsions constitute 18·5 per cent. (Gowers) of cases of convulsions. They form a frequent manifestation of hysteria, occurring in my experience in about 30 per cent. of the cases. The convulsions are irregular, but consist of tonic spasm, sometimes amounting to opisthotonos, *alternating* with clonic spasms, often accompanied by screaming, lasting longer than true epilepsy, and usually with purposive movements, such as dashing the head against a pillow, throwing the arms about, and struggling with those restraining her. The patient may then have a succession of fits for a few hours. The severer form described by Charcot, Richer, and others as hystero-epilepsy consists of four stages—tonic and clonic spasms, emotional attitudes, and delirium. Trance or lethargy may follow and last for days. The *diagnostic* features of a hysterical from an epileptic fit are: (1) The onset dates from some emotional disturbance, and it is not preceded by a definite aura, though it is frequently preceded by the globus or an epigastric sinking. (2) The convulsions do not follow the regular order of epilepsy; there is more tonic rigidity throughout, and many of the clonic movements have a distinctly purposive character. (3) The consciousness is very rarely lost; the conjunctivæ are sensitive, and the patient may be talking nonsense all the time (which never occurs in epilepsy), though she may not afterwards remember who was there. The urine and fæces are not voided. On recovering, the patient may burst into tears or laughter.

Hystero-Epilepsy, as described by Charcot and Paul Richer is very rare in England. In all my experience at the Paddington Infirmary I only saw three cases like those I saw in France. One of these was a man aged twenty-nine,

whose attacks were of great violence, and consisted of four stages : (1) a tonic, (2) a clonic, (3) a stage of purposive movements, and (4) a stage of incoherent delirium.[1] Another was a woman who had many fits in series of such violence that it took four people to prevent her harming herself *and others*, until I discovered that a hypodermic of apomorphine and copious emesis cut them short.

Intracranial Syphilis is the most frequent cause of convulsive fits occurring for the first time over twenty-five years of age. Convulsions are, moreover, a very frequent manifestation of cerebral syphilis (Charcot and others). Sometimes the convulsions are due to a gumma, sometimes to meningeal thickening, sometimes to a diffuse meningitis, but occasionally no gross changes are found beyond congestion of the cortex. In gummatous cases the convulsions have Jacksonian characters (see below), but in other cases the fits are indistinguishable from idiopathic epilepsy excepting by attendant circumstances—namely (1) the age of onset (over twenty-five) ; (2) severe headache, which is present in over 77 per cent. of the cases and heralds the attack—continuous, bilateral, markedly worse at night, and getting progressively worse up to the attack. Pains in the limbs may also be present. (3) There is more often mental hebetude between the fits. (4) If the lesion be gummatous there may be scattered lesions of the cranial nerves, and especially optic neuritis, or partial transient palsies or transient aphasia. (5) A history of primary or secondary syphilis.

Irritative Lesions of the Cerebral Cortex are always attended by convulsions.

(1) **Jacksonian Epilepsy** may be caused by a syphilitic gumma or other tumour, or a cicatrix in the brain after operation, injury, or hæmorrhage, *occupying the cortex* in the motor area (Fig. 150). (i.) When a lesion irritates one of the motor areas of the cortex, the clonic spasms are always partial—at any rate, at the outset, the convulsions *starting in, and sometimes limited to, the fingers or toes*, one forearm or leg, or the face on one side. The point of starting indicates the precise position of the lesion in the cortex. The clonic spasms may remain limited, or may gradually spread to the rest of the body. (ii.) There is *no loss of consciousness*, unless the movements become general and violent. (iii.) The attacks are apt to follow in frequent succession in a series. (iv.) There is no aura ; as a matter of fact, the twitching of the thumb or big toe, as the case may be, constitutes a motor aura, and is known as the " signal symptom."

(2) **Hæmorrhage and Embolism** of the brain are attended by a *generalised* epileptiform seizure in about half the cases. They are more frequent in hæmorrhage than in embolism, but this fact has not the value for purposes of diagnosis, which was attributed to it by the older authors. The convulsions under these circumstances usually predominate on one side, are at first attended by tonic spasm, and later by paralysis on the side opposite to the lesion. The age and history also aid in the diagnosis. Mitral stenosis favours the diagnosis of embolism.

(3) Various **Chronic Degenerations** of the nervous system are sometimes attended by *generalised* convulsions. The most frequent, perhaps, of these is general paralysis of the insane, especially in the congestive form. They are comparatively rare in disseminated sclerosis.

(4) **Acute Meningitis** (at the outset) and **Hydrocephalus** in children, and **Pachymeningitis** in the adult are frequently attended by *generalised* convulsions, but these have not Jacksonian characters. They are said to be relatively infrequent in alcoholic meningitis and cerebral abscess (Gowers).

[1] Clin. Soc. Trans., 1889.

Uræmic Convulsions are associated with renal disease, especially the chronic interstitial form. Convulsions, especially if associated or alternating with coma, occurring suddenly for the first time in a person over fifty years of age, offer a presumption of uræmia or apoplexy. Uræmia is distinguished from apoplexy by (1) the absence of hemiplegia and the pupillary reactions (§ 530); (2) an examination of the urine reveals renal disease. Finally, it should be remembered that apoplexy and uræmic convulsions may be present at the same time.

Puerperal Eclampsia (Synonym: Puerperal Convulsions) is the term applied to convulsions occurring before, during, or after labour, and they only differ from idiopathic epilepsy in the circumstances under which they occur. They are practically always associated with albuminuria, and are in all probability of toxæmic origin. They may be associated throughout with some rise of temperature. Bleeding and the transfusion of normal saline fluid (§ 406) have been warmly advocated. Chloroform and chloral are very useful.

Various **other toxæmic** conditions may be attended by epileptiform convulsions such as (1) ALCOHOLISM, especially in a chronic alcoholic after a drunken orgie. (2) LEAD POISONING occasionally, when other symptoms of plumbism would be present. (3) Various DRUGS given in overdose or attempted poisoning—strychnine (when the convulsions are mainly tonic), belladonna, strophanthus, stramonium, aconite, veratrium viride, sabadilla, hydrocyanic acid, in all of which the convulsions come on suddenly, other symptoms of the drug are present, and the case terminates one way or the other in twenty-four hours. (4) TETANUS (in which the spasms are chiefly tonic), and MALARIA in adults, and the onset of the ACUTE SPECIFIC FEVERS in children. (5) Convulsions also occur with DIABETES and with cholæmia in the terminal stages of DISEASES OF THE LIVER, such as acute yellow atrophy.

Cardio-Vascular Disorders give rise to epileptiform seizures in adult and advanced life more frequently than is generally supposed. Kussmaul has shown experimentally[1] that either anæmia or congestion of the brain may produce convulsions, and we know that profuse hæmorrhage in cut throat cases and cases of asphyxia may be so attended. This explains their occurrence in cardiac weakness, especially when associated with vascular disease in the aged, and in congenital heart disease in youth.

Adams-Stokes Disease, which illustrates the same principle, and has been already briefly referred to in § 58, is a rare disease, first described by R. Adams[2] in 1827, characterised by a permanently slow pulse, with syncopal and epileptiform attacks. The patients are usually forty-five and upwards, complain of dyspnœa, and have marked bradycardia, the pulse rate ranging from 30 to 40. Any mental excitement is liable to bring on an attack. The breathing becomes stertorous, the face cyanosed, there is dilatation of both pupils, rigidity of the body, accompanied by clonic movements of the limbs, the pulse beats more and more slowly, and finally ceases, the jaw drops, and for forty to eighty seconds the patient is to all appearance dead. No pulse is felt in either wrist, and on auscultation the cardiac sounds are inaudible. Then a feeble sound is heard, followed by a stronger, and a second later the pulse begins beating (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Such fits may occur in succession and are indistinguishable from epilepsy, excepting by the age of onset, the slow pulse, and the cardiac condition. Sometimes the attack resembles syncope, and is more transient.

Pathologically the condition is now believed to be due to a fibrosis of the bundle of His. This prevents the physiological transmission of the auricular wave of contraction to the ventricle, and consequently the ventricle misses about half the normal beats. With complete "heart-block" the ventricle temporarily stops beating and thus insufficient blood is sent to the head. The *Prognosis* is grave;

[1] Publications of the New Sydenham Society.

[2] Dublin Hospital Reports, iv., p. 396. See also the *Lancet*, 1885, vol. i., pp. 10, 288, and 447; also June 4, 1904, p. 1569, and February 13, 1904, p. 411.

patients die in these attacks or of heart failure. Treatment consists of heart tonics and spare living. Inhalations of strong ammonia may arrest an attack.

Reflex Causes occasionally produce epileptiform convulsions. In addition to those already mentioned under the etiology of idiopathic epilepsy it may be remembered that washing out the pleural cavity after aspiration and severe renal and biliary colic are occasionally followed by convulsions. Intestinal parasites, a tight prepuce, and any chronic peripheral irritation may cause convulsions in childhood (see below).

§ **599. Convulsions in Infancy** and **Childhood** may be produced by some of the foregoing conditions, but owing to the susceptibility of the nervous system in childhood, other and slighter causes may also produce convulsions. Judging by the frequency with which slight causes are followed by convulsions in children, it seems as though the instability of the cortical motor cells, which constitutes the essential disorder in idiopathic epilepsy, exists as a normal condition in infancy.

(1) *Blood Poisons.*—The acute eruptive fevers in adult life may be ushered in by a general tremor (rigor), but in childhood their advent is frequently marked by generalised convulsions. The advent of infantile paralysis, acute pneumonia, and many other acute diseases might also be mentioned. Rickets is often accompanied by convulsions, probably due to gastro-intestinal toxæmia. (2) *Reflex Causes.*—Constipation, worms, or any other irritative condition of the alimentary canal, teething, and the like, are frequently attended by convulsions caused either in a reflex or toxic manner. (3) *Gross Lesions of the Brain*, such as injuries to the brain at birth, hydrocephalus, tumours, abscess following otitis media, and some of the other gross conditions above named. Meningitis should be suspected if there is retraction of the head, peevishness on movement, or squint. Many of the causes of tetany, trismus, and carpo-pedal contractions, may also give rise to convulsions. (4) In children under ten *Cerebral Hæmorrhage* may occur, with convulsions, usually followed by hemiplegia (§ 555). In children a small cortical hæmorrhage or embolism may occur without a definite or very noticeable hemiplegia, and a few years later the cicatrix may be sufficient to cause recurring fits practically identical with idiopathic epilepsy. When fits have commenced under ten years of age, careful inquiry should be made of the mother as to whether any hemiplegia accompanied or preceded the first fit. It has been estimated[1] that in about one-half of these cases the convulsive seizures date from the occurrence of the hæmorrhage ; but in the other half they do not commence until some time afterwards. In some cases convulsive attacks preceded by an aura and indistinguishable from idiopathic epilepsy may continue at intervals throughout life, but in other cases the clonic spasms are unilateral or Jacksonian in character (see also cases referred to on p. 865, *ante*).

On the whole, the *Prognosis* of infantile convulsions is much more favourable than the same symptom in adults, a statement which follows from the opening considerations. In the *Treatment* attention should always be first paid to the intestinal canal, which is so easily upset in children, and as a general rule the administration of castor-oil and lime water in equal parts, a teaspoonful every four hours, is a good adjunct to other measures of treatment. Hyd. cum cret., magnesia, and soda are also useful. The cause in nine cases out of ten will be thus relieved, but careful inquiry should be made for other possibilities. To relieve the convulsions, bromide, preferably of ammonium, in 1 to 10 grain doses, is very useful, and it may be given with ½ to 2 grains of chloral. In severe cases chloroform may be administered, and a very little is sufficient to relieve the spasms.

[1] Hare, " Practical Diagnosis," p. 482 *et seq.* London, 1897.

δ. Muscular Atrophy.

§ **600.** Muscular wasting occurs in two groups of disorders : (1) Those due to a lesion of some part of the lower motor neuron (the peripheral nerves or their cells of origin in the anterior horns of the spinal cord), when it is spoken of as atrophic or amyotrophic paralysis, or briefly as amyotrophy ; (2) those due to a lesion in the muscle itself, when it is spoken of as myopathy, or idiopathic myopathy.

At least eleven clinical types of amyotrophy have been described, some with very little real difference, and those may be classified into four fairly marked clinical and anatomical groups.

a. Amyotrophies without Sensory Changes.

Anterior horns of spinal cord.	I. Acute anterior poliomyelitis (rare in adults).	Flaccid atrophic paralysis, with R. D. No sensory changes and no pain after onset.
	II. Acute and subacute anterior poliomyelitis of adults.	
	III. Progressive muscular atrophy (rare in children).	
	IV. Amyotrophic lateral sclerosis. Bulbar paralysis.	

b. Amyotrophies with Pain and Sensory Changes.

Nerve trunks.	V. Plexus paralysis.	Flaccid atrophic paralysis with pain and sensory changes. R. D. present.
	VI. Localised neuritis.	
	VII. Multiple neuritis.	

c. Myopathies.

Muscles only.	VIII. Myopathy, with loss of volume : Duchenne's infantile myopathy. Erb's juvenile myopathy.	Simple muscular weakness, with loss of volume, predominating in one or other place. No pain. No sensory changes. No R. D.
	IX. Pseudo-hypertrophic paralysis.	Same as preceding, but with increase of volume.
	X. Peroneal type (doubtful whether muscular or neuro-muscular).	May also have slight sensory changes and fibrillary twitchings.

d. Reflex Amyotrophy.

?	XI. Arthritic atrophy.	Extensor amyotrophic paralysis.

In the Clinical Investigation there are several important *fallacies* to be borne in mind. In the first place the amyotrophies must not be confused with the general wasting due to constitutional debility (Chapter XVI.). (2) They must not be confused with the different forms of upper neuron paralysis, which, when of long standing, are sometimes followed by a slight degree of wasting from disuse ; or (3) the wasting consequent on disuse after the application of Sayre's jackets or other apparatus.

The *Clinical Features* to investigate are (1) the flaccidity of the paralysis, the absence of the knee-jerks, and the electrical alterations, which are the three fea-

tures (in addition to atrophy) which distinguish lower neuron paralyses. (2) The distribution of the amyotrophy. (3) The presence of sensory alterations is of value to localise a lesion. (4) The mode of onset of the disease ; and (5) the age of the patient are also important.

§ **601. Acute Anterior Poliomyelitis** (Synonyms : Poliomyelitis, Infantile Paralysis, Atrophic Spinal Paralysis) may be defined as a disease of infancy, coming on suddenly with feverishness, characterised by rapid wasting and loss of power in one or more groups of muscles, due to inflammation of the anterior cornua mostly located in the lumbar or cervical enlargements. Clinically the malady has considerable resemblance to an acute infection, and epidemics have been observed. Histologically the lesion is a capillary thrombosis, not a true inflammation, and it is now coming to be regarded (Marie, Batten, and others) as microbic in origin.

Symptoms.—(1) The patient is usually about the age of the first dentition, very rarely older than fourteen years. The advent is sudden, and is ushered in by moderate pyrexia (like an acute specific fever), general prostration, and more rarely by convulsions. Pains in the limbs may be complained of at the outset, but do not form a prominent feature. (2) Sometimes the paralysis attacks only one limb, sometimes all four ; and it not infrequently takes the form of paraplegia. At first (in the course of twenty-four to forty-eight hours) all the muscles of the limb or limbs are affected with flaccid paralysis and rapid wasting, and they are tender to touch or movement. In a few weeks some of the muscles begin to recover, while the others undergo progressive wasting. All the affected muscles show the reaction of degeneration as early as a week after the attack. (3) The superficial and deep reflexes are abolished in the paralysed parts ; the knee-jerk remains lost only when the quadriceps is permanently paralysed. (4) There is absolutely no affection of sensation. The bladder and rectum are not usually affected, and there is no tendency to bedsores. (5) In the course of a few months the amyotrophy settles down into a muscle or group of muscles—for instance, in the leg the tibialis anticus or quadriceps (more rarely the hamstrings or glutei), and in the arm the deltoid, brachialis, or supinator. These muscles atrophy, degenerate, and finally, a year or so later, undergo contraction, producing various characteristic and familiar deformities (club-foot, etc.). Some hold that the deformities are due to contracture of the atrophied muscles ; and some that they are due to neurotrophic changes in the joints and ligaments ; but the unopposed contraction (which is constant) of the unaffected muscles has always appeared to me sufficient, bearing in mind the age-period at which the disease occurs. The affected limb becomes blue and cold, and the bones do not grow so much as on the healthy side. It seems probable from this that the anterior cornual cells control the tone of the involuntary as well as the voluntary muscles of the part.

Diagnosis.—The disease is practically confined to early childhood. At the onset the malady is mostly mistaken for a "chill," or the onset of an acute specific fever. In the pseudo-paralysis of *rickets* there is no atrophy. In *meningeal* affections the spinal pain is very marked, and the muscular atrophy is not so pronounced. *Peripheral neuritis* has more pain and does not produce such a rapid or localised amyotrophy. In old-standing cases the history of acute onset differentiates it from *idiopathic myopathy*. *Syphilitic* pseudo-paralysis due to separation of the cartilage at the end of the diaphysis has crepitation and pain on movement. In *infantile scurvy* there is tenderness and swelling of the affected limbs.

Prognosis.—The disease is not fatal in itself, but as a rule complete recovery is rare. Within a few weeks or months after the acute disturbance has subsided, the muscles may usually be grouped into three groups: (*a*) Those which will recover of themselves, and in these the electrical changes are but little altered. (*b*) Those which may recover under treatment. In these the galvanic changes are typical—*i.e.*, increased, with A.C.C.>K.C.C., and the faradic reaction is not quite lost. (*c*) Those which probably will not recover under any treatment, and in these faradism gives no response whatever. Death may occur from pneumonia or other intercurrent diseases.

Etiology.—The disease occurs chiefly in children under ten, usually about the age of one, two, or three years. It only occasionally affects adults. Chill has been supposed to cause it; and it has appeared during convalescence from acute diseases. It is more frequent in summer, and has occurred in epidemics. Its clinical course resembles an acute microbic disease, which results in destruction of certain groups of multipolar cells in the spinal cord.

Treatment.—At first rest in bed, salines, and quinine, or salicylates are all that can be ordered, with anodynes if there be much restlessness. At the end of a few weeks treatment should be begun for the atrophied muscles. For these the galvano-faradic current, or the galvanic current alone, followed by massage and shampooing form undoubtedly the most efficacious treatment. Electricity or massage alone is not nearly so valuable. It is generally held that no treatment is of any use after a few years have elapsed, but I have obtained considerable benefit in patients even twenty and twenty-five years later. The treatment of the deformities which result is surgical.

Acute and Subacute Poliomyelitis in adults (Synonym: Atrophic Spinal Paralysis of Adults) is almost the counterpart of infantile paralysis, occurring between twenty-five and thirty years old. It commences with fever and pains in the back, which may last a week or two, accompanied or followed by paralysis, frequently of both arms and legs. In the subacute form one group of muscles after another may become paralysed till the whole body is affected. The disease is frequently mistaken for multiple neuritis (§ 561), in which, however, pain in the limbs is a prominent feature. Time clinches the diagnosis, for whereas recovery is nearly always perfect in multiple neuritis, groups of muscles remain perma-

nently paralysed when the disease is acute or subacute anterior poliomyelitis. The latter is certainly very rare, and most cases so diagnosed have probably been multiple neuritis.

§ 602. **Chronic Anterior Poliomyelitis in Adults** is now considered identical with **progressive muscular atrophy.** It is not a common disease, but is of more frequent occurrence than the preceding. It is recognised by (1) a slowly progressive atrophy of the muscles, starting in the thenar and hypothenar muscles, usually of both hands, and spreading thence to the muscles of the arms and scapulæ. (2) The patient is generally an adult of thirty or forty years of age, or more, very rarely younger. (3) Partial reaction of degeneration is present ; there is progressive loss of faradic and galvanic contractility precisely as the muscle substance wastes (Ferrier). (4) There are no sensory symptoms. Owing to the early wasting of the interossei there are hollows between the metacarpals, and from the unopposed action of the extensors the first phalanges are over-extended, the two distal phalanges flexed—the typical " claw hand," or " main en griffe." The muscles show the fibrillary twitchings which used wrongly to be regarded as pathognomonic. Instead of the hand muscles, the deltoid may be the first to atrophy or the extensors of the forearm. Later the supra- and infra-spinatus, the lower two-thirds of the trapezius, and the trunk muscles are involved. The legs are spared till very late ; the knee-jerk goes when the least sign of atrophy of the thigh muscles is present.

The *Diagnosis* is not usually difficult, on account of the peculiar limitation to the hand in its earlier stages and its bilateral distribution. In local atrophies due to single nerve paralyses (§ 569), a history of a cause is usually present. In other forms of *neuritis* the paralysis follows a certain nerve distribution, and sensory changes are usually present. The peroneal type of muscular atrophy (§ 603) begins usually in childhood, in the peronei, or extensor muscles of the foot. Some years later the hands and arms atrophy, and the claw hand may be produced. The condition is possibly a neuritic affection. In *cervical pachymeningitis or tumour* there are paroxysmal pains and stiffness or twitchings of the muscles. In *amyotrophic lateral sclerosis* the muscles are rigid and the tendon reflexes increased. *Chronic disseminated myelitis* has symptoms of irregular damage to other parts of the cord. The *idiopathic myopathies* may be difficult to differentiate, but, as a rule, they commence at an earlier age, and other members of the family are affected. *Syringomyelia* produces amyotrophy of the arm or arms, but is attended by sensory symptoms and lateral sclerosis.

Prognosis.—The disease progresses very slowly. It may start in one arm first, and the other may not be affected for a year or longer. On the other hand, after a fairly rapid progress at the commencement, it may become stationary. The hand may be useless in six months, the arms in two years. The prospect of arrest is better in symmetrical cases, but is always bad in old age. Complications arise when the chest muscles are affected. The disease may spread to the medulla, when the symptoms of bulbar paralysis may ensue. On the other hand, the speech may be impaired for years without further symptom of disease in the medulla.

Treatment.—The course of the disease may be considerably delayed by appropriate treatment by galvanism and massage, with strychnine, which is more efficacious when given hypodermically, in the form of the nitrate, $\frac{1}{50}$ to $\frac{1}{20}$ grain once daily for months. Cod-liver oil and other tonics are also given.

Certain other mixed *lesions of the spinal cord* are attended by amyotrophy.

The Amyotrophic Lateral Sclerosis of Charcot is a very rare condition, which commences with atrophy of one of the upper extremities, attended by increased reflexes. Following this, after an interval of a few months, are the signs of descending sclerosis in the leg of the same side. The lesion gradually spreads to the pons, and the patient rarely lives more than two or three years. No known remedy seems to have any effect upon its course.

Bulbar Paralysis, or glosso-labio-laryngeal palsy consists of atrophic paralysis of the muscles supplied by several of the cranial nerves (§ 623).

(*b*) *Amyotrophy with Pain and Sensory Changes.*

Amyotrophy attended by *pain*, *numbness*, or other *sensory changes* is due to disease or injury of the *nerves*, *nerve trunks*, or *plexuses*, all of which it will be remembered are parts of the lower motor neuron.

IV. **Plexus Paralysis** due to lesions involving the nerve plexuses (§ 570).

V. **Single Nerve Paralysis**—*e.g.*, wrist-drop due to musculo-spiral paralysis consequent on injury or lead poisoning (§ 569).

VI. **Multiple Neuritis,** usually the result of some toxæmia such as alcohol (§ 561).

c. Myopathies.

§ **603. Myopathy** or idiopathic muscular atrophy (Synonyms : Primitive Myopathy, Muscular Dystrophy) is a somewhat rare condition full of obscurity. It may be defined as muscular wasting, starting, for the most part in childhood, in certain groups of muscles, and tending to become generalised, due to changes in the muscular substance, generally of hereditary origin. It was first studied and carefully described by Duchenne of Boulogne in 1850. Since then it has been studied by many different observers, whose names have been applied to the particular sub-variety which has come under their notice. These varieties, however, only differ from one another in (1) the particular age at which the symptoms are first noticed ; (2) the particular group of muscles first involved ; and (3) following Duchenne's original description,[1] whether the muscles are diminished or increased in volume, depending on the amount of interstitial fibrous tissue present in the muscles.

The features *common to all varieties* of this curious disorder are (i.) the presence of a hereditary taint and the fact that several of the same family may be affected. (ii.) The disease dates from very early life, though it may not be noticed till adolescence, or even later. (iii.) It runs a very slow course, lasting sometimes throughout life, though the patient generally dies of some intercurrent malady about thirty years of age, or earlier. (iv.) The muscular wasting, wherever it may begin, becomes widespread, and finally involves all the muscles. (v.) There is no definite reaction of degeneration, but cases have been recorded by the author[2] and others, in which electrical changes have occurred. As a rule the electric irritability is diminished equally to both currents in proportion to the degree of wasting present. (vi.) There are no sensory changes.

Idiopathic myopathy is met with clinically in two principal types, based on loss or increase of volume in the muscles. In both there is an atrophy and final disappearance of the muscle fibres, but increased volume is due to great increase of the interstitial fibrous tissue.

Myopathy with loss of volume, and without initial hypertrophy of the muscles (the hereditary infantile amyotrophy of Duchenne) has been grouped into two types :

α. Cases beginning in youth or adolescence (Erb's juvenile myopathy).

β. Cases beginning in childhood and infancy. (i.) Form without involvement of the face. (ii.) Form with involvement of the face (the facio-scapulo-humeral or Landouzy-Déjérine type. (iii.) Form beginning in the legs (rare). In most idiopathic myopathies the muscular wasting *begins* and predominates in the biceps, triceps, and supinator longus, and the shoulder muscles, the latissimus

[1] The two chief types described by Duchenne were the Hereditary Infantile Muscular Atrophy and Pseudo-hypertrophic Paralysis—Sur l'Electrisation Localisée (Sydenham Society's publication).

[2] *Brain*, vol. xviii., p. 605.

dorsi, teres major, and lower two-thirds of the pectoralis major. The hands are rarely involved. In the Landouzy-Déjérine type the face muscles are first attacked ; the naso-labial fold is lost, the lips are apart, the lower lip projecting, and a peculiar dull expression is present. In a good many cases first noticed in adolescence or later there is a history of the patient being unable to close the eyelids properly from earliest childhood.

Pseudo-Hypertrophic Paralysis, or **Duchenne's Paralysis** (Fig. 5, p. 25), is the only form of idiopathic myopathy in which there is an increased volume in the muscles, and this is due to an increase in the interstitial tissue. The clinical features by which it may be recognised are : (i.) The patient is always a child, the disease having been first noticed between the ages of four and fourteen, and he is generally brought to us for " weakness of the legs." (ii.) On account of the disease predominating in the legs, the walk is a waddle, and very characteristic, and when the patient lies down he is unable to get up without clambering up by placing his hands on his own knees. (iii.) There is a notable increase in the volume of some of the muscles, especially of the calves and buttocks, which gives the child the appearance of an infant Hercules. The muscles in pseudo-hypertrophic paralysis which are increased in volume are the calf muscles, the glutei, deltoid, supra- and infra-spinati. Muscles which are apt to be diminished in volume are the lower two-thirds of the pectoralis major, the latissimus dorsi, and the teres major. The weakness of the muscles of the shoulder girdle leads to a very characteristic symptom—namely, when one endeavours to lift the child under the arms the shoulders slip up to the ears. The hand muscles escape. Deformities such as lordosis and talipes equinus occur from the weakness and contracture of the respective muscles of the spine and legs.

As in the myopathy with diminished volume, heredity is a potent cause, and one may find in members of the same family examples of this disease and the other forms of primitive myopathy. In some members of a family there is myopathy with loss of volume, in other members increase of volume (pseudo-hypertrophy), a fact specially pointed out by Duchenne and Charcot. This goes to show that the cause, whatever it may be, is a developmental one, and that all these diseases are identical. This is further proved by the occurrence of both wasting and increase of volume even in the same patient.

Diagnosis of primitive myopathy.—The gradual onset and family history distinguish this disease from acute anterior poliomyelitis, which is the usual cause of muscular atrophy in children. Pseudo-hypertrophic paralysis is not difficult to diagnose by reason of the enlargement of the calves associated with weakness of the shoulder muscles. In neuritic muscular atrophy—*e.g.*, multiple neuritis—paralysis exceeds the atrophy, and sensory symptoms are usually present. Progressive muscular atrophy begins in the hands, and develops at a later age, and there are fibrillary twitchings. Neuro-muscular atrophy of the peroneal type is described below.

Prognosis.—All cases of muscular dystrophy have a progressive course, though very slow, lasting from ten to fifty years. Death occurs by involvement of the respiratory muscles, pneumonia, or other intercurrent maladies. In pseudo-hypertrophic paralysis the prognosis is grave, the child often dying before adult life. Gowers says that they rarely live for seven years after the power of standing is lost. Sometimes, however, the disease remains stationary for many years.

Etiology.—The disease is hereditary, and symptoms are most frequently noticed before puberty. Males are more often attacked than females. It is transmitted generally through the mother, she being healthy. These muscular dystrophies are closely associated on the one hand with progressive muscular atrophy, due to lesion in the anterior horns of the cord, and with the neuro-muscular or peroneal atrophy which is probably due to changes in the nerves.

Treatment.—The progressive wasting appears to be retarded when carefully selected gymnastic exercises are given, and electricity and massage may aid. In the pseudo-hypertrophic form it is important to retain the power of walking as long as possible.

Progressive Neuro-Muscular Atrophy (Synonym: Peroneal Type of Muscular Atrophy) is associated with the names of J. M. Charcot, Pierre Marie, and Howard Tooth, who have described it. It forms a connecting link between progressive muscular atrophy and the muscular dystrophies above described. Like the dystrophies, it is hereditary, and commences in childhood. It is of gradual onset, usually attacking first the peroneal muscles, leading to club-foot (pes equino-varus), but it may begin in the hands. The intrinsic muscles of the hand are affected, as a rule, several years later than the legs. There are fibrillary twitchings, slight sensory changes, and diminished excitability to electricity with reaction of degeneration. Gradually the wasting extends all over the body. When a case is first seen it may be difficult to *diagnose* from an old-standing case of acute anterior poliomyelitis, but the latter is not hereditary, and has no sensory symptoms. As a cause of acquired club-foot this disease should be remembered, and "claw-hand" in childhood is practically always due to this disease.

Arthritic Amyotrophy.—It was, I believe, Charcot[1] who first pointed out that joint lesions are sometimes followed by muscular wasting, quite independently of the disuse which may attend the injury or disease of the joint, since it may supervene too rapidly to be explained in that way. It may follow a contusion or joint trouble which was so slight as to be overlooked. The atrophy in such cases has three clinical features: (i.) It is limited to the muscles which move the affected joint; (ii.) it predominates in the extensors; and (iii.) there are no qualitative electrical changes, only a quantitative diminution. When the arthritic trouble is cured, massage and electricity will speedily restore the amyotrophy.

GROUP V. PAIN AND SENSORY SYMPTOMS.

Pain.

Pain is a symptom which has been referred to many times in this work, and the importance of investigating its *position*, *character*, *degree*, and *constancy* has already been insisted on. Pain is present in many nervous disorders, but it is almost the only symptom in Neuralgia and Migraine, two disorders which which will now be considered.

§ **604. Neuralgia** may be defined as a paroxysmal pain referred to the seat of some sensory nerve or its branches, in the absence of any obvious local organic affection. We will first consider neuralgiæ in general and then certain recognised types.

The pain of a true neuralgia is usually described as sharp, shooting, or knife-like, with *paroxysmal exacerbations*, and between the exacerbations there is a dull aching pain. It is usually associated with localised tender spots, especially at certain points called the "points of Valleix," by whom they were first studied. *Destructive* lesions of a sensory or mixed nerve cause mainly anæsthesia. *Irritative* lesions of the spinal ganglia (or their analogue the Gasserian ganglion), or of the sensory fibres on either side of these, cause pain, and may be attended by vascular dilatation, redness, œdema, and various trophic changes of the area of skin in which the sensory fibrils terminate. Herpetic vesicles are prone to appear;[2] and, in long-standing cases, atrophy of the skin (glossy skin) and subcutaneous tissue may ensue.

[1] "Leçons Cliniques sur les Maladies du Système Nerveux," tome iii.

[2] The author has elsewhere shown that herpetic vesicles are a definite indication, not of a destructive, but an *irritative* lesion.—*Clinical Journal*, September 7, 1898.

Clinical Investigation and *Causes of Neuralgia.*—The causes differ somewhat with the seat of the neuralgia, but certain general causes may be mentioned in the order in which the examination of the case should proceed—viz. : (*a*) Local, (*b*) reflex, and (*c*) constitutional causes. (*a*) A careful *local* examination should always be made of the nerve trunk chiefly involved—*i.e.*, in the position where the pain started, to see if it be the seat of tenderness, or other signs of irritation. The parts beneath and around the nerve should also be examined to see whether it be pressed upon by a tumour or disease of the bone through which it passes, or any other lesion ; for instance, what appears to be an intercostal neuralgia may be due to a small patch of dry pleurisy, or the pressure of a swollen gland, or rheumatic nodule. (*b*) In many cases of true neuralgia, some *reflex cause*, sometimes of a very trivial nature, is in operation, and the discovery of this depends very much on the clinical acumen of the physician. This is especially true of trifacial neuralgia, where a source of irritation in the teeth or some other part of the body is so frequently overlooked. The teeth may, to all appearances, be perfectly sound, and yet, as I have frequently found, an inflamed or irritated pulp may be revealed by the tenderness of some tooth when tested by tapping or the application of hot and cold liquids alternately. (*c*) *Constitutional* and general conditions act mainly as predisposing factors. Neuralgia is essentially a disease of adult life ; it is rare in children, and not common at the other extreme of life. It is said to be more common in the female sex, and a neurotic family history is often obtainable. Neurasthenic, nervous, and hysterical subjects are certainly more sensitive to pain, and more liable to be attacked by neuralgia. Anæmia is often in operation, and over-fatigue of mind or body, anxiety, over-lactation, unhygienic surroundings, or any other debilitating influence, may predispose to or determine an attack. Convalescence from an acute illness, tuberculosis, and various other cachectic states afford predisposition to attack. Neuralgias are frequently associated with gout, rheumatism, and ague. Many other toxic influences may give rise to neuralgia, such as alcoholism, plumbism, diabetes, and the other conditions mentioned under peripheral neuritis may be heralded by neuralgic pains. Syphilis may produce neuralgia in two ways ; the nerve may be pressed upon by syphilitic disease of the periosteum, theca, or bone through which it passes ; or the nerve may itself be the seat of a neuritis of syphilitic orgin. The former is the more frequent. Among the general exciting causes, exposure to cold and chill is by no means infrequent.

General Remarks on the *Treatment of Neuralgia.*—In all cases the cause should be carefully sought for, and if possible, removed. Treatment should be directed to any dyscrasia present, such as gout, rheumatism, ague, anæmia ; the latter especially offers a predisposition to

the development of the different neuralgiæ. For the rest the treatment resolves itself into tonics, hygienic measures, palliative measures, and radical measures. Among *tonics* quinine (in large doses) and arsenic take a leading place; iron, phosphorus, cod-liver oil, and strychnine are also useful. Nervous excitants, such as strychnine, should be avoided when the nervous system is exhausted, and nervous sedatives, such as bromides, administered when the nervous system is irritable. The *nutrition* should be attended to, cod-liver oil, and two or three pints of milk a day being given in addition to other food. Alcohol, as a rule, should be avoided, though it may relieve temporarily. Overfeeding, combined with perfect rest, the liver being attended to, often succeeds where other measures have failed. The patient should be kept in bed, and fourteen to sixteen hours of sleep per diem should be procured by large doses of bromide. Warm baths are certainly of great value, and I have sometimes been able to cure painful affections that have resisted all other means by Turkish baths, regularly and freely administered. The *internal palliative remedies* are numerous. Among the modern remedies, phenacetin, 5 to 10 grains, antipyrin (combined preferably with caffein citrate), acetanilid, grs. iii. to viii., and chloralamide are useful. It will be observed that many of the analgesic drugs are antipyretic as well, a point of interest when we remember that fever is accompanied by generalised pains; the two facts together support the idea that blood conditions alone are capable of producing pain. Tincture of gelsemium ℥ 10 to 20 every two hours certainly allays hypersensitiveness of the nerves, and I have found this combined with ℥ 20 of tinct. cannab. indica of use in migraine, an attack of which which may be sometimes thus aborted. They are also useful in other neuralgias. A favourite palliative prescription of my own is tinct. gelsem. ℥ x., antipyrin, gr. v., am. brom. gr. x., aq. chlorof. ℥ss. every three hours till relieved. Croton chloral, paraldehyde, and drugs of that class may also be useful, but their effect on the heart should be watched. Bromides, among which bromide of ammonium I believe to be the best, given regularly, combined with the "rest cure" (*vide supra*), are especially useful when uterine conditions are suspected. Chloride of ammonium in large doses is sometimes useful, especially for ovarian neuralgia. In the neuralgia of anæmia, inhalation of amyl nitrite often affords relief. I have often found that glonoin affords relief in neuralgiæ of various kinds; and both these last-named remedies are invaluable for angina and pains referable to a labouring heart. Finally we have in morphia a prince of remedies, either hypodermically or internally, but it should never be administered except by the medical man himself, and then in full view of the possibility of the development of a habit. Minute doses of morphia hypodermically may possibly have a curative as well as a palliative action on some neuralgias depending on neuritis.

Chief among *external palliatives* is warmth, whether by hot water, a muff-warmer, roasted cotton-wool, or poultices. Counter-irritation (*e.g.*, mustard leaf behind the ear), emplastrum belladonna, chloroformum belladonnæ, are useful ; and menthol and peppermint rubbed in, veratrin or aconite ointment, or oleate of aconitine painted on a *limited* area are all worth trying. Paquelin's cautery dotted over the course of the pain will sometimes produce marvellous results. Electricity may be employed for neuralgia in two ways. (1) A strong current (faradic or galvanic) causing pain will occasionally remove neuralgia at once. It acts as a counter-irritant and is suitable only for slight idiopathic cases and recent cases of hysterical neuralgiæ. (2) Weak faradism, galvanism (2 to 5 m.a.), just enough to be felt. Place the positive pole on the seat of pain, and the negative pole anywhere ; avoid sudden variations in strength of current. Faradism used as a sedative must be extremely weak, and with very rapid interruptions. Employ large rheophores well wetted and gently *slid on* to the desired spot. The pain increases at first but diminishes after a few minutes. The action is analogous to and equal to the mechanical percussion (Gowers). Local application of chloroform by cataphoresis[1] is said to be very valauble.

Radical or operative measures may sometimes be necessary. Acupuncture, nerve stretching, or neurectomy have all been tried with more or (mostly) with less success. The removal of a portion of the nerve certainly relieves for a time, but if the nerve joins again, the pain generally returns, sometimes worse than before, and the risk of trophic lesions is great. The injection of alcohol into the sheath of a sensory nerve by means of a hypodermic syringe has been lately attended with considerable success, more particularly in cases of trifacial neuralgia.[2]

Trifacial Neuralgia (Trigeminal Neuralgia, Neuralgia of the Fifth Nerve, Tic Douloureux, Prosopalgia, " Face-Ache ").—Of all the sensory nerves the fifth is the most frequent seat of neuralgia, possibly because of its exposed position and tortuous course through bony passages. The sensory branches of the fifth nerve are distributed to the skin of one entire half of the face, the mucous membrane of the mouth, nasal cavities, conjunctiva and the frontal sinuses ; its other functions are dealt with elsewhere (§ 615). The pain may involve any or all of the sensory branches in varying proportions, and may radiate in different directions, and there is usually a considerable degree of tenderness, many tender spots, sometimes redness and œdema. In cases of a purely reflex nature this is all. But irritative lesions of the Gasserian ganglion, or parts of the nerve in front of this, are indicated also by various vaso-motor and trophic troubles. In cases of moderate severity vascularity and œdema of the face, watering of the eye, sweating, and sometimes muscular twitchings and flashes of light may be observed ; but acute or severe

[1] This is a method supposed to cause the applications to penetrate the skin, and to have a selective action on the nerve fibres. The method is to soak the sponge of the positive pole in, say, a mixture of chloroform and alcohol, equal parts, apply it over the painful spot, and place the negative pole over the nucha. The current should be just as strong as the patient can bear comfortably, and may with advantage be reversed every few minutes.

[2] Dr. J. Wilfred Harris, the *Lancet*, 1909, vol. i.

irritative lesions of the fifth result in herpes of the face or scalp, sloughing of the cornea, pan-ophthalmitis, and even swelling and ulceration of the gums. In cases running a more chronic course, atrophy of the skin (glossy skin) and subcutaneous tissue of the face, and loosening of the teeth may be observed.

The *Causes* may be local, reflex, or constitutional. (*a*) If any of the irritative signs just mentioned be present, we must seek carefully for some *local* irritative or inflammatory lesion affecting the nerve trunk or Gasserian ganglion. That this nerve may be the seat of definite inflammatory lesions is certain from the fact that similar signs to these appear after an irritative lesion of a nerve-trunk artificially or experimentally produced. Such inflammatory lesions may be due to deeply-seated bone disease—*e.g.*, from syphilis or tubercle. (*b*) The cause of trifacial neuralgia of moderate severity is, however, more often some *reflex* condition, especially that dependent on affections of the teeth. Decay, the presence of stumps, periosteal disease, or affections of the gums, or an inflamed pulp, should all be suspected. Sometimes these affections are obvious, but the last-named (disease of the pulp) is not always so, and may readily be overlooked for a long period of time, as, for instance, when a late wisdom tooth presses on the second molar from below.[1] The best sign of pulp irritation is hypersensitiveness to heat and cold in the mouth alternately. Distant organs may also be a cause in a reflex manner, and these should always be carefully examined in unexplained cases. A good many years ago Anstey recorded a case of neuralgia of the fifth produced by injury to the ulnar, and another case due to injury to the occipital nerve. (*c*) Any of the *constitutional* causes previously mentioned may play their part. Ague is a frequent causal agent in the tropics, where it has earned for the neuralgia of the supra-orbital branch the term " brow-ague."

Epileptiform Neuralgia (Trousseau) is a violent and intractable form of neuralgia of the fifth, attacking people usually beyond middle life, and consisting of a succession of attacks of acute darting pain. Its etiology does not materially differ from that of trifacial neuralgia.

The *Treatment* of neuralgia is given on p. 877.

Sciatica is pain in the course of the sciatic nerve. It is difficult to demarcate sharply between neuralgia and neuritis, and every stage between these occurs in sciatica. Pressure and movement increase the pain, and therefore walking and sitting are difficult. The nerve trunk is tender, and painful points can often be elicited—*e.g.*, near the posterior iliac spine midway behind the great trochanter and the tuber ischii, and below the head of the fibula. The sciatic phenomenon aids the diagnosis. It consists of flexing the leg upon the hip-joint when the patient is in the recumbent posture. Owing to the stretching of the sciatic nerve this process causes pain, which disappears on flexing the leg on the thigh. Symptoms occasionally noticed in chronic cases are fibrillary tremor and slight atrophy. When anæsthesia, atrophy, and partial R. D. are found, the case is one of neuritis, and pressure within the pelvis should be suspected ; and if on the left side carcinoma of the sigmoid flexure. Hip-joint disease does not cause pain limited to the sciatic nerve. In spinal disease the pain is usually bilateral.

Prognosis.—Sciatica may recover in a few weeks, or it may last for years, especially in the aged. Much depends on the early treatment. It is apt to recur. Complications such as herpes and other cutaneous eruptions are rare. Occasionally the disease ascends the nerve to the cord, with corresponding symptoms.

Etiology.—Sciatica affects men more than women in the proportion of 5 to 1. It occurs mostly between thirty and fifty, and is unknown under fifteen. Rheumatism and gout attacking the sheath of the nerve are the chief predisposing causes. Anæmic neurotic people of a rheumatic diathesis also suffer. Exposure to cold is the usual exciting cause. Tumours, especially cancer of the rectum or sigmoid flexure, extension from hip disease, peri- or para-metritis, and a loaded rectum, are the chief local causes of sciatica by pressure within the pelvis.

Treatment.—In the acute stage rest is all important, and particularly the avoidance of movements (such as bending the thigh) which stretch the sciatic nerve.

[1] Tomes's " Dental Surgery," 4th edition, p. 539.

Heat is the next most important palliative agent. Mustard plasters and blisters may perhaps abort the attack (see also Treatment of Neuralgia). In the later stages and for chronic cases galvanism, nerve-stretching, counter-irritants, acupuncture, with or without cocaine or morphia injections, may be tried. The nerve may be stretched daily in chronic cases by forcible bending of the thigh on the abdomen, the foot being flexed all the time. Any rheumatic or other diathesis present must be treated, and remedies which I have frequently employed with great benefit are guaiacum, sulphur, or chian turpentine (see also Neuralgia, *supra*).

In **Brachial Neuritis** (inflammation of the nerves of the arm), the main symptom is severe neuralgic pain shooting down the nerves of the arm. There may also be paresis of a greater or less degree, muscular wasting, and the other symptoms mentioned under Neuritis (§ 564, single nerve). It is met with in an acute and more or less chronic form, but in any case it is a severe and often intractable condition. Only one arm is usually affected, and the condition may have to be diagnosed from an occupation neurosis by the circumstance under which it occurs.

Any of the *Causes* of neuritis may be in operation, but particularly the toxic or constitutional conditions. I have met with a certain number of cases of brachial neuritis in which the most careful investigation has failed to discover other determining cause than *exposure to cold*, though some of these patients have been rheumatic or gouty subjects.

In regard to the *Treatment* of brachial neuritis there are two means which generally effect considerable benefit in cases of brachial and other varieties of neuritis due to toxic causes.

(1) The application of Paquelin's thermo-cautery in a white-hot condition rapidly dotted down the painful limb. If the point be made only red, it sticks to the skin and causes great pain, but if white-hot, it really causes very little inconvenience, and gives so much relief in these painful cases that the patient asks for more.

(2) The application of galvanism may be of great value to relieve the pain and improve the nutrition of the muscles (see also Neuralgia, *supra*).

Neuralgia may affect any of the **other nerves** (sensory or mixed) in the body besides the three types specially referred to above. The chief causes of pain in the various situations will now be briefly mentioned. Head's sensory areas may be consulted in such cases.

Pains in the Limbs have been discussed in § 422.

Intercostal Neuralgia of an inveterate kind frequently precedes, accompanies or succeeds zoster (shingles), especially in old people. Sometimes it is due to pressure of a tumour on the *spinal nerves*, meningeal thickening, or vertebral disease. If the pain be persistent in a male over thirty years of age, *aneurysm of the descending aorta* may be suspected. The pain may be on one or both sides, and often shoots down the left arm. As in other neuralgiæ, it may be due to a *chill* or any of the other causes mentioned above.

Neuralgia in the Chest is a common accompaniment of affections of the *pleura* and *pericardium*, but is rare in those of the lungs and heart. However, in cases of cardiac valvular disease and disease of the aorta, the pain may occasionally be of a very aggravated character, and may be attended by a sense of suffocation and impending death (angina pectoris).

Mammary and Infra-Mammary Neuralgia are generally the result of *hysteria*, but the organ should be carefully examined for *adenoma* in the young, *carcinoma* in the aged. The word "agony" used by a female patient in connection with the pain is sometimes an aid to diagnosis. Pain in the breast is sometimes reflex from uterine disorder, functional or organic.

Gastric Neuralgia is somewhat rare. It is an intermittent pain in the region of the stomach, attended sometimes by nausea and even vomiting, relieved rather than aggravated by food or pressure, unattended by loss of flesh or strength, the digestion being good in the intervals. Gastric pain is one of the four classical symptoms of *Addison's disease*, the other three being progressive emaciation, vomiting, and pigmentation. *Tabes dorsalis* is attended by attacks of severe pain

in the stomach (gastric crises). Hydrocyanic acid is worth trying in all intestinal neuralgiæ.

Abdominal Pains are described in Chapter IX.

Neuralgic Pain in the Neck is suggestive of stiff-neck (" rheumatic "), spinal caries, or other local affection. Pain in the occiput and back of the neck is frequent in gastric and hepatic disorders. Occipito-cervical neuralgia is a somewhat rare form of idiopathic neuralgia, probably of reflex origin.

Spinal Neuralgia may arise from *spinal caries* or some other disease of the spine or theca pressing on the posterior roots. It is generally increased by exertion, and there is a characteristic history. *Meningeal affections* give rise to paroxysms of pain in the region of the spinal nerve roots, shooting round to the front on both sides, or down the limbs, accompanied by great tenderness on movement, and perhaps anæsthesia.

Neuralgia of the dorsal spine is a very frequent and troublesome symptom in *neurasthenia* and *hysteria*, in which cases more tenderness may be produced by light touches than by deep pressure, and movement may not aggravate it. Similar to the foregoing is the pain in the spine, which forms one of the most intractable results of *railway accidents* (railway spine). It is usually accompanied by tenderness, increased by sitting, or by the vibration of a train. In *dyspepsia* or *gastric ulcer* pain is often referred to a tender spot in the mid-dorsal region. A constant grinding or boring pain in the dorsal region, shooting, perhaps, round to the side and front in a male adult, is always suspicious of *aneurysm*. In the young and delicate pain in the back may be due to *laxity of ligaments*. Under these circumstances it only comes on after sitting or standing upright, and leads to stooping. It is best treated by carefully regulated gymnastic exercises.

Lumbar Neuralgia may herald variola, erysipelas, or influenza, or arise reflexly from uterine, renal, and other visceral diseases. Lumbago is described elsewhere (§ 443).

§ **605. Migraine** (Synonyms: Megrim, Sick Headache, Nervous Headache, Paroxysmal Hemicrania) may be defined as a headache having a paroxysmal character, and running a more or less definite course, occurring at intervals over a long period of time, generally ushered in by malaise, and frequently associated with and relieved by nausea and vomiting.

In the *Symptoms* there are usually two stages. The first stage is attended by some transitory disorder of sensation, such, for instance, as bright spots or dark figures or lines before the eyes (scintillating scotoma). In some individuals other sensations are complained of, such as a feeling of chilliness or of cold feet, or of mental depression, with a dread of impending evil, or restlessness, or pins and needles in the limbs. This stage may last from five to thirty minutes, or longer, and is then succeeded by the second stage—that of headache—which is usually very severe, and lasts from a few hours to two or three days. It generally commences in one spot, and gradually involves one half or the whole of the head. It is usually terminated by a feeling of nausea and actual vomiting, after which the headache passes away. It is distinguished from other varieties of headache by its paroxysmal character, periodic recurrence, its definite march, and by its usually being preceded by sensory disturbances and followed by vomiting.

Causes.—Megrim is an eminently hereditary disorder. It is, like most other paroxysmal neuroses, more frequent in the female, affects early and middle life chiefly, especially women at the climacteric. Attacks are certainly predisposed to by any general want of tone in the system, and determined by anything of a depressing or exhausting nature, physical or mental, such as grief, anxiety, or bodily fatigue, improper food, or impure air.

Prognosis.—Megrim usually starts soon after puberty, and the attacks recur until about fifty years of age, when they gradually become less frequent. It is never fatal, but is very often of such severity as to completely prevent the person following her avocation during the two or three days of an attack.

Treatment.—(1) For the attacks.—If slight, a cup of strong coffee, or a walk, or drive, or cheerful companionship, will help to stave it off. But if severe, the

patient must be kept in bed, and perfectly quiet, in a darkened room, with nothing but iced milk to drink. Bromide, 10 grains every hour with a little sal volatile, may be combined with phenacetin, antipyrin, and many of the other drugs mentioned under Neuralgia. In some cases chloride of ammonium, in doses of 15 grains, or guarana powder relieves. If these means fail, any of the following may be tried: caffein, cannabis Indica, croton, chloral, gelsemium, and amyl nitrate. The latter inhaled often gives wonderful relief, and strongly supports the theory of vaso-motor origin. That it fails in some cases is readily understood, because the poison, whatever it may be, is still circulating in the blood. (2) Between the attacks the mode of life should be regulated so as to avoid any known exciting condition. And as regards food, there are three opposite modes of treatment. One is to diminish the diet as much as possible, and especially in its nitrogenous constituents; the other, with which I have met with more success, is to give a liberal dietary, with copious libations of milk, in addition to the ordinary diet, and two glasses of old Burgundy with each of the two chief meals. Another method, which has also been attended by much success, is that recommended by Dr. Francis Hare—namely, limitation of the carbohydrates. Any reflex cause of irritation—*e.g.* eye-strain—must be searched for and removed. Salicylate of soda, in view of Haig's researches, which certainly have much to support them, is a valuable remedy, and should be taken regularly. Morning and evening glasses of hot water to purify the blood, combined with an occasional purge, are useful. Tonics, as a rule, are of very little use (see also Neuralgia).

Cutaneous Sensation.

§ **606. Disorders of Cutaneous Sensation** are rare as substantive affections apart from motor defects, excepting in hysterical cases. Moreover, when motion and sensation are simultaneously affected, as after division of a mixed nerve, the loss of sensation is less and is recovered from much more quickly than the loss of motion. Symptoms referable to cutaneous sensation may be grouped into Hemianæsthesia; Anæsthesia of the lower half of the body; Localised Anæsthesia; Hyperæsthesia; Paræsthesia and perverted or subjective sensation. Syringomyelia may be appropriately described in this place.

The Clinical Investigation is given on p. 739. Time and patience are required to elicit the precise nature of the defect and its boundaries. All three kinds of sensation may be lost; tactile and painful generally go together, thermal may be lost alone. Muscle sense is not connected with the skin; it is the sense of position or degree of contraction of a muscle.

Hemianæsthesia, or loss of tactile sensation in one half of the body, may be due to either functional or organic causes, the former being far the most frequent.

1. In **Hysteria,** anæsthesia, and particularly hemianæsthesia is such a frequent symptom as to form one of the hysterical stigmata. Hysterica hemianæsthesia has special characteristics of its own, which enable one to recognise the condition from these alone. (i.) In its typical form it is complete, more absolute than any other hemianæsthesia, and involves not only the skin and mucous membranes, but also the joints and muscles. (ii.) It is sensorial—*i.e.*, involving the special senses, as well as common sensation. Vision alone may be partially retained on the paralysed side, but in its characteristic form this sense also is altered by a contracture of the field of vision on both sides, more marked on the anæsthetic side. There is also an inversion of the colour fields.[1] (iii.) The hemianæsthesia varies from day to day, and perhaps from hour to hour, and may shift suddenly to the

[1] *E.g.*, a case recorded in the "St. Thomas's Hospital Reports," 1888.

opposite side after some emotional shock. It may be accompanied by paresis of varying degree on the same or the opposite side, or the other side may be hyperæsthetic. (iv.) The condition may also be recognised by the presence of the other three hysterical stigmata—viz., globus, fainting or nervous attacks, and the *ovarie*. This latter consists of a hyperæsthetic patch in the inguinal region, which is known as the ovarian tenderness, though it is wholly independent of the ovary, for it is present in the male.

2. **Organic Lesions of the Brain or Spinal Cord** may also give rise to hemianæsthesia, though it is usually less profound than the preceding, and is accompanied by paralysis of the same side.

(*a*) Hemianæsthesia, sensory and sensorial, due to a very extensive organic lesion of the *cortex*, is recognised by convulsions and paralysis having a distribution corresponding to the position of the lesion.

(*b*) Hemianæsthesia, sensory and sensorial, of a more complete kind than the preceding, may be due to a lesion at the posterior end of the *internal capsule* (sensory crossway), and is known by being accompanied by slight hemiparesis, the leg being more involved than the arm, because the leg fibres pass down the posterior part of the motor tract, adjacent to the sensory crossway.

(*c*) Hemianæsthesia without loss of sight or smell may arise from a focal lesion below the internal capsule, but above the pons—a small hæmorrhage into the *tegmentum of the crus*, for instance.

(*d*) Crossed hemianæsthesia (one side of body, other side of face) may arise from a lesion in the *fillet* or *pons*, in which case it is attended always by crossed hemiplegia. Crossed hemianæsthesia may also arise from damage to one of the *peduncles of the cerebellum*, especially the middle one, and is then probably accompanied by paresis, staggering gait, vertigo, etc.

Anæsthesia of the lower half of the body or loss of tactile sensation of the legs and lower part of the trunk is very rarely functional. It indicates practically in all such cases an organic lesion of the cord, and is accompanied by paralysis. The segment of the cord involved may be localised by the upper limit of the anæsthesia. It will also be remembered that in myelitis a narrow band of hyperæsthesia is a good indication of the upper limit of the mischief, which may also be localised by means of Head's algesic areas (§ 558).

Brown-Séquard Paralysis.—A stab or bullet wound strictly limited to *one half* of the cord produces on the *opposite* side loss of tactile (partly), and loss of pain- and temperature-senses ; on the *same* side loss of power, sense of tactile position and discrimination (by compasses, Head) and vasomotor tone. At the level of the lesion on the same side is a band of anæsthesia with a band of hyperæsthesia above it.

Localised or Limited Anæsthesia.—(1) *Hysteria* is the commonest cause of complete SEGMENTAL anæsthesia of one limb—*i.e.*, one which is limited by a horizontal ring round the level of a joint (wrist, elbow, knee, or shoulder). Such an anæsthesia is always cerebral in origin.

(2) *Tabes dorsalis* is a disease which may be suspected when there are small PATCHES or streaks of lost or modified sensation. All manner of sensory variations are met with in the preataxic stage, such as anæsthesia of the soles of the feet or along the ulnar margin of the wrist, delayed sensation, a zone of an- or hyperæsthesia round the body, or transferred sensation (allocheiria).

(3) *Syringomyelia* is a spinal lesion characterised by muscular atrophy and THERMAL ANÆSTHESIA of one or more extremities, not infrequently the arms, tactile and pain sense being retained (§ 607).

(4) Lesions affecting the *spinal nerve roots*—*e.g.*, spinal caries or tumours—produce PATCHES of anæsthesia along the limbs, and are accompanied by severe neuralgic pain. A transverse lesion of a *spinal segment* not involving the spinal roots is unaccompanied by pain. The distribution of the anæsthesia is given in the figures in § 558, and it differs from that due to peripheral nerve lesions.

(5) Peripheral neuritis and destructive lesions of the *peripheral nerves* also produce similar patches of anæsthesia in their areas of distribution (figs. § 569) ;

but it should be remembered that loss of sensation is produced much less readily than loss of motion. In multiple peripheral neuritis there is very often analgesia with preservation of the tactile sense quite at the ends of the limbs. In lesions of the nerve trunks (neuritis, leprosy, etc.) the distribution of the anæsthesia corresponds only roughly with the distribution of the affected nerve, the adjacent areas overlapping considerably. But in lesions of a spinal root or of a spinal segment the area of anæsthesia does not correspond with the distribution of the nerves connected with that root, and the margins of the analgesic areas are abrupt and definite (see Head's Areas, § 558).

Anæsthesia Dolorosa is a condition of anæsthesia attended by neuralgic pains, present sometimes in peripheral neuritis, local or multiple.

Hyperæsthesia.—Much that has been said about anæsthesia applies also to hyperæsthesia, for irritative lesions will produce this latter condition where destructive lesions produce anæsthesia.

General Hyperæsthesia occurs in hysteria, hydrophobia, and rickets. It is also found combined with the neuralgic pains in spinal meningitis (especially the acute form).

Localised Hyperæsthesia.—In alcoholic or other multiple neuritis the muscles, nerves, and skin are often acutely tender. The band of hyperæsthesia in myelitis associated with girdle pain has already been referred to. It is a marked feature in cancer of the vertebræ and in acute spinal caries.

In neuralgia *hyperæsthetic spots* on the skin are met with in the distribution of the affected nerve.

Tender areas may be associated with irritation of certain spinal root zones or with visceral disease (compare § 558, Head's Sensation Areas). But perhaps the most common cause of hyperæsthetic spots is hysteria. Such spots are found very constantly in hysterical cases in the inguinal (so-called ovarian), inframammary and sometimes in other regions. If pressure over these spots gives rise to hysterical attacks of any kind, they are called hysterogenic zones.

Paræsthesia (perverted sensation) in its strictly etymological sense means the perverted perception of an external stimulus. Used in this sense it is met with either in the form of (i.) *transferred* or misplaced sensations (allocheiria), when the patient locates a tactile sensation to the opposite side or some other part of the body ; or (ii.) *delayed* sensation. Both these alterations are met with in tabes dorsalis, and they may also be present in hysteria and sometimes in syringomyelia. *Polyæsthesia* indicates an apparent multiplication of the parts touched. But the term " paræsthesia " is also frequently used for various subjective sensations independent of external stimuli, such, for instance, as the numbness, tingling, creeping cold sensations which are met with in the early stages of peripheral neuritis.

Neurasthenia is frequently accompanied by a number of perverted sensations, such as burning, crawling, throbbing, etc. In this disease also we meet with those indescribable *somatic sensations*, in which, for example, the patient feels as if he were " sinking through the earth," " treading on air," or a general feeling of bodily discomfort, such as words fail to describe.

Acroparæsthesia also comes under this head. It is a symptom but little referred to in books, but is common enough in out-patient practice. The patients complain of a feeling of coldness, pricking, smarting, pins and needles, and often severe pains in the hands. It is a symptom of many nervous and gastric disorders, and may go on to erythromelalgia (§ 429).

§ **607. Syringomyelia and Intra-Medullary Tumours** (*i.e.*, within the spinal cord). —Tumours within the spinal cord consist mainly of syphilitic gummata, syringomyelia with or without gliomatosis, sarcoma, tubercle, and myxoma. They are less frequent than the external and extra-medullary tumours (see Compression Paraplegia, § 557), and give rise to a very different set of symptoms. They involve the grey matter and the central parts of the cord primarily, consequently paraplegia and pain are usually absent until late in these diseases, and only arise as a result of the secondary lateral sclerosis. *Syringomyelia* is an interesting condition

which has received considerable attention of late years. It consists essentially of a patency or dilatation of the central canal of the cord, the epithelial lining of which is prone to take on a gliomatous (small-celled ? sarcomatous) growth having a malignant tendency to spread upwards and downwards. The chief seat, and therefore presumably the origin of this new growth, is the cervical region, and consequently one or both arms exhibit the earliest symptoms.

The *Symptoms* of these conditions vary somewhat with the position and the part of the cord involved (Spinal Localisation, § 558), but the commonest position is the anterior part of the posterior columns in the cervical enlargement, and we meet, therefore, with three principal symptoms : (1) Loss of sensibility to thermic impressions, and sometimes to painful impressions, tactile sensations being sometimes retained, chiefly, but by no means always, limited to the hands and arms, is the most characteristic symptom, and is not known to occur so typically in any other disease. Thermic, painful, and tactile sensations are all translated simply as touch. (2) Muscular atrophy in one or both arms occurs as the tumour extends forwards, accompanied by R. D. (3) Later on there are paraplegic rigidity, increased reflexes and spasms due to descending sclerosis. In one case under my care there was atrophic flaccid paralysis of the legs, with greatly increased knee-jerks and ankle clonus. Diminished power of the limbs on one side with diminished sensation on the other (Brown-Séquard phenomenon) may be met with if the lesion extends laterally. Combined with the thermal anæsthesia and atrophy in the arms and the spastic paralysis in the legs is a number of other symptoms which may or may not be present, such as various trophic lesions of joints and bones. At other times there are vesicles, bullæ, extensive desquamation of the cuticle (in one case under my care the epidermis of the fingers was shed like a glove), ulcerations, or whitlows (which, coming on without pain, resemble the painless whitlows of Morvan's disease), all of which chiefly affect the upper extremities. When the lesion spreads to the medulla and pons the cranial nuclei are apt to become involved, causing nystagmus, deranged respiratory and cardiac movements, paralysis of the vocal cords, tongue, palate, or pharynx. Sudden death may ensue, as in the case just mentioned, from extension to the " nœud vital." These are the symptoms of tumours in the cervical region and of syringomyelia. In the other positions intramedullary tumours give rise to symptoms which depend on the column mainly involved.

The *Diagnosis* is difficult in the earlier stages. The pathognomonic symptoms of syringomyelia are thermal anæsthesia, combined with atrophic paralysis. Progressive muscular atrophy has no such impairment of sensation. In multiple peripheral neuritis the symptoms are more scattered. Hypertrophic cervical meningitis is characterised by severe pain, and the anæsthesia, if present, involves all forms of sensation.

The *Prognosis* depends upon the position and rate of advance of the disease. Syringomyelia usually runs a very slow but progressive course of many years unless the neoplasm extends rapidly upwards and causes death. Other cases in which apparently no gliomatosis occurs, and in which some joint[1] or other trophic manifestations are the only symptoms, may live to old age.

Causes.—Syringomyelia is probably due to a congenital condition of the cord, but the age at which the symptoms are first noticed is not often under fifteen, and very rarely over thirty. It is more frequent in males (two-thirds). The symptoms have often been noticed to follow an accident of some kind. The central canal (so-called) of the cord consists normally of a solid rod of epiblastic cells remaining from the grooved infolding of that stricture which develops into the spinal cord. Everything tends to prove that this remains patent in syringomyelia, and that the cavity enlarges and involves the adjacent trophic and sensory tracts, especially that of the temperature sense. In some cases the condition remains stationary, and the patient lives indefinitely ; in others gliomatosis occurs in the epiblastic cells (determined, perhaps, by a slight injury to the back), and the patient dies in

[1] As in a case exhibited by Mr. J. R. Lunn at the Clinical Society of London in London in 1899 or 1900

a year or two. Or syringomyelia may consist of multiple cavities in the cord, with or without gliomatosis, these affording a predisposition (Gowers says a necessary predisposing factor) to hæmorrhage into the cord.

The *Treatment* is purely symptomatic, though iodide may be tried.

GROUP VI. CRANIAL NERVES AND SPECIAL SENSES

§ **608.** The investigation of the cranial nerves and special senses is of great importance from the standpoint both of general medicine and neurology. Here is a tabular statement of their function.

TABLE OF CRANIAL NERVES AND THEIR FUNCTIONS.

Cranial Nerves.	*Functions.*
I. Olfactory nerve.	Smell.
II. Optic nerve.	Sight.
III. Motor oculi.	Supplies all the *muscles* of the eyeball (except the superior oblique and external rectus) and the levator palpebræ superioris ; also the sphincter pupillæ and ciliary muscle.
IV. Motor oculi.	Supplies the superior oblique ; turns the eye down and outwards.
VI. Motor oculi.	Supplies the external rectus ; turns the eye outwards.
V. Trigeminal nerve. First division, Ophthalmic.	*Sensory* to forehead and part of vertex, anterior part of nose to tip, upper eyelid and temple, eyeball and lachrymal gland. Contains dilator pupillæ fibres from sympathetic.
Second division, Superior maxillary.	*Sensory* to cheek, lower eyelid, side of nose and upper lip ; the upper teeth and gum ; lining membrane of nose, roof of mouth, soft palate, tonsils, and roof of pharynx. *Taste* of anterior two-thirds of tongue (through Meckel's ganglion by chorda tympani nerve). *Trophic* and vaso-motor fibres.
Third division, Inferior maxillary.	*Sensory* to lower part of face, lower lip, side of head, ear, tongue, lower teeth, gum, and inner side of cheek. *Motor* to masticatory muscles, temporal, masseter, pterygoids, anterior belly of digastric and mylo-hyoid, tensor tympani and ? tensor palati. *Taste* of anterior two-thirds of tongue (by chorda tympani from lingual nerve) ; of posterior one-third of tongue through glosso-pharyngeal nerve, Jacobson's nerve, and otic ganglion.
VII. Facial nerve.	*Motor* to all muscles of face and scalp (excepting levator palpebræ superioris), platysma, posterior belly of digastric, and stapedius muscle. It is joined by the chorda tympani (conveying taste fibres of anterior two-thirds of tongue from lingual branch of V. to Meckel's ganglion.
VIII. Auditory nerve.	Hearing.
IX. Glossopharyngeal nerve.	*Sensory* from pharynx. Collects taste fibres from posterior one-third of tongue, which ultimately join V. *Motor* to middle constrictor of pharynx and stylo-pharyngeus.
X. Vagus nerve.	*Motor* for soft palate (except tensor palati), pharynx and larynx (through accessory portion of XI.). *Motor* (involuntary) and *sensory* for heart, respiratory passages and abdominal viscera (through sympathetic ganglia).
XI. Spinal accessory nerve.	*Motor* to sterno-mastoid and trapezius. (Supplies vagus with motor fibres for larynx, pharynx, and palate.)
XII. Hypoglossal nerve.	*Motor* to tongue and depressors of hyoid bone.

§ **609.** The **First Nerve** is the olfactory bulb. To test the power of smell on one side close the other nostril with your finger and tell the patient to sniff some odour such as peppermint, clove oil, or onions (not ammonia, which is a stimulant to the second division of the fifth nerve in the nasal mucous membrane), and see if he can name the odour. It is convenient to have some little bottles containing these substances.

Anosmia is loss of smell ; *parosmia* is a perversion of smell. Loss of smell often depends on atrophic rhinitis or some other nasal disorder (see Chapter VII.). The first nerve alone may be damaged by concussion, the fine branches becoming torn against the cribriform plate of the ethmoid. This is extremely difficult to restore. In one case I was partially successful by localised cataphoresis with cocaine and strychnine. Smell may be lost after influenza, diphtheria, and other specific fevers, after thrombosis or embolism of the anterior cerebral artery, and with tabes dorsalis. Paralysis of the fifth may produce it by dryness of the nostrils. Hallucinations of smell—*i.e.*, odours recognised when no odorous substance is present—often form the aura of an epileptic fit.

§ **610.** The **Organ of Vision** is innervated mainly by four cranial nerves—the *second*, *third*, *fourth*, and *sixth*. The *fifth* and the cervical *sympathetic* are also concerned in its innervation. Careful examination of the eye is of the greatest importance in many diseases.

The symptoms which reveal disease of the eye may be arranged under six headings : Pain, Superficial Alterations, Acuteness of Vision (§ 611), Pupils, (§ 612), Ocular Movements (§ 613), Changes in the Fundi (§ 614). The reader should turn to the section dealing with the defect to which the patient's symptoms appear to belong.

The systematic examination of the eye consists of : I. Investigating pain if present ; II. noting any superficial alterations ; III. testing the acuteness of vision ; IV. examining the pupils ; V. the ocular movements ; and VI. the fundi. But in routine examination for medical purposes the acuteness of vision is left until the end.

I. **Pain in the Eyes** is not infrequently absent in ocular affections. Its commonest cause is some error of refraction (asthenopia—*i.e.*, eye-strain). Eye-strain may cause headache, or a dragging pain round the eyes, or blepharitis and blinking in children, or neuralgia of the fifth nerve. Glaucoma or any other cause of increased ocular tension may cause pain. And, as Tomes[1] has pointed out, dental disorders may not only give rise to reflex pain, but also to both functional and organic affections of the eyes. Among subjective sensations other than pain may be noted muscæ volitantes (black dots) and scintillating scotoma (zigzag lines). The former occur in anæmia, debility, or functional disorders of the liver, the latter in association with migraine.

II. **Superficial Alterations** mainly come within the province of the surgeon ; they are six in number. A good light and a lens are all that is necessary.

1. Proptosis is an undue prominence of the eyeballs, and is met with in exophthalmic goître, ophthalmoplegia externa, and in marked myopia. In one

[1] "A System of Dental Surgery," Charles S. Tomes, F.R.C.S., p. 572 *et seq.* London, 1897.

eye only it suggests orbital tumour or some intracranial lesion. To detect small degrees, look down over the top of the patient's head from behind. Recession of the balls occurs in paralysis of the cervical sympathetic, the other symptoms of which are contraction of the pupil on that side and loss of the cilio-spinal reflex (reflex dilatation of the pupil when the skin of the neck is pinched).

2. The Eyelids.—The eyelids are puffy in renal disease, cardiac dropsy, and the arsenical poisoning. Ptosis, or drooping of the upper eyelid is (i.) one of the symptoms of paralysis of the third nerve (*infra*). It may also be due to (ii.) a cortical lesion, without involvement of any other part of the third nerve ; (iii.) idiopathic muscular atrophy when the face is affected ; (iv.) myasthenia gravis ; (v.) hysteria ; (vi.) a congenital condition ; (vii.) Nothnagel has described a pseudoptosis, in which the eyelid gives the appearance of drooping, owing to recession of the eyeball in paralysis of the cervical sympathetic (§ 612). A diminution of the palpebral fissure may be due to recession of the eyeball (*ante*), or weakness of the levator palpebræ superioris. Blepharospasm is an involuntary clonic twitching of the eyelid (see Habit Spasm, etc.). Inability to close the eye is a notable feature in Bell's (facial) paralysis. It is also one of the earlier symptoms in infancy of the hereditary infantile amyotrophy described by Duchenne.

Deficient Falling of the Upper Eyelid when the patient looks rapidly down constitutes the so-called Von Graefe's sign in exophthalmic goître, but this symptom is only present in an advanced stage of the disease when other means of identification are not wanting. The same remark applies also to Stellwag's sign (the almost total absence of involuntary blinking), and to Mœbius's sign (a deficient power of convergence), which are occasional symptoms in Graves' disease.

The diseases of (3) the conjunctiva, (4) the cornea, (5) the iris, and (6) the alteration of tension are dealt with in surgical works.

§ **611. Defects of Vision** may consist of (1) defective sense of form or acuteness of vision, (2) alteration in the field of vision, (3) defective sense of colour.

(1) Acuteness of Vision implies the estimation of forms of objects. It may be roughly tested by asking the patient to count the number of fingers held up before him. The defect may be so great that the patient cannot perceive light from darkness. The eyes must be examined separately, as it is often found that defect of one eye has existed a long time without the patient being aware of it. If the external parts of the eye are normal, the media transparent, the ophthalmoscope reveals no disease, and there is defective acuity of vision, it is probable that the patient suffers from an error of refraction. Asthenopia (eye-strain) is due to continuous overaction of the internal muscles of the eye, and is manifested by ocular pain, ciliary congestion and headache. Excessive blinking and inability to use the eyes for long at a time are often the only symptoms in children. It may be due to overuse of the eyes in a bad light, but its commonest cause is some error of refraction.

Errors of Refraction.—For accurately testing the visual sense of form, the power of reading Snellen's types at a given distance (usually 6 metres, or 20 feet) is employed. The error of refraction is ascertained (after paralysing the iris by homatropin) by placing various lenses in the frame-spectacle before the eyes until it is found which of them completely corrects his error. Convex lenses are indicated by the sign +, concave by the sign −. The defect is measured by the focal length of the lens required to correct his error, and is now generally expressed in diopters, indicated by the sign D. A lens of one diopter has a focal length of 1 metre. Thus, a + 3 D. lens indicates a convex lens with a focal length of $\frac{1}{3}$ metre, being three times as strong as a lens of + 1 D. Retinoscopy is a more accurate method of testing refractive errors (below). In *myopia* (or near sight) the image is formed in front of the retina, and the patient cannot see distant objects clearly. In *hypermetropia* (or far sight) the image is formed on a plane behind the retina, and the patient cannot look at near objects for any length of time. Both may be due to either defective shape of the globe or to defective accommodation. Con-

cave lenses are used to correct myopia, and convex to correct hypermetropia. In *presbyopia* the rigidity of the lens renders it either difficult or impossible to accommodate for near objects ; it occurs in old people whose far vision may be remarkably good, though it is impossible for them to read or to see near objects distinctly without convex glasses. *Astigmatism* is a non-correspondence of the curve in the different meridians of the cornea. In *simple* astigmatism one meridian is normal, the other myopic or hypermetropic ; in *compound* astigmatism the error of the two meridians, though of the same kind, differs in degree ; in *mixed* astigmatism there is a myopic error in one meridian, and a hypermetropic error in the other meridian ; in *irregular astigmatism* the curves of the cornea vary even in the same meridian. Astigmatism is detected accurately by a skilled examination by retinoscopy, or with the astigmometer.

In **retinoscopy** the procedure is as for indirect ophthalmoscopy (§ 614), only without the large lens. No details of the fundus are visible in this way, but normally a perfectly red field is seen. If the bloodvessels are seen, then the patient's refraction is abnormal. *Errors of refraction* are revealed by tilting the mirror upwards and downwards and from side to side. A black shadow passes across the field in this procedure. In a normal eye the shadow moves very rapidly across a straight, defined edge. The greater the error of refraction the more slowly does the shadow move, and the more curved and hazy is its margin. If the edge of this shadow moves in the opposite direction to the mirror, the refraction is either normal, hypermetropic, or consists of less than one D. of myopia. If there be myopia of more than one D., the shadow moves in the same direction. Astigmatism is discovered by the shadows moving differently in opposite meridians. *Opacities* in the media may be thus detected as dark shadows upon the red field. The radiating streaks of commencing cataract or moving opacities in the vitreous may also be thus detected.

(2) The Field of Vision is the extent of the picture presented to the eye at any given moment. It may be roughly tested by instructing the patient to cover one eye and look fixedly at the tip of your nose at a distance of about 2 feet. Then hold up one hand on each side of you in the same vertical plane as your face, and bring it gradually towards your nose, asking the patient to cry " stop " the moment it comes into his view. Repeat the same procedure below, above, and at the two sides, and in this way you will roughly ascertain in what part of his field the vision is defective. The dimensions of the visual field can be tested accurately only by the perimeter (below). *Scotoma* is a word used to indicate a spot of blindness or imperfect vision within an otherwise healthy field—*e.g.*, a central scotoma is a blind spot in the middle of the visual field.

The Perimeter.—The patient covers one eye and places the other cheek bone against the round knob of the vertical pillar ½ inch below the palpebral fissure. The patient must be educated to keep his eye steadily fixed on the spot opposite, while the operator moves a small piece of white paper (or coloured for testing colour vision) mounted on the stick provided along the movable semicircular metal band from periphery to centre. The position in which the patient can first see the paper (while looking fixedly all the time at the central spot) is then marked on the chart provided. With stupid patients this is a tedious operation, and without due care erroneous results may easily be obtained. The perception of colours in the peripheral field varies normally in extent with the different colours. Thus, from without inwards they are white, blue, yellow, red, green.

(3) Colour Vision may be tested by means of a collection of different coloured wools. The patient should be instructed either to (i.) identify and name different colours, or (ii.) to pick out all the pieces of wool which match, three or four pattern pieces being given to him at the outset.

Colour Blindness (achromatopsia) is a symptom in some diseases of the retina and optic atrophy, both primary and secondary. It is also present in tobacco and some other forms of amblyopia in their early stages (*infra*). Partial achromatopsia may be congenital. In some hysterical cases the colour defect takes the form of dyschromatopsia, where the fields for different colours are inverted, the field for

red being larger than that for blue, the reverse of normal.[1] Charcot,[2] who was the first to describe this condition, regarded this as a special and quite distinctive feature of hysterical achromatopsia. *Coloured Vision* (chromatopsia) is an occasional symptom after the extraction of cataract in aged persons, and in exhausted states of the nervous system. I have met with it in some cases of tabes dorsalis. It is also occasionally a symptom of glaucoma ; red vision (erythropsia) is the most common. Yellow vision is a symptom of santonin poisoning.

The **Causes of Defective Vision** without any very obvious ocular changes may be considered under Amblyopia (bilateral and unilateral), hemianopsia, and night-blindness. The defective vision due to errors of refraction has already been dealt with.

Amblyopia is diminished vision, Amaurosis loss of vision, without discoverable changes in the fundi oculorum or error of refraction. It is obvious that amblyopia may be due either to some functional disturbance of the visual apparatus, or to some gross lesion of the brain or paths of vision behind the retina or optic discs.

Bilateral Amblyopia may arise under various toxic conditions and functional states, as follows :

1. *Tobacco Amblyopia* arises sometimes in hard smokers of over 3 or 4 ounces per week, or in debilitated persons from a much less quantity. The patient first complains of defective vision in bright light ; he sees better at dusk than at noon. The defect is slowly progressive, becoming most marked in the *central* part of the field, and there is central colour scotoma. At first there may be no changes in the fundi, then the discs become slightly congested in the earlier stages, and pale and atrophied, especially on the temporal side, in the later. In several cases which I have seen a defective vision was the earliest symptom to attract the patient's notice. Tobacco amblyopia, it should be borne in mind, is very occasionally met with in females.

2. *Toxic* amblyopia is caused also by other poisons such as uræmia, diabetes, large doses of quinine, bisulphide of carbon in indiarubber manufacture, iodoform and dinitro-benzol. Little in the way of treatment can be done for such cases, unless they are seen early, when absolute cessation of the cause and functional rest to the structures involved may lead to recovery.

3. Various *Structural Diseases of the Retina and Optic Nerve* in their incipient stages, when attended by slight alterations that none but a very skilled observer can detect, may, nevertheless, be attended by an alteration of the visual fields and of the acuteness of vision. This is sometimes the case with insidious papillitis, and generally with primary optic atrophy. The double primary atrophy which so often accompanies tabes dorsalis is a marked instance of this ; it may precede the ataxy and other symptoms of tabes by many years. I have seen several such cases, where the amblyopia was regarded as functional, and its spinal origin overlooked for a long time. Primary atrophy more rarely accompanies disseminated sclerosis and general paralysis of the insane. In all these cases the field of vision is diminished, either eccentrically or concentrically, and colour vision also is reduced.

4. *Hysterical Amblyopia* is a very characteristic affection, and consists of a concentric retraction of both fields, though most extreme on the same side as the hemianæsthesia, by which it is almost invariably accompanied. The colour vision is also affected in a characteristic manner (see *ante*).

5. *Neurasthenic* or *Retinal Asthenopia*, a condition in which the patient ceases to be able to see after exercising the visual function for a time, is also unattended by changes in the fundi. The field of vision is also contracted, and temporary scotomata appear on looking at objects.

6. *Sudden and Copious Hæmorrhage—e.g.*, from the stomach, bowels, uterus, etc.—may produce amblyopia from deficient blood supply to the eyes, and pos-

[1] A case is recorded in " St. Thomas's Hospital Reports," 1888.

[2] " Clinical Lectures on Diseases of the Nervous System," vol. iii. ; New Syd. Soc. Trans.

sibly to the visual cortical centres. If it does not shortly clear up, optic neuritis is to be apprehended.

7. Exposure to *blinding sunlight* or electric light may cause acute amblyopia.

Mononuclear Amblyopia.—It must be borne in mind that defective vision in one eye may be overlooked for many years, the patient thinking that he suddenly becomes blind when he has by chance shut the good eye on one occasion.

1. The remarks made above relative to *optic neuritis* and *atrophy* apply here. Local diseases of the choroid or retina in the macular region produce central amblyopia or central scotoma, often of one eye only.

2. *Squint* in some cases produces a form of congenital amblyopia in childhood, which is only very slowly, if ever, remedied. It happens thus : squint results in double vision, and in order to see clearly the child unconsciously gets into the habit of neglecting the image produced by one of the two eyes, usually the squinting one. After long habit this " suppression of image " becomes permanent. It would, perhaps, be more correct to say that the mind has lost the power of perceiving the image formed in that eye, and can only be slowly educated up to it.

3. *Astigmatism, Myopia or Hypermetropia* in a very high degree (where a clear image may never have been formed on the retina) leads to defective vision, so that when a full correction of error of refraction takes place, it is generally found that the patient cannot see. This probably arises from the fact that the retina has never received the necessary practice or education for the appreciation of true images of objects.

4. *Chill.*—A few cases appear to be produced by exposure to cold. Occurring for the most part in young adults, the onset is fairly rapid, and accompanied by neuralgic pains in the same side of the head. The affection is not usually serious, and recovers under the same means as those employed for paralysis of the facial nerve under like circumstances.

5. *Reflex Amblyopia* is certainly not common. It generally occurs only in one eye, and is associated with a carious tooth. Sir Thomas Watson[1] mentioned cases, and quite a number of cases have been collected by Tomes,[2] in which the blindness was cured by the adequate treatment of a decayed tooth, and returned when this again gave trouble.

Hemianopsia (hemianopia, hemiopia) means loss of sight in one half of the visual field in both eyes, unaccompanied by changes in retina or disc. Four kinds are described, but practically the first is the only one met with, and that is sufficiently rare.

Homonymous or lateral *Hemianopsia* means loss of vision of the two left or right halves (the corresponding halves) of the visual fields of each eye. Right lateral hemianopsia means abolition of the right halves of the patient's visual fields. This corresponds to the temporal half of the left and the nasal half of the right retina (see Fig. 164). Apart from its occasional occurrence in migraine, it is due to a gross central lesion situated in some part of the visual path behind the chiasma : (i.) in the optic tract ; or (ii.) behind the corpora quadrigemina—that is to say, the hinder end of the internal capsule or the white fibres of the occipital lobe ; or (iii.) in the visual centres of the cortex, situated in the occipital lobe. By employing Wernicke's test (§ 612) the first may be excluded. For the rest, the precise position and character of the lesion can only be diagnosed by the accompanying symptoms. Cortical lesions are usually associated with some form of aphasia, or with paralysis due to some cerebral lesion. Lesions affecting the optic tract are syphilitic gummata or meningitis, tubercle, and neoplasmata, softening and hæmorrhage (rare). Tumours in the optic thalamus, lenticular nucleus, and temporo-sphenoidal lobe may extend to or press upon the optic tract. Other associated basal paralyses may aid the diagnosis.

The remaining variety (*Heteronymous* Hemianopsia) is very rare. It may occur on one side only. *Temporal Hemianopsia* is the loss of the outer half of each field, due to a blindness of the inner half of each retina. In the only cases on record it

[1] " Lectures on the Principles and Practice of Physic," 4th edition.

[2] " A System of Dental Surgery," 4th edition, p. 574 *et seq.*

has arisen from damage to the middle of the optic chiasma, or, rather, just behind or just in front of the middle part. Pressure of a tumour, distended third ventricle, or localised basal meningitis have produced it. It has been met with in some cases of acromegaly. Recurrent attacks have been produced by gummata. *Nasal Hemianopsia* is still rarer. It is usually unilateral, due to a lesion situated in one optic tract just as it leaves the chiasma. *Altitudinal Hemianopsia* means loss of the upper or lower part of the fields, and if bilateral, is due to a lesion involving the upper or lower part of the chiasma, optic neuritis, or symmetrical cortical lesions.

Night-Blindness (nyctalopia) is defective vision in dim lights. It is a feature of retinitis pigmentosa, syphilitic retinitis, and it may be congenital (without fundal changes). Eccentric contraction of the visual field supervenes, and finally complete blindness. Acute night-blindness may attack those with defective general health who have been exposed to very strong sun or artificial light, and in these cases the prognosis is good.

§ 612. Defects in the Pupils.—The iris comprises dilator fibres (supplied by the cervical sympathetic), and constrictor fibres (supplied by the third nerve); associated with these latter is the ciliary muscle, also supplied by the third nerve. Each of these may be paralysed separately, and paralysis of all three is known as *ophthalmoplegia interna*. The pupils must be tested in regard to their shape, size, equality, mobility to light, and mobility to accommodation. The patient should be placed opposite a good light.

The *shape* of one or other pupil may be irregular, and this may be due to old iritic adhesions or the result of previous iridectomy. Coloboma iridis is a congenital abnormality which sometimes exists without error in vision, consisting of a deficiency of the iris, usually in the lower part, generally on both sides.

The *mobility* of the pupils must be tested both by the *light* and *accommodation* reflex (see below).

The average *size* of the pupils varies in healthy persons with the light and irritability of the retina and optic nerve.

When two pupils are *unequal* in size, and it is desired to ascertain which is the normal one of the two, it may generally be inferred that the immobile pupil is the abnormal one. Slight inequalities between the pupils may also be observed in health. Inequality of the pupils of the two eyes is a frequent symptom in general paralysis of the insane, and an occasional one in tabes and migraine. It may exist obviously in any condition causing contraction or dilatation of the pupil on one side only (see below), as, for instance, in paralysis of one third nerve causing dilatation or paralysis of the cervical sympathetic causing contraction.

The Mobility of the Pupils to Light.—In testing the light reflex, *both* eyes should be covered for half a minute, and each uncovered in turn opposite to a bright light, which makes the pupils contract. In a good light the iris can sometimes be observed to contract and dilate rhythmically, a phenomenon which is called *hippus*. Its clinical significance is unknown. *The pupil light reflex* depends on the integrity of the retina and the following tract (Fig. 164), the optic nerve (*o*), the chiasma (*c*), optic tract (*t*), to the corpora quadrigemina (*cq*). These last-named nuclei (*cq*) are connected, by means of Maynert's fibres (*m*), with the nuclei of the third nerves (III.) situated in the floor of the aqueduct of Sylvius. The fibres of the third nerve, through the long or short ciliary branches, conduct contracting impulses to the sphincter iridis.

Loss of Light Reflex (light iridoplegia) may be produced by a lesion situated anywhere in these afferent or efferent tracts. (i.) In tabes the pupils may be unequal, unduly contracted, or fail to react to light or accommodation, but the change most commonly met with is the Argyll-Robertson pupil, which is Loss of reflex to Light, though Acting to Accommodation. This phenomenon, met with occasionally in other conditions, indicates a partial damage, involving either of the optic tracts or Maynert's fibres. (ii.) Atrophy of both optic nerves. (iii.) De-

structive lesions of the third nerve or its nucleus. (iv.) General paralysis of the insane. The light reflex is preserved in blindness of central origin. In unilateral optic atrophy, when the sound side is uncovered, both pupils contract (consensual contraction), but the pupil on the affected side does not contract when the affected side alone is uncovered.

Wernicke's pupil reflex assists one to determine the seat of a lesion in a case of hemiopia. Hemiopia may be due to a lesion (i.) of the optic tract between the chiasma and corpora quadrigemina (see Fig. 164) ; (ii.) of the optic fibres between the *cq* and the occipital cortex ; or (iii.) of the occipital cortex. If the lesion be in the optic tract, the light reflex is lost, but if behind the *cq*, a beam of light thrown directly on the blind half of the retina by the concave mirror of the ophthalmoscope produces contraction of the pupils. A little study of Fig. 164 will make this apparent.

Mobility of the Pupils to Accommodation.—Whenever the eyes converge and the patient looks at a near object, the pupils contract. Ask the patient first to look at a distant object, and after observing the pupils, tell him suddenly to look at the tip of your finger, held 6 inches in front of his face. This is really a reaction to convergence of the eyes. Loss of power of accommodation depends on paralysis or weakness of the ciliary muscle (cycloplegia) usually associated with paralysis of the sphincter of the iris. Loss of mobility of the pupil to accommodation is one of the earliest and most common symptoms in (i.) diphtheritic paralysis, and occurs on both sides. It is also seen in (ii.) belladonna poisoning, and (iii.) occasionally tabes and G. P. I. It is preserved in the Argyll-Robertson pupil (*vide supra*).

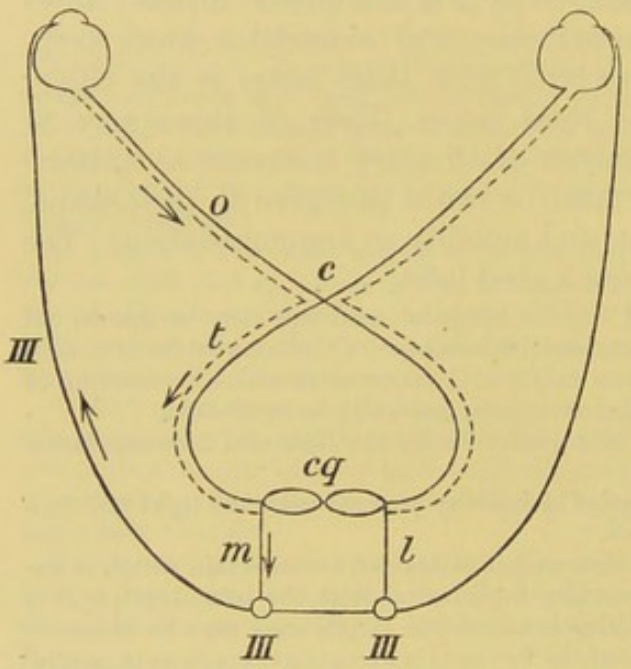

Fig. 164.—Diagram showing Reflex Arcs concerned in the Movements of the Pupil (Horizontal Plane). —*o*, optic nerves ; *c*, optic chiasma ; *t*, optic tract ; *cq*, corpora quadrigemina ; *III*, third nerves and nuclei ; *m* and *l*, Maynert's fibres communicating between the third nuclei and the corpora quadrigemina. (Vertical plane Fig. 168).

Ophthalmoplegia interna is paralysis of the three internal muscles of the eye (the sphincter and dilator pupillæ and the ciliary muscle). The pupils are of medium size, and immobile to light and accommodation. It constitutes an early symptom of diphtheritic and sometimes syphilitic paralysis, in either of which it is due to toxic affection of the oculo-motor nuclei. It is often associated with ophthalmoplegia externa.

Cilio-Spinal or Skin Reflex is the dilatation of the pupil when the skin of the neck on one side of a healthy person is stimulated by stroking, pinching, or faradisation, and is due to the stimulation of the dilator fibres contained in the cervical sympathetic (see Fig. 165). The loss of this skin reflex may be observed occasionally in tabes and G. P. I., but is chiefly met with as one of the symptoms of paralysis of the cervical sympathetic.

Contraction of both Pupils (myosis) may be caused by (i.) undue retinal irritability, which may ensue from overuse of the eyes, and from hypermetropia. (ii.) It is also present in congestion of the iris from any cause, which possibly explains their contraction in mitral regurgitation. (iii.) Various drugs, such as opium, tobacco, eserine, or pilocarpin. (iv.) Tabes dorsalis, which is often attended

also by loss of the light reflex (*supra*). (v.) General paralysis of the insane is sometimes attended by the same condition of pupils, the "pin-point" pupils—*i.e.*, strong contraction with sluggishness of movement. (vi.) Irritation of the third nerve as by pressure of tumour or the early stage of all inflammatory or meningeal affections, or irritation of the pupil contracting centre—*e.g.*, hæmorrhage into the pons causes a state of coma with contracted pupils, which may distinguish it from hæmorrhage into other situations, opium poisoning being excluded. (vii.) Paralysis of the cervical sympathetic.

§ 612*a*. **Iritis**, inflammation of the iris, is manifested by (1) immobility, loss of lustre, and exudation ; (2) pain (which may be absent in serous iritis), and dimness of vision ; (3) adhesions between the iris and anterior capsule, revealed under atropin ; (4) circumcorneal injection of the ciliary vessels, indicating hyperæmia of the ciliary body (irido-cyclitis). Care must be taken not to mistake (on account of 4) this disease for conjunctivitis, because the treatment suitable for the latter will make iritis worse.

The *Causes* of iritis are now known to be very numerous. Rheumatic, syphilitic, oral sepsis, and many other toxæmic and infective conditions hitherto unsuspected, may cause iritis. Syphilitic iritis is usually non-recurrent, but all the others are very liable to relapse. Chill, bright light, and injury are determining causes.

The *Treatment* consists of regular application of atropin drops (1 per cent.), constant dry heat, and leeches to the temple. For chronic iritis blisters behind the ear, and dionin (1 per cent.) drops once or twice daily may be tried. If total synechiæ form, iridectomy should be performed, to prevent glaucoma.

§ 612*b*. **Paralysis** of the **Cervical Sympathetic** causes contraction of the pupil, usually on one side only. The cervical sympathetic conveys *dilator fibres* to the iris, which, arising probably in the floor of the aqueduct, pass down the spinal cord to the cilio-spinal centre (Fig. 165), which is situated at the junction of the cervical and dorsal regions, thence outwards by the rami communicantes to join the sympathetic ganglia in the neck, and upwards along the carotid and cavernous plexuses to be finally distributed to the eyeball along with the ophthalmic division of the fifth. The symptoms of paralysis of the cervical sympathetic are (i.) contraction of the pupil of that side, with absence of dilatation of the pupil on shading the eye or the instillation of cocaine ; (ii.) abolition of the cilio-spinal reflex (*vide supra*) ; (iii.) some recession of the eyeball, so that the eye looks smaller than its fellow ; (iv.) slight drooping of the upper lid on that side due to paralysis of its unstriped muscular fibres ; and (v.) absence of sweating on that half of the head and neck, and perhaps the upper extremity of that side, even after the administration of pilocarpin or other sudorific.

Paralysis of the cervical sympathetic may arise from the pressure of an aneurysm of the aorta or other intrathoracic tumour, from exostoses or other tumours in the neck, or from injury or disease of the spinal cord below the medulla, but above the second dorsal segment.

Dilatation of Both Pupils (Mydriasis) may be produced by (i.) a condition of myopia. (ii.) The pupils are larger in childhood. (iii.) Certain drugs, belladonna, duboisin, cocaine. (iv.) Neurasthenia and other exhausted states of the nervous system—*e.g.*, anæmia and typhoid. (v.) During an apoplectic or epileptic attack the pupils are dilated, and in such states it forms a valuable means of diagnosis, for the symptom cannot be feigned, and is present in all the genuine cases of coma, excepting from hæmorrhage into the pons. (vi.) In diphtheritic paralysis, bilateral dilatation of the pupil and loss of power of accommodation (cycloplegia) is an early and constant symptom, and may be accompanied also by complete ophthalmoplegia interna (dilator and sphincter iridis and ciliary muscle). (vii.) Paralysis of the third nerve (destructive lesions of the trunk or its nucleus), a condition which gives rise to four symptoms : Dilatation of the pupil, loss of accommodation, external strabismus (producing diplopia), and ptosis. (viii.) Irritation of the cervical sympathetic as by spinal growths or meningitis. (ix.) Certain psychic emotions such as fear and deep inspiration dilate the pupil. Unilateral recurrent mydriasis often precedes mental derangement.

§ **613. Oculo-motor Defects.**—The external muscles of the eyeball (as distinct from the internal or involuntary muscles of the iris) are six in number, and they are supplied by three cranial nerves: External rectus (VI. nerve); superior oblique (IV. nerve); internal, superior, and inferior recti and inferior oblique are supplied by the III. nerve (which also, it will be remembered, supplies the levator

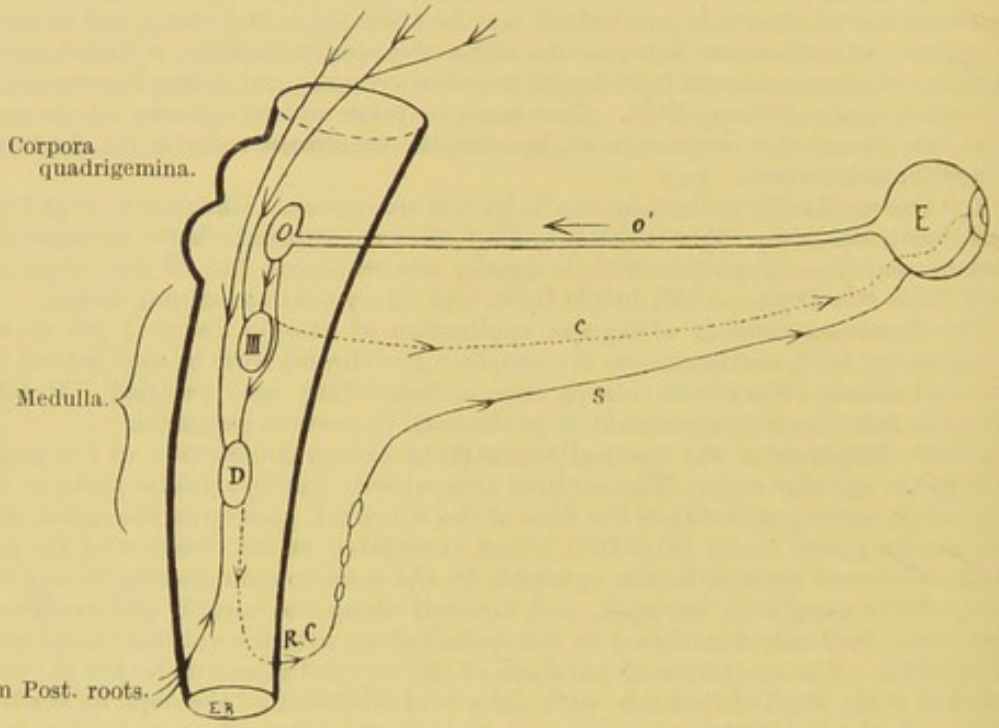

Fig. 165.—Diagram of the REFLEX ARCS concerned in MOVEMENTS OF THE PUPIL (vertical plane).—*O*, nucleus of optic nerve in corpora quadrigemina; *O'*, optic tract (see Fig. 164); *III*, nucleus of third nerve (the upper part only being concerned in the pupillary movements); *C*, constrictor branches accompanying third nerve; *D*, dilator cilio-spinal centre; *S*, dilator fibrils from cervical sympathetic accompanying the ophthalmic division of the fifth; *R.C.*, Rami communicantes of sympathetic through anterior spinal roots of eighth cervical nerve.

palpebræ, the contractor fibres to the iris, and the ciliary muscle of accommodation). It follows therefore that:

Complete paralysis of the third nerve is attended by	Ptosis; external strabismus; pupil dilatation and immobility; loss of accommodation; inability to move eyeball inwards or upwards, and only imperfectly downwards; eyeball slightly protruded.
Paralysis of the sixth nerve is attended by	Internal strabismus; inability to move eye outwards; homonymous diplopia.
Paralysis of the fourth nerve is attended by	Slight deviation of cornea upwards; homonymous diplopia on looking downwards.

Defects in the ocular muscles are revealed (1) by defective movements of the eyeball; (2) by squint; (3) when the defect is only slight, by double vision (diplopia); (4) by ophthalmoplegia externa; or (5) by nystagmus. The *first* question to answer is which of the muscles

is affected ? The *second* question relates to the position and nature of the lesion.

(1) The **movements of the eyeballs** may be tested in simple cases by fixing the patient's chin with one hand and asking him to follow with his eyes your other hand moved slowly inwards, outwards, upwards, and downwards. Notice any deficiency of movements, jerkiness, or nystagmus. One eye may be tested at a time. The power of convergence should also be tested by asking him to look steadily at the tip of your finger while it is moved from a distance of 18 inches up to the tip of his nose.

Deficient movement of the front of the eyeball—	*indicates paralysis of*
outwards	external rectus—sixth nerve.
inwards	internal rectus—third nerve.
upwards	{ *superior* rectus / *inferior* oblique } third nerve.
downwards	{ *inferior* rectus—third nerve. / *superior* oblique—fourth nerve.
downwards and outwards	superior oblique—fourth nerve.

(2) **Squint**, or strabismus, is a want of parallelism between the two visual axes due to defect in one of the muscles. It is called *convergent* when the eyeballs both look inwards, and *divergent* when they look away from each other. *In children* it is mostly caused by the weakness of one muscle or overaction of the other, or both combined, and is due to some error of refraction—hypermetropia, with concomitant internal strabismus (the commonest in children), or myopia, with external strabismus. In adults squint is more often due to definite paralysis of an ocular nerve, and the matter is of much greater significance (see below). In CONCOMITANT (or spasmodic) squint, the affected eye follows the sound eye with equal defect in all directions, that being the reason why it is called " concomitant " squint. Each eye, when the other is covered, moves perfectly in all directions, but when examined together, the squint is present in all positions of the eyeball, especially when the eye is looking straight forward, which is the position of rest for all the healthy muscles. The defect of parallelism remains the same in all positions. PARALYTIC squint disappears when the eye is directed straight forward, which is the position of rest, and only appears when the eye is moved into that position which necessitates the use of the paralysed muscle. Diplopia is present in paralytic, but not in concomitant, squint.

To examine for squint and *to detect whether it is due to paralysis or spasm*, and which is the affected muscle, the patient is told to look at an object straight in front of him, that being the normal position of the eyes at rest, and to fix some object. The eye with which he fixes is the normal eye. The deviation of the affected eye from the middle line is known as the " primary deviation." Now partially cover the sound eye and let him fix with the affected eye. The sound eye will be found to deviate (" secondary deviation "). In concomitant squint the primary and secondary deviation are equal, but in paralytic squint the secondary exceeds the primary. The *position of the head* affords a valuable hint as to which muscle is paralysed. Several rules are given in books, and the student can, if he likes, work out the direction in which a child would naturally look to compensate for the faulty position of the eye. But he will find that the patient unconsciously turns his face, or rather his chin, *towards the side of the weak or paralysed muscle.*

(3) **Diplopia,** or double vision, is the most delicate test for slight weakness or paralysis of one of the ocular muscles, even when the weakness is too slight to produce any discoverable defect in the movement or position of the eyes. To detect which is the affected muscle, hold a pencil vertically in front of the patient, and move it rapidly to the right, to the left, and in various directions, and ask him whether he can see two pencils in any of these directions. *The weakened muscle*

is on the same side as the direction in which the double vision appears. But to detect accurately which eye and which muscle is in fault, further procedures may be necessary (see below). Erroneous projection—*i.e.*, error in judging the distance of objects—and vertigo (due to the same cause) are frequently associated with diplopia. The false image which is seen by the affected eye is generally hazy and less distinct than the true image seen by the sound eye. Diplopia may be homonymous or crossed. In simple or homonymous diplopia the false image lies on the same side as the affected eye ; in crossed diplopia the false image lies on the side opposite to the affected eye. Paralysis of the external rectus causes homonymous diplopia, paralysis of the internal rectus causes crossed diplopia.

Method of Detecting the Affected Eye and Paralysed Muscle.—Place a red glass before the patient's left eye, and place a candle before him in a dark room, on a level with his eyes, and about 3 yards distant. Suppose that it is found that the red image overlaps, or is a little to the left of the white image, and both images are on the same level. To determine which muscle is affected the candle must be moved to the right and to the left, and we must notice *in which direction the distance between the images becomes increased.* Suppose that on moving it to the right the image approaches till only one candle is seen, and on moving to the left, the distance between the true and false images increases. Bearing in mind the rule that the weakened muscle is on the same side as the direction in which diplopia appears,

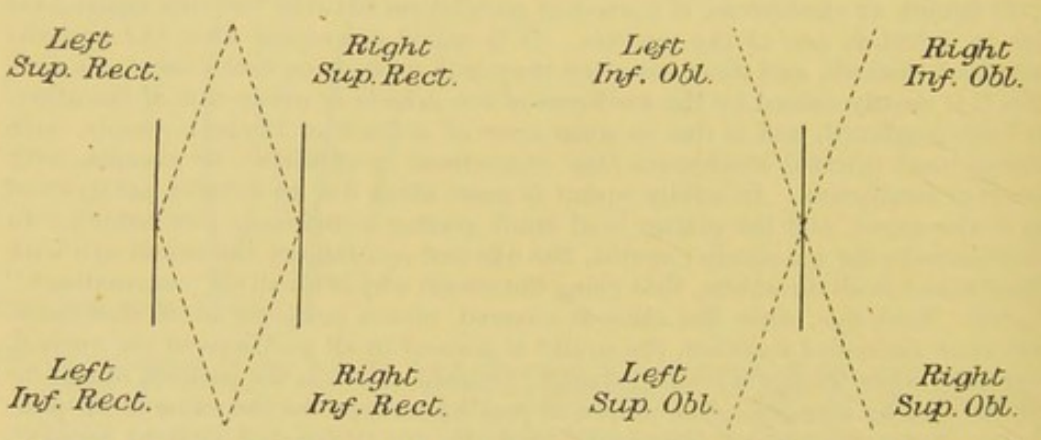

Fig. 166.—WERNER'S DIAGRAMS for detecting which is the affected muscle in cases of diplopia.

it is evident that either the left external rectus or the right internal rectus (which turn the eyes to the left) is affected. Ask then on which side the red image appears. If on the left of the white image, homonymous diplopia is present ; therefore the left *external* rectus is the paralysed muscle. If, however, the red image is to the right of the white image, crossed diplopia is present ; therefore the right *internal* rectus is the paralysed muscle. Paralysis of the superior and inferior recti, and of the superior and inferior oblique, give rise to vertical diplopia, and there is more difficulty in discovering which muscle is affected. Loss of motion upwards is due to paralysis of the third nerve ; loss of motion downwards may be due to paralysis of the inferior rectus (third nerve) or the superior oblique (fourth nerve). Werner's diagrams (Fig. 166) simplify the detection of the affected muscle in vertical diplopia. The black lines in the diagrams represent the true images, the dotted lines the false images. The dotted lines extend above and below the white lines, indicating that the false images are higher and lower than the true images. The names of the muscles in the upper and lower part of the diagrams indicate that the diplopia is caused by upward and downward movements respectively of the eyes when these muscles are affected. Thus, for example, in paralysis of the right inferior rectus an analysis of the diagram shows that (1) the diplopia occurs with downward movements of the eyes ; (2) the diplopia is crossed, the false being to the left of the true image ; (3) the false image has its upper part

inclined towards the true image ; and (4) the false image lies lower than the true one. With the oblique muscles it must be remembered that the superior oblique moves the eye downwards, and therefore the false image due to the paralysis of the superior oblique appears on moving the eyes downwards. By remembering the diagrams it is comparatively simple to diagnose the paralysed muscle causing a diplopia.

Monocular Diplopia is rare. It is recognised by the persistence of the symptoms when one eye is completely closed. The symptom, which should be carefully verified, may arise in some defects of the lens or the retina, and in some central or functional disorders.

The **Position** and **Nature of the Lesion in Ocular Paralysis** is the next question to consider, after having ascertained which muscle is affected in a case of squint or diplopia.

The concomitant squint of *children* in about 90 per cent. of the cases is due to hypermetropia or some other error of refraction, and when this is remedied the squint disappears. It is only rarely due to some congenital weakness of a muscle, or to a nuclear lesion like that of anterior poliomyelitis, and occasionally to intracranial lesions like those met with in adult life.

In *adults*, however, ocular paralysis assumes a much graver significance. Here we have to do with a gross or dynamic lesion involving (*a*) the nerve trunk of the third, fourth, or sixth somewhere in front of the medulla, or (*b*) the nuclei of these nerves in the pons or medulla, or (*c*) the cortex cerebri or motor fibres passing thence to the oculo-motor nuclei.

(*a*) Lesion of a *nerve trunk* is the most frequent cause of squint or diplopia in adult life. In these cases we often find paralysis of a single nerve or a single muscle. There is never any conjugate paralysis (see below) with lesion of a nerve trunk. Of single muscles the external rectus is the one most commonly involved, on account of the long course of the sixth nerve within the cranium. It may, indeed, be affected on one or both sides simply by increased intracranial pressure from a distant tumour or an acute meningitis. Other ocular nerve lesions are due to syphilitic or other neuritis, pressure by syphilitic or other growths in the orbit, pituitary fossa, or base, aneurysm of the carotid or thrombosis within the cavernous sinus (with involvement also of the second and fifth nerves), or extravasation of blood after injury to the base. Acute peripheral neuritis of all the ocular nerves is rare, but sometimes is seen with alcoholic neuritis. It causes ophthalmoplegia, which is only differentiated from ophthalmoplegia of nuclear origin by the absence of other head symptoms and the presence of peripheral neuritis elsewhere.

(*b*) *Nuclear Lesions.*—The third nerve has a long nucleus in the floor of the aqueduct. The upper or anterior end of this nucleus probably controls the pupil and accommodation, then in order the levator palpebræ, internal rectus, superior rectus, inferior oblique, and lowest of all, the inferior rectus. Immediately below this comes the fourth nucleus, and below the latter the sixth nucleus in the floor of the upper part of the fourth ventricle. Sudden and acute nuclear lesions are rare in these situations, but chronic degenerative lesions are met with. The commonest result of nuclear lesions is ophthalmoplegia externa (see below), caused by progressive involvement of all three nuclei, and another instance is met with in the loss of light reflex in tabes dorsalis, when the upper end of the *third nucleus* is affected. The simultaneous onset of paralysis of the third nerve and hemiplegia (including the face) on the opposite side points to a lesion in the crus cerebri. A lesion of the *sixth nucleus* of one side, such as softening, has two peculiarities in which it differs from sixth nerve trunk lesions. (i.) Conjugate ocular paralysis results, and both eyes are deviated *away from the side of the lesion* by the unaffected muscles. This is due to the paralysis of the external rectus on the side of the lesion being associated with paralysis of the internal rectus of the other side. This is probably explained by the existence of commissural fibres connecting the two nuclei which are so frequently associated physiologically. (ii.) In some cases where the lesion is a little more extensive the entire facial nerve of the same side is also paralysed, since the fibres of this nerve twine round the sixth nucleus.

(c) *Cerebral* or *supra-nuclear lesions* never paralyse single muscles as do lesions at the base of the skull involving the oculo-motor nerve trunks. Cerebral lesions always take the form of a **conjugate deviation of the eyes and head,** the neck muscles, which are so closely associated with the eye muscles in turning the head, being also involved, and such lesions are attended by hemiplegia or other evidences of a cerebral lesion. There is one spot in the cerebral cortex just in front of the upper end of the Rolandic fissure, which, when stimulated, causes conjugate deviation of the head and eyes to the opposite side. An irritative lesion in this situation, or anywhere in the course of the fibres between this and the oculo-motor nuclei, will cause deviation, as already pointed out, *away from the source of irritation* (!). But destructive or paralytic lesions (which are more frequent) in this position cause deviation *towards* the side of the lesion, from the unopposed action of the unaffected muscles. For instance, hemiplegia due to cortical hæmorrhage (an irritative lesion) is often attended during the first few days by transient conjugate deviation of the head and eyes to the opposite side. In lesions at a lower level, as in certain rare focal lesions situated in the pons,[1] the above rules as to the direction of the deviation are reversed, and the deviation is apt to be of a more lasting character. Destructive lesions cause a deviation of the eye or eyes and head *away* from the lesion. These are interesting examples of associated paralyses (paralyses of muscles physiologically associated) due to cerebral lesions.

(4) **Ophthalmoplegia externa** means total paralysis of all the external muscles of the eyes, the gaze being fixed and the eyes being motionless ; and since partial ptosis is generally present at the same time, the patient has a sleepy expression, which is very characteristic. It may occur alone, or associated with ophthalmoplegia interna. Acute ophthalmoplegia is due to inflammation or hæmorrhage involving the nuclei, and is met with in diphtheria, diabetes, influenza, and lead poisoning. The chronic form comes on so gradually that the patient may not have noticed it excepting by the continual necessity of moving his head to look round. It indicates a chronic degeneration of the oculo-motor nuclei, generally of syphilitic, diphtheritic, or other toxic origin. External and internal ophthalmoplegia may also occur, either alone or together, in some spinal lesions, such as tabes and bulbar paralysis, and in some cerebral diseases involving the cranial nuclei. Weakness of the orbicularis palpebrarum (supplied by the facial) is often met with in cases of ophthalmoplegia externa, and this is an interesting fact when we remember that the fibres of the facial nerve wind round the nucleus of the sixth.

(5) **Nystagmus** is a rapid involuntary oscillation of the eyeballs, usually from side to side (lateral nystagmus), occasionally in a vertical direction (vertical nystagmus), or in a circular direction (rotatory nystagmus). Both eyes are usually involved, though each eye should be separately examined. The movements may be constantly present, but slighter degrees can only be brought out by causing the patient to follow your finger or a bright object to the extreme left or right. If so, it indicates a weakness of the muscles on the side to which the eye is turned at that time. The symptom is notably present in disseminated sclerosis, cerebellar tumour, and Friedreich's disease, and in tumour involving the corpora quadrigemina, or one side of the pons. It occurs also with meningitis, thrombosis of the sinuses, and cerebral softening in various situations. It may be an indication of corneal, lenticular, retinal, or choroidal defects, especially when these start in infancy. Sometimes it is congenital, especially in albinos. It is also met with in miners, compositors, ironfounders, and those who work at close quarters, or in a strained position with deficient light.[2]

The *Prognosis* and *Treatment* of oculo-motor defects are those of their causal conditions. A large proportion of them are syphilitic in origin, and therefore amenable to treatment. In general, recovery is more probable in nerve trunk lesions than in chronic nuclear conditions. Ophthalmoplegia interna may remain

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[1] See a case recorded by Dr. A. Hughes Bennett and the author in *Brain*, 1889-1890, vol. xii., p. 102.

[2] Snell, *Brit. Med. Journ.*, 1896, vol. i., p. 1503.

stationary for many years. For chronic cases counter-irritation and galvanism (applied through the nurse's fingers over the eyelids) may be tried.

§ 614. **Ophthalmoscopic Defects** in the fundi oculorum.—The **ophthalmoscope** is an instrument which is indispensable for the discovery of the affections of the retina, choroid, and optic nerve. It is also of great use in general medicine. It is used for three procedures: Indirect ophthalmoscopy, direct ophthalmoscopy, and (as mentioned in § 611) retinoscopy.

The ophthalmoscope has two concave mirrors, the larger one for indirect ophthalmoscopy and retinoscopy, and a smaller one, which is tilted, and capable of being turned on its own axis, for direct ophthalmoscopy. Behind each of these is a hole, through which the observer looks, and across which a series of lenses can be made to turn, of different refractile power. The room should be darkened, and a bright, steady light placed just beside and behind the patient's head, on a level with his ear, on the side of the eye to be examined. An Argand burner is the best, either gas or oil. An electric lamp is not so good, as the edge of the light is not straight. Take the ophthalmoscope in your right hand and correct your own error of refraction (if any) by rotating the appropriate lens into the hole behind the mirror through which you will look. It makes it easier for a beginner even with normal sight to use a + 1 lens. If the patient is sitting in a chair, make him sit up (not backwards), with his head slightly forwards, about 18 inches or 2 feet away from your own. First learn to reflect the light steadily into his pupil, previously dilated with a 2 per cent. solution of atropin or homatropin. Tell the patient to look away into the distance over your shoulder.

The next step for *indirect ophthalmoscopy* is to interpose between your eye and that of the patient the convex lens. This lens should be of 2½ inches focal length, and should measure at least 2½ inches across. This lens should be held about 2 inches from the patient's cornea, between the thumb and index of your left hand, while the little finger rests upon the patient's forehead. You can only find the correct position of the lens by moving it backwards and forwards until a clear image of some retinal vessel is obtained. *The image of the retina seen is an inverted one.*

There are three parts to be examined: (i.) The optic disc, which is seen by telling the patient to look in the direction of the tip of your left ear for his left eye, and *vice versa*. (ii.) The peripheral parts of the retina may be examined by the patient turning the eye as far as possible in various directions. (iii.) The macular region is situated two disc breadths to the outer side of the disc, and is brought into view when the patient looks at the hole in the ophthalmoscopic mirror, or slightly to the side of it when the light is too strong for him to bear.

Direct Ophthalmoscopy is conducted with the patient placed as before, but your head is moved up *quite close* to the patient's eye, so as to look straight into it, as in Retinoscopy (§ 611). Change the mirror on the ophthalmoscope for the smaller tilted one spoken of above, and rotate it on its own axis, so that its prominent *forward edge will be next to the bridge of the patient's nose*, the lamp being on the patient's right hand for his right eye, on his left hand for his left eye, and always at the level of his ear. First adjust the ophthalmoscope accurately to your own eye (right for examining the patient's right eye, and left for his left), and get the handle flat against your cheek. Then tilt your head a trifle sideways close to that of the patient, and tilt his head slightly the other way to avoid his breath. If the fundus is not illuminated, shift the lamp or the angle of the mirror, or your head, until the red glow from the retina is seen through the little hole in the mirror, Now, to get a good image, *you must completely relax your own accommodation*. Herein lies the difficulty, but if you imagine that you are looking away at some distant object, you will by degrees acquire this necessary condition. *The image, in direct ophthalmoscopy, is always an erect one and magnified*, and therefore the different parts of the fundus can be more carefully investigated in detail than by the indirect method. To see the disc you should look obliquely inwards when the patient is looking straight in front of him, and the other parts of the retina may readily be examined by the patient moving the eyes slightly in different directions.

The *optic disc* should be examined as to its shape, its borders, its colour, its vessels, and its level. Normally the disc is slightly oval or circular, with a clearly defined border, especially at the outer edge. It appears oval in astigmatic eyes. The *colour* of the disc is a rosy vermilion, but paler than the rest of the fundus. The vessels curve from the centre, and then lie flat. Arteries and veins go together, but the arteries are narrower (two-thirds) than the veins, a trifle paler, and have a broader, more continuous light stripe running along the centre. Normally the arteries do not pulsate, but the veins, curiously enough, may do so. Pulsation in the arteries may indicate (i.) increased intraocular tension or (ii.) aortic regurgitation. The *level* is important, but a little difficult to gauge. Cupping of the disc is usual in high myopia, and if the cup be deep, the disc is always pale, a condition which should not be confused with optic atrophy. The level of the disc is best detected by the direct method of ophthalmoscopic examination. If when thus using the ophthalmoscope the *retina* can be seen clearly without the aid of any lens placed in the mirror hole, but the *disc* cannot be seen clearly without the aid of the lens, it must be at a different level. If a little + glass is necessary to see the disc under these circumstances, then the disc must be clearly behind the retinal level (cupping). If, on the other hand, a little — lens is necessary, then the disc is on a level anterior to the retina (swelling). One can even gauge the amount of swelling or cupping in this way, for roughly each 3 D. = 1 mm. of swelling or cupping. Thus, supposing it is necessary to use 1·5 D. to focus the retinal vessels precisely, and 4 D. to focus the disc, then there must be 2·5 D. swelling or cupping. This is an accurate method of measuring, provided the observer is able to *thoroughly relax his own accommodation*.

Two important **Morbid Changes in the Optic Disc** are met with (in addition to hyperæmia and anæmia)—optic neuritis and optic atrophy. In *hyperæmia*, the colour of the disc resembles the rest of the fundus. Active hyperæmia may be present in hypermetropia. Passive hyperæmia is present in optic neuritis and in the venous congestion secondary to cardiac or pulmonary disease. *Anæmia* of the optic disc is indistinguishable from an early stage of atrophy. It requires some experience to detect the difference in these delicate shades.

Optic Neuritis, or Papillitis, is inflammation of the optic nerve at its entrance into the globe, and is evidenced, in its typical marked form, by swelling, increased redness and vascularity of the disc, and blurring of the edges. The arteries become narrower, and the veins are enlarged and tortuous, the vessels appearing to curve over the œdematous edge. The arteries, moreover, may appear broken here and there, as they are hidden by the œdema. In the early stage the disc has simply a fluffy look, and then the upper and lower edges only are blurred, a condition which is only apparent when the direct method is employed. These inflammatory changes may gradually subside, but more commonly they go on to " consecutive " atrophy. It should be remembered that the *power of vision may be undisturbed*, even when there is considerable papillitis, though the visual field is usually diminished in some degree. Disturbance of vision is generally more marked as the acute stage subsides. Early and progressive loss of vision points to damage of, or pressure on, the chiasma, and in such a case the pupil is dilated and immobile to light. In optic neuritis with preservation of vision the pupils react to light. *Double optic neuritis* is very strongly suggestive of intracranial disease, and especially (i.) cerebral tumour, in which it is present at some time in about 80 per cent. of the cases. It is less common in cerebral abscess and in cerebellar and other diseases in the posterior fossa, and is rare in cerebral hæmorrhage and embolism. (ii.) Increase of pressure from any other cause—*e.g.*, tuberculous meningitis—though it may be at a late stage. It is not common with simple acute meningitis. It is occasionally present in the cerebral form of disseminated sclerosis. Syphilis may produce papillitis in several ways. Various *toxic conditions* of the blood may produce optic neuritis, chief among which is renal disease giving rise to a special form (see below). Plumbism and anæmia are occasional causes, also influenza, rheumatism, typhoid, and scarlet fevers. Sudden suppression of the menses has been known to cause optic neuritis.

Unilateral Papillitis indicates disease at the back of the orbit—*e.g.*, tumour or gummatous infiltration, cellulitis, or periostitis.

Optic Atrophy—that is, atrophy of the optic disc—is characterised by excessive pallor of the disc, a very sharply defined outline, and smallness of the vessels. In marked cases sight is completely lost, excepting, perhaps, for light. Optic atrophy may be "*consecutive*" to a severe papillitis. It may also succeed embolism of the central artery, retinitis pigmentosa, syphilitic choroido-retinitis, or pressure or injury of some part of the optic nerve or chiasma (as when blindness of one eye follows a blow on the head). *Primary* atrophy, part of a sclerosis of the optic nerve, usually arises in connection with some spinal lesion, such as tabes or disseminated sclerosis. Primary optic atrophy without other cranial symptoms should always make us suspect tabes. It is seldom possible to differentiate primary or consecutive atrophy without a knowledge of the clinical history. Optic atrophy may supervene on toxic amblyopia.

Retinitis, inflammation of the retina, is evidenced by a smokiness or haziness and loss of transparency. It may be either diffuse or patchy. Pigmentary deposits in the retina are superficial to the vessels, and appear as a network. In the choroid, pigmentary deposits are beneath the vessels, and occur for the most part in rings or blotches. Choroidal atrophy appears as pale or white patches, with rings or spots of pigment in or near them. Only a few of the chief forms of retinal and choroidal disease can be mentioned here.

Albuminuric Retinitis is really a neuro-retinitis, consisting of three elements: (i.) Papillitis (see above); (ii.) hæmorrhages into the retina, usually most plentiful towards the disc; and (iii.) fine white shining spots near the macula, and larger ones on the retina near the disc, with clearly defined margins, supposed to be due to a kind of fatty degeneration of the retinal structures. One or other of these is sometimes wanting, but in its typical form this kind of retinitis is sufficiently distinctive to diagnose renal disease without examining the urine. It may occur in any form of renal affection, but is frequently associated with granular kidney. Hæmorrhages into the retina and choroid are met with as dark red patches, with flame-like margins. They accompany any severe retinitis or papillitis, are met with in albuminuric retinitis, also in pernicious anæmia, leucocythæmia, pyæmia, ague, scurvy, and other purpuric conditions. Retinal hæmorrhages are sometimes met with in elderly gouty persons with degenerated arteries. Embolism of the Central Artery of the Retina occurs most frequently in the course of cardiac disease, especially in disease of the aortic valves, and in acute and ulcerative endocarditis. It is known by the sudden occurrence of blindness in one eye in a case of cardiac disease. On examination, the retinal vessels are found empty, and a peculiar round, cherry-red spot is seen in the macular region. The disc is pale. Retinitis Pigmentosa is characterised by pigmentation in stellate patches near the periphery at first, and over the vessels. The disc is greyish yellow. Night blindness is the chief symptom, and the disease starts in childhood.

Choroiditis Disseminata (usually bilateral, though sometimes limited to one eye) is an evidence of syphilis (hereditary or acquired), in which disease it may arise from three months to three years after the commencement. On examination, discrete, white, atrophic patches, with irregular black edges, are found scattered over the fundus, *most marked at the periphery*. Atrophic Patches in the Choroid may be found in the central region in myopia or as the result of previous choroidal hæmorrhages, or after the absorption of tubercle. Tubercle of the Choroid appears as yellow, roundish, ill-defined spots, one-third to half the size of the disc, situated near the disc, and unaccompanied by other choroidal changes. They may thus be distinguished from choroidal atrophy, where the spots are white, irregular, and pigmented. They appear in general tuberculosis, and in tuberculous meningitis, though usually late in the history of the case, and are therefore not of great diagnostic value.

§ **615.** The **Fifth Nerve** supplies sensation to the face, the sense of taste to the tongue, and motion to the muscles of mastication.

The fifth supplies the face and forehead, the oral, nasal, and buccal cavities, the conjunctiva and sclerotic with *sensation* (see Fig. 167), the whole of the tongue with the sense of *taste;* and it carries—as we know by the effects of lesions of its nuclei or trunk—trophic, vaso-motor, and secretory (to the salivary and lachrymal glands) fibres. A *motor* root arises close beside it, and accompanies its lowest or third division to supply several muscles (the temporal, masseter, two pterygoid, buccinator, mylo-hyoid, anterior belly of digastric, and tensor tympani). Looking upon the nuclei of the cranial nerves as continuations upwards of the anterior (motor) and posterior (sensory) grey horns of the spinal cord, the sensory portion of the fifth nerve constitutes the sensory nerve which corresponds to the facial nerve which is entirely motor.

Clinical Investigation.—The *sensation* of the areas above mentioned can be tested in the usual way. When it is lost, the patient in drinking feels only one-half of the tumbler, and may imagine it is broken. The *motor* functions of the fifth are tested by asking the patient to clench his teeth while you place the fingers on the temporal muscles of both sides and then on the masseters. It may be inferred that the pterygoids of one side are paralysed if on opening the mouth or projecting the chin forward the jaw deviates towards one side—*i.e.*, towards the paralysed side (by the action of the unaffected pterygoids of the opposite side). Paralysis

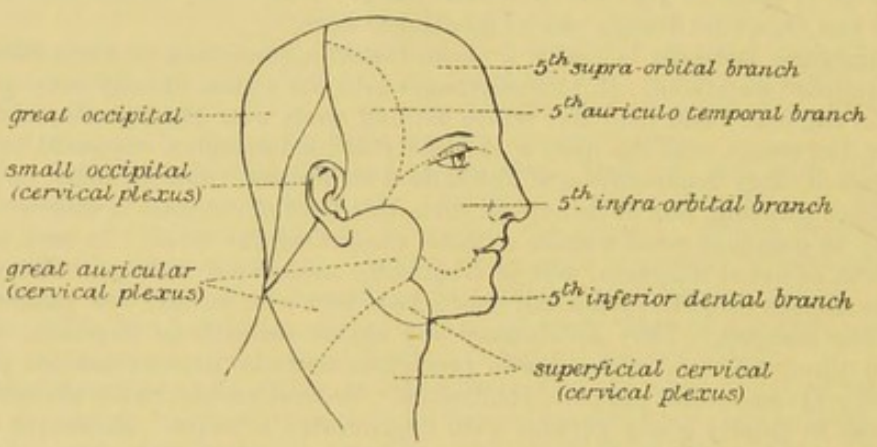

Fig. 167.—Nerves supplying Cutaneous Sensation to the Head.

of the tensor tympani produces a diminution of hearing for high notes. The function of *taste* will be considered below. This should always be tested in suspected lesions of the trunk or ganglion of the fifth.

The *Symptoms* of *destructive* or paralytic lesions of the fifth nerve or nucleus consist of the following alterations, according to the part involved: Anæsthesia (preceded, perhaps, by severe burning pain) of the area supplied by the fifth; loss of conjunctival, palatal, and nasal reflexes on that side; loss of secretion of lachrymal and salivary glands; loss of smell, owing to the dryness of mucous membrane; loss of taste; diminished masticatory power, and collection of food between cheek and jaw; hemiatrophy facialis (§ 617) occasionally.

Irritative lesions of the fifth nerve give rise to facial neuralgia and hyperæsthesia or tender points. Irritative lesions of the Gasserian ganglion are attended also by very serious trophic changes—cloudiness of the cornea, going on to perforation, and destructive panophthalmitis, herpes frontalis, or vesicles in other positions, and in rare cases sloughing of the mucous membrane of the cheeks. *Reflex* irritation of the fifth nerve may give rise through the facial nerve to twitchings of the face, or through the optic nerve to flashes of light.

Lesions of the Fifth.—(i.) Lesions of the *cerebrum*, involving the sensory crossway of Charcot, imply facial as well as corporeal hemianæsthesia, both of which are on the side opposite to the lesion. (ii.) Lesions *within the pons*, attended with corporeal hemianæsthesia, produce a "crossed" anæsthesia, face on same side,

body on the other. Lesions, however, such as hæmorrhage, softening, or tumour in this situation more frequently involve part of the widespread fifth nucleus than the whole. (iii.) *Within the cranial cavity* syphilitic meningeal conditions, caries of the petrous bone on the tip of which the Gasserian ganglion rests, tumours, aneurysms, and very rarely fractures of the base, produce total or partial paralysis of the fifth. Disease in the pituitary region, cavernous sinus, or orbit—*e.g.*, aneurysm of the internal carotid, or orbital cellulitis—damage the first division of the nerve. (iv.) *Outside the cranial cavity* one or other division may be paralysed by *tumours*—tumours of the parotid, for instance, extending into the sphenomaxillary fossa, and damaging the second and third divisions of the nerve. The deep position of the fifth usually protects it from *injury*, and *neuritis* is said to be rare. It is, however, to my mind very difficult to account for the fact that the branches of this nerve form the favourite seat for neuralgia, and that herpes so constantly occurs, excepting on the supposition that those branches, or the nerve or its ganglion, are the seat of neuritis. The fifth nerve is a frequent seat of *reflex irritation* from distant parts (Neuralgia, § 604). It is also the source of reflex irritation in cases of facial twitchings, cough, and other motor spasms.

The *Prognosis* and *Treatment* of neuralgia of the fifth has already been dealt with in § 604. The prognosis and treatment of the other lesions depend upon their nature.

§ **616.** The sense of **Taste** should always be tested in suspected lesions of the fifth, which is the nerve of taste. Powdered salt, sugar, and quinine are necessary. The patient should put out his tongue and keep it out till the end of the test. Having wiped the tongue, place on it a minute portion say of powdered sugar, gently rub in, and ask ask "Is that salt ?" "Is it bitter ?" "Is it sweet ?" The patient can reply by shaking or nodding his head. Citric acid may also be employed. A weak galvanic current causes a metallic taste. Sweets and acids are tasted best by the anterior part of the tongue, bitters by the posterior part.

Tract of Taste (Fig. 168).—Between the tongue and the main trunk of the fifth nerve, taste is conveyed by two separate and circuitous routes. (*a*) The chorda tympani running in the tongue with the lingual branch of the third division of the fifth collects taste fibres from the anterior two-thirds of the tongue (some say the tip and anterior two-thirds of the edge). The chorda then joins the facial nerve just above its exit from the stylo-mastoid foramen, and after running its well-known course through the tympanum doubles back as the Vidian nerve from the facial to Meckel's ganglion, and thence to the second division of the fifth. (*b*) From the middle and posterior part of the tongue taste fibres pass first along the glosso-pharyngeal nerve, which they leave by Jacobson's nerve to join the tympanic plexus, thence by the small superficial petrosal and the otic ganglion they pass to join the second division of the fifth. After reaching the pons, the path of taste decussates, and reaches the posterior part of the internal capsule of the opposite side, and is believed to terminate in the cortex at the tip of the temporo-sphenoidal lobe.

The commonest *Cause* of loss of taste in the tip and edges is (i.) a lesion of the facial in the Fallopian aqueduct, as in middle ear disease. (2) Loss of taste in the posterior part of the tongue is met with in lesions of the third division of the fifth ; of the anterior part in lesions of the second division, and of the whole of one side of the tongue in lesions of the trunk of the fifth just outside the pons. (3) Loss of taste of one-half of the tongue occurs in many cases of hemianæsthesia, whether of functional or organic origin. (4) Disturbances of taste are frequent in influenza, diphtheria, and some other acute specific disorders. (5) Taste is greatly aided by the sense of smell. Flavours, such as those of cheese, wine, or roast

beef, really consist of compound perceptions of smell and taste. Consequently catarrh and many nasal diseases are attended with impairment of taste.

Paragusis (perverted taste) and hyperagusis (increased sensibility to taste) are met with as auræ to epileptic fits, in conjunction with insanity, with hysteria, and sometimes with middle ear disease.

§ **617.** The **Facial Nerve** (the seventh) is a purely motor nerve supplying all the muscles of the scalp and face (except the levator palpebræ superioris), the platysma, two small muscles of the hyoid bone and the stapedius. The facial and the sixth are more frequently

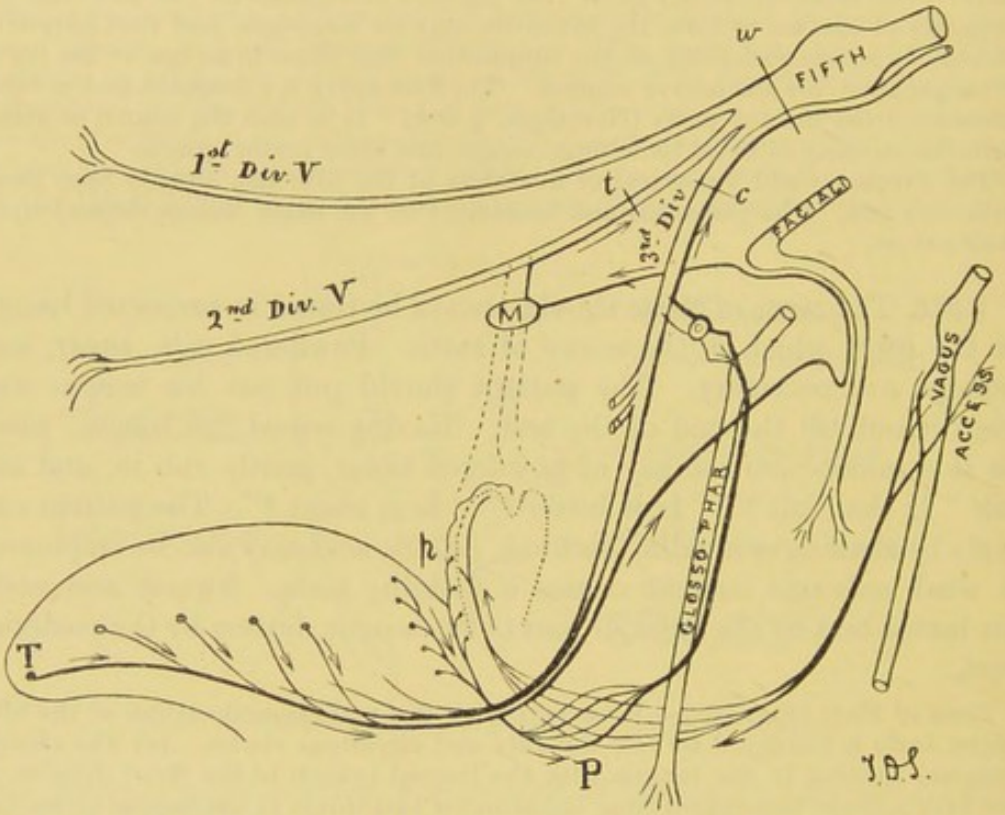

Fig. 168.—Sense of Taste and Nerve-Supply of Palate.—Diagram to show how taste impressions reach the fifth nerve ⟹, and the motor-supply of the palate ⟸. T, branches of the chorda tympani conveying taste from the tip and sides of the tongue, and running with the lingual branch of the fifth, then through the facial to Meckel's ganglion (M), and thence to the second division of the fifth. *p*, soft palate from which, and from the dorsum of the tongue, taste fibres pass through the pharyngeal plexus (P) to join the glosso-pharyngeal, and thence through the otic ganglion (*o*) to the third division of the fifth. The palate, it will be seen, is supplied by the accessory portion of the spinal accessory through the vagus.

involved singly than is any other cranial nerve. The facial is also very frequently partially involved in cases of hemiplegia.

Anatomy of the Facial Nerve (Fig. 169).—The *nucleus* of the facial nerve is situated in the floor of the fourth ventricle, just beneath the most prominent part of the eminentia teres. Its lower end is in close proximity to the nucleus of the twelfth (hypoglossal), a fact which corresponds to the close association between the muscles of the lips and those of the tongue. There is good reason to believe that the orbicularis oris is innervated from the *nucleus* of the hypoglossal (see Bulbar Paralysis). Its upper end is close to the sixth (abducens) nucleus, around which its fibres curve as they pass to the superficial origin. The *cortical centre* for the face is situated at the lower end of the ascending frontal convolution, and this centre is connected with the facial nucleus *through the internal capsule*. Hence

the frequency with which the lower half of the face is affected in cases of hemiplegia. The facial fibres cross to the opposite side in the upper part of the pons, and appear on the surface at the lower edge of the pons. The nerve then accompanies the eighth (auditory) nerve to the internal auditory meatus. At the lower end of the internal auditory meatus the facial nerve presents a swelling (the geniculate ganglion), which gives off three important branches. It then enters the aqueductus Fallopii, which curves forward over the foramen ovale on the inner wall of the tympanum, then passes downwards and out through the styloid foramen, and while traversing the parotid gland breaks into its two main terminal divisions, the temporo-facial to the muscles of the upper half of the face, the cervico-facial to those of the lower half, the platysma, the mylo-hyoid, and the posterior belly of the digastric.

The first and clinically most important branch of the facial is the great petrosal (or Vidian) nerve, which joins the geniculate ganglion to Meckel's ganglion. Meckel's or the spheno-palatine ganglion is connected above with the second division of the fifth, and the lower branches coming off from it supply the palate (Fig. 168). It was formerly thought that these were motor branches originating from the facial, but clinical research shows that the great petrosal nerve is really sensory, and contains taste fibres derived from the second division of the fifth, which join the geniculate ganglion, pass along the facial trunk, and leave the facial trunk as the chorda tympani nerve. It was Hughlings Jackson who first declared he had never seen undoubted paralysis of the palate in association with lesions limited to the facial nerve, that the palate is to a certain extent asymmetrical in most people, and that this fact had not hitherto been sufficiently allowed for. It is, moreover, a clinical fact which Gowers has insisted upon, that loss of taste is only associated with lesions of the facial nerve situated at some point between the geniculate ganglion and the styloid foramen. Lesions of the facial nerve behind the latter point do not give rise to loss of taste. Clinically, it is of great importance to remember that the Fallopian aqueduct is pierced by three structures, and through these holes inflammation may spread to the facial nerve. The first contains a small nerve twig from the facial nerve to the stapedius muscle. The second contains an arterial twig from the tympanum for the nutriment of the structures in the aqueduct. The third contains the chorda tympani nerve which comes off from the facial $\frac{1}{4}$ inch above the stylo-mastoid foramen, and passes forwards between the handle of the malleus and the stapes, turns downwards, and after giving a vaso-constrictor branch to the submaxillary ganglion, terminates in the tongue, which it supplies with taste fibres at the tip and along the anterior two-thirds of the margin.

In the Clinical Investigation of cases of facial paralysis, as in other disorders of the nervous system, the first step is to discover the position of the lesion and then its nature. This is done by investigating first, the muscles that are affected; secondly, the condition of the hearing; thirdly, the condition of the taste; and fourthly, the electrical reactions.

The *Symptoms* of complete paralysis of the facial nerve (Bell's paralysis) are (i.) an obvious one-sided alteration of the face which is pulled up away from the paralysed side by the unaffected muscles, the forehead and cheek of the paralysed side being smooth and expressionless. (ii.) The patient cannot shut his eyes, or in slight cases he cannot keep them shut when we try to forcibly open them. (iii.) The corner of the mouth is drawn up when he attempts to screw up the eyes, he cannot smile or show his teeth. He cannot whistle, and the food collects between the cheek and teeth. (iv). The taste and (v.) the hearing may be affected in certain cases (see below). Paresis of the palate

used to be mentioned, but the palate is now known to be supplied by the spinal accessory. In the course of many months the *affected side*

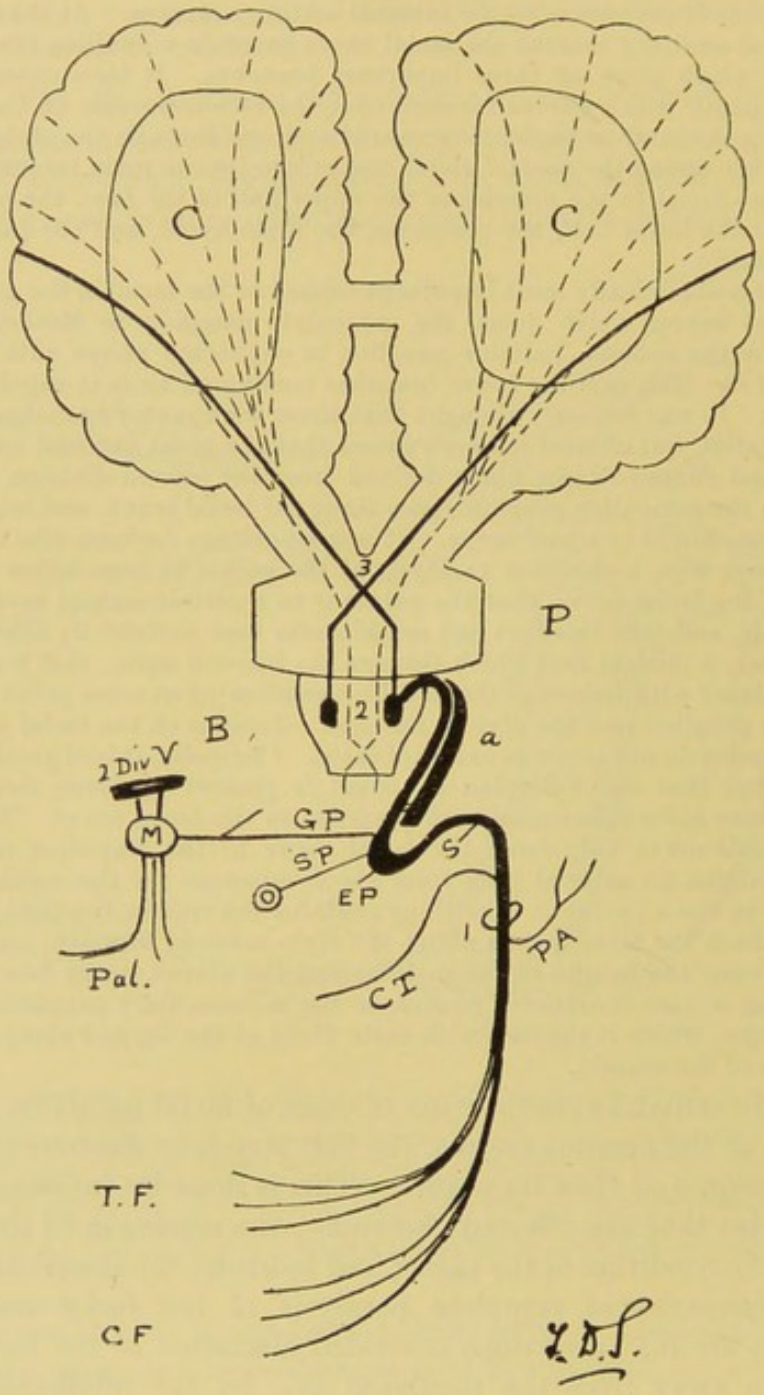

Fig. 169.—The FACIAL NERVE, its course, and connections.—1, stylo-mastoid foramen; 2, above pyramidal decussation; 3, decussation of facial fibres in the pons. C.T., chorda tympani; S., branch to the stapedius; E.P., S.P., and G.P., external, superficial, and great petrosal nerves; O., the otic, and M., Meckel's ganglia; P.A., posterior auricular branch.; *a*, auditory nerve; Pal., sensory branches from the palate to the second division of the fifth (compare Fig. 171).

becomes drawn up by contraction of the paralysed muscles (Fig. 170). Facial paralysis must not be confused with hemiatrophy facialis (see below).

The **Varieties of Facial Paralysis** differ according to whether the lesion is (*a*) in the brain, (*b*) in the facial nucleus, or (*c*) the facial nerve. (*a*) is incomplete, but (*b*) and (*c*) are complete (or Bell's) paralysis. What follows should be compared with Fig. 169.

(*a*) With Cerebral (Supra-nuclear) Lesions facial paralysis is extremely common, and it differs considerably from that of lesions situated at or below the facial nucleus. (i.) It is usually associated with hemiplegia on the same, or occasionally on the opposite, side. If (as is usual) the hemiplegia is on the same side as the facial palsy, the lesion is situated in the internal capsule or above the pons, but in crossed hemiplegia it is situated *in* the pons. (ii.) The upper half of the face is exempt from the paralysis (the patient can close his eyes), because, it is

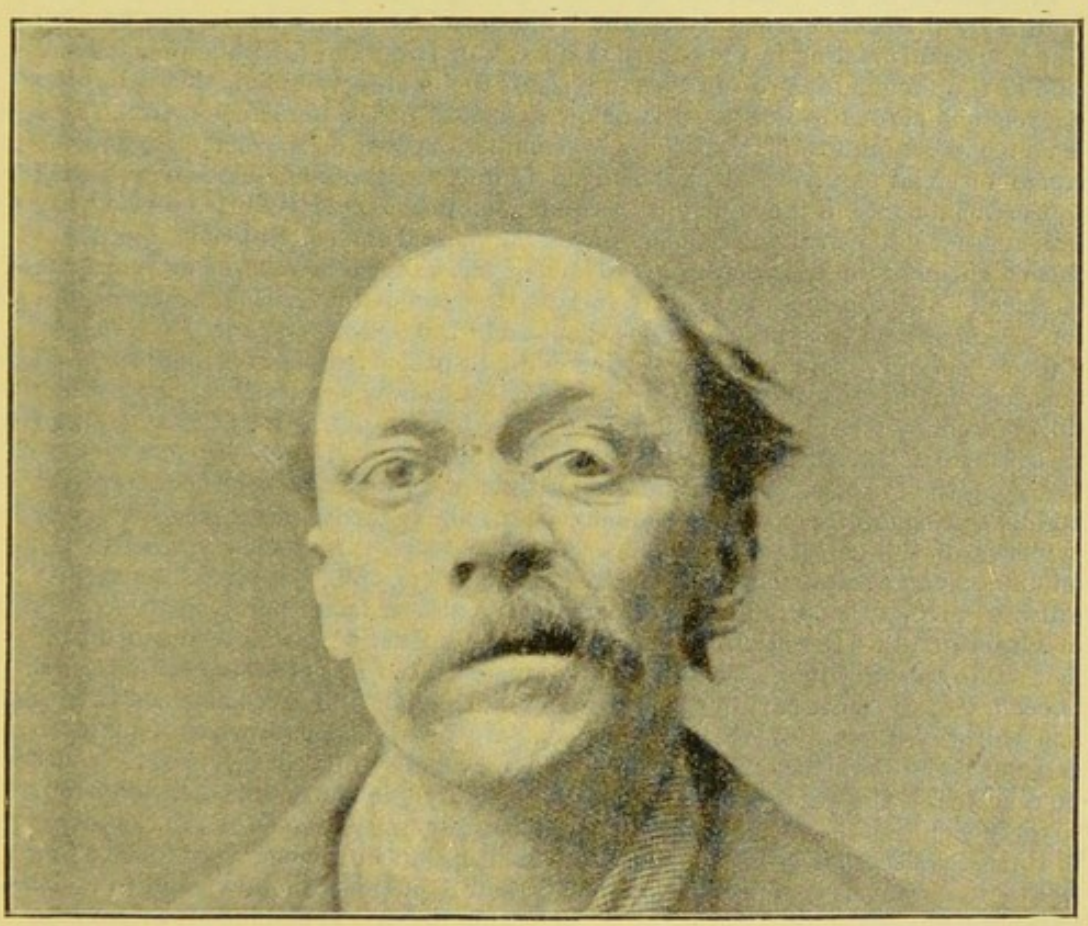

Fig. 170.—Facial Paralysis.—This picture appears to represent *right* facial paralysis, but in reality it represents the face of a man aged forty-two with paralysis of the *left* side, which had occurred two years previously consequent on middle-ear disease. It exhibits, therefore, late contracture of the paralysed muscles on the left side.

suggested, a certain number of the fibres of the facial nerve escape destruction. Infra-nuclear lesions, on the other hand, give rise to paralysis of all the facial muscles—Bell's paralysis, as it is called. (iii.) The muscular power is speedily restored ; and (iv.) there are no electrical changes. The anatomical lesions are those of hemiplegia (*q.v.*). Hysterical facial palsy is extremely rare. It is slight, and the platysma is said to be exempt.

(*b*) Nuclear Lesions are rare. (i.) Atrophy and marked R. D. supervene early. (ii.) Diplopia may be present owing to the proximity and involvement of the sixth nucleus. (iii.) All the facial muscles are affected, but some more than others. If the lips are mainly affected, we must suspect glosso-labio-laryngeal paralysis, and should investigate movements of the tongue and larynx. If the lips

are exempt, and the upper part of the face alone is paralysed, we have probably to do with a nuclear lesion affecting chiefly the upper part of the nucleus, such as arises in rare cases of diphtheria, spinal affections, such as tabes and disseminated sclerosis, rare cases of focal lesion, and anterior poliomyelitis spreading upwards.

(*c*) **Bell's Paralysis.**—INFRA-NUCLEAR LESIONS give rise to paralysis of *all the facial muscles* on one side (Bell's paralysis), but the symptoms will vary somewhat with the precise locality of the lesion.

(1) Lesions of the *facial trunk* just in front of the pons are known by (i.) complete paralysis of all muscles ; (ii.) R. D. and wasting ; (iii.) possibly some nerve deafness by involvement of the adjacent auditory nerve ; but (iv.) taste is always normal. Gummatous and other tumours may affect the nerve here.

(2) Lesions in the *Fallopian aqueduct* (perhaps the commonest position) are known by (i.) complete paralysis of all the muscles, coming on very suddenly ; (ii.) R. D. and wasting in the course of a week or two ; and (iii.) taste of tip and side of the tongue lost, owing to paralysis of the chorda tympani, this being the pathognomonic feature of neuritis or other lesions in the aqueduct. (iv.) There is no nerve deafness, but the hearing is generally affected. One of the commonest causes of facial paralysis is disease of the middle ear, the inflammation spreading to the facial nerve in the aqueduct, a condition of some gravity, because the bony walls do not allow of the expansion of the nerve, and the compression aggravates the condition. In some cases paralysis of the stapedius muscle may, in the absence of otitis media, give rise to increased acuteness of hearing, especially for low tones, on account of the unopposed action of the tensor tympani.

(3) *Neuritis External to the Skull* (rheumatic or other) produces (i.) paresis of all muscles, but usually incomplete ; (ii.) recovery usually ensues in a short time ; (iii.) there may be some diminution of faradic reaction, but no R. D. ; and (iv.) no loss of taste unless the inflammation spreads into the aqueduct.

Among the *Causes of Bell's Paralysis* otitis media or some other affection of the middle ear is perhaps one of the commonest, as above mentioned, and a history or evidence of this is often obtainable. A simple sore throat may spread to the middle ear and cause facial paralysis. Exposure to chill, as, for instance, in a railway carriage, is certainly a very frequent cause of facial paralysis, especially of Variety *c* (3). It sets up a neuritis or peri-neuritis, which may spread up the nerve into the aqueduct, as evidenced by an associated loss of taste in some cases. It occurs for the most part in gouty or rheumatic people and those between twenty and thirty-five years of age of either sex. In nearly three-fourths of nineteen cases of facial paralysis lately under my care a history of chill or exposure could be revealed. The paralysis comes on quite suddenly in the course of twenty-four to forty-eight hours after the exposure, and is rarely quite complete. In the course of three days there may be some loss to faradism, but in the course of four or five weeks the paralysis generally clears up. Fracture of the base may result in facial paralysis, forceps applied during birth, or boxing the ears in childhood, as in a case mentioned by Gowers. Basal meningitis, especially of syphilitic origin, may involve the facial nerve, but usually some other cranial nerve as well. Rare cases of hæmorrhage into the aqueduct have been confirmed by post-mortem. Inflammation and new growths of the parotid are generally sufficiently obvious as causes of the condition. Diphtheria and alcohol are occasional causes.

The *Prognosis* of facial paralysis as to recovery depends partly upon the position and partly upon the cause of the lesion. The prognosis of supra-nuclear conditions depends upon the concomitant symptoms. The prognosis is much more serious in all lesions within the skull than those external, and especially when the lesion is within the aqueduct, which invariaby results in permanent degeneration of the nerve. In cases of external neuritis due to chill recovery is the rule. The electrical changes give us valuable information as to prognosis. My general rule is as follows : If there are no, or very slight, electrical changes in the second or third week, recovery is the rule, but if the reaction to faradism remains markedly lowered, and especially if it is quite lost for three months, the chance of recovery is small, if for six months, *nil*.

The *Treatment* in most cases is hopeful. It must be directed to the cause, and the assistance of an aural surgeon is generally required. Even apart from syphilis, iodide of potassium in small doses is very useful in rheumatic cases, especially if combined with quinine. As a remedial measure galvanism is of great use to restore the nutrition of the muscles and the function of the nerve, if applied regularly.

Diplegia Facialis, or double facial paralysis, is somewhat difficult of detection, but it is characterised by a total absence of expression, an inability to close both the eyes, and an absence of adequate movement around the mouth in talking. It is a much more serious condition than one-sided facial paralysis because of the lesions which it accompanies.

Double facial paralysis may be *caused* by some of the lesions previously mentioned occurring on both sides, such as (i.) basal lesions (*e.g.*, syphilitic meningitis or tumours), or (ii.) double otitis media. In both of these it generally happens that first one facial nerve is affected, and then some weeks or months later the nerve of the other side. (iii.) Diphtheria, and (iv.) focal lesions in the upper part of the pons may affect both sides at once. Diphtheria is, I believe, a more frequent cause of slight double facial paralysis than is generally recognised. It is evidenced by the expressionless aspect of children suffering from diphtheritic paralysis. (v.) Focal lesions in the upper part of the pons in the position of the decussation of the facial fibres are extremely rare.

Hemiatrophy Facialis is a rare condition, consisting of atrophy of the skin and its appendages, the subcutaneous tissue, and sometimes of the bones on one side of the face, unattended by alterations in the sensation or muscles of the face. It must not be confused with facial paralysis. It is believed to be due to a degenerative lesion (possibly of the nature of poliomyelitis) of the upper part of the nucleus of the *fifth nerve*, with atrophy of the upper part of the nerve trunk within the pons. One case observed by the author [1] followed a severe neuralgia which dated from an injury to the vertex.

§ 618. The **Auditory Nerve** (the eighth) is the nerve of hearing and orientation.

It arises by two roots. The dorsal or auditory root arises from the internal auditory nucleus, and passes out behind the restiform body. The ventral root (orientation) arises from the external auditory nucleus and passes out in front of the restiform body. The two roots join and enter the internal auditory meatus to be distributed, the former to the cochlea, the latter to the semicircular canals. The cortical centre for hearing is in the first and second temporo-sphenoidal convolutions of the opposite side, that for orientation is unknown. The auditory apparatus is shown in Fig. 171.

Clinical Investigation.—*Hearing* should be tested by the voice or a watch and a tuning-fork. The presence of wax in the external meatus must be first excluded, and if necessary the ear should be syringed with warm water after the wax has been softened by warm oil or bicarbonate of soda solution (2 teaspoonfuls to the pint). To test the *acuteness* of *hearing*, stand behind your patient, close one of his ears with one of your hands, and place a watch in the other hand outside the range of his hearing, then approximate it slowly, asking the patient to speak directly he hears the tick, and then estimate the distance. Examine the other ear in the same way. Ascertain on yourself what is the normal distance at which that particular watch should be heard, and supposing this is 60 inches, and the patient hears with the left ear at a distance of 5 inches, and with the right at 60 inches, then the acuteness of his hearing is represented by the fraction $\frac{5}{60}$. The hearing for the voice is indicated by the distance at which the patient can hear whispered sounds, or by noting whether at 2 yards the patient can hear whispered, ordinary, loud, or shouting conversation.

[1] Trans. Clin. Soc. Lond., 1903.

Weber's Test.—To ascertain whether an impairment of hearing is due to *nerve deafness* or to *obstructive deafness*, test the perosseus hearing by placing a watch or a vibrating tuning-fork on the mastoid bone or on the centre of the patient's forehead. If the deafness is due to disease of the auditory nerve it cannot, of course, be heard in the affected ear, or, at any rate, not as well as in the good ear. This is known as Weber's test negative, and indicates nerve disease. If the deafness is due to obstructive ear disease (the nerve being intact) the sound will be heard quite as well, and probably *better, on the defective side* than on the healthy side (Weber's test positive).

Rinné's Test.—In a normal person a tuning-fork placed on the mastoid bone until no longer heard in that situation by the patient, can still be heard by him if

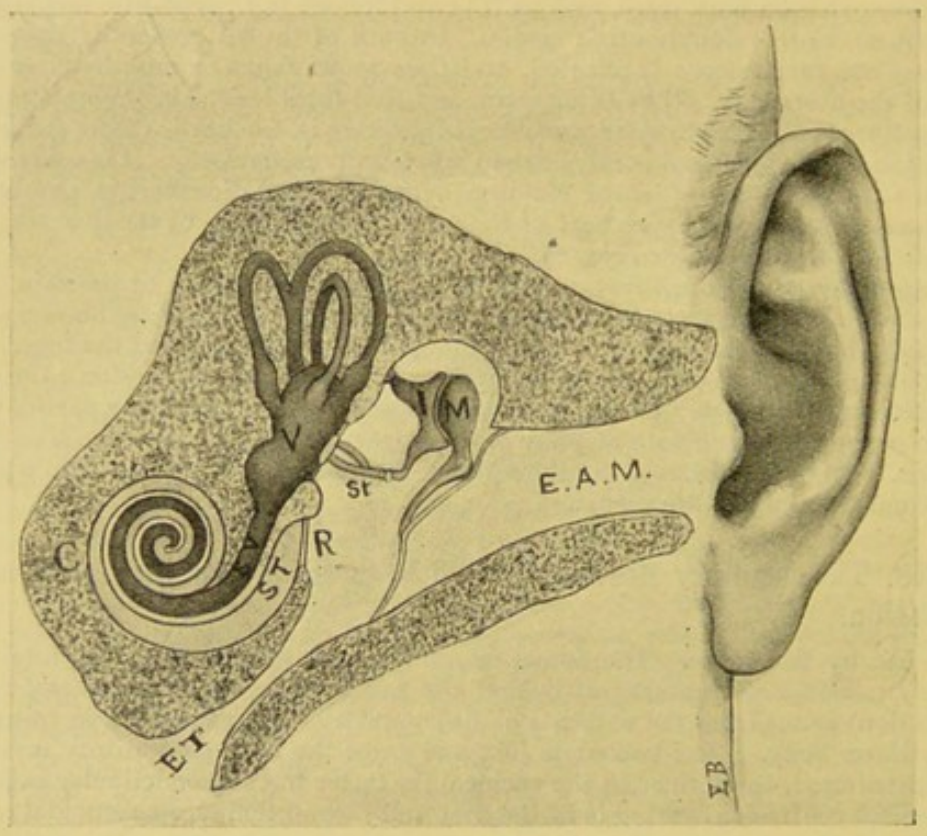

Fig. 171.—Auditory Apparatus (diagrammatic representation) of the left side seen from the front, the internal parts being magnified two-fold.

E.A.M., External auditory meatus, separated by tympanic membrane from tympanum in which R. is situated.
M., on head of Malleus.
I., Incus fixed to wall by its short process, and articulating with the stapes by its long process.
St., Stapes, the foot of which fits into the fenestra ovale.
V., Vestibule consisting of saccule (below) and utricle (above). Into the latter open the three semicircular canals, superior, posterior, and external (or horizontal). The vestibule leads on to the scala vestibuli (S.V.) of the cochlea (C.).
R., Fenestra rotunda leading from the scala tympani (S.T.) to tympanum.
E.T., Eustachian tube.

moved and held opposite the meatus (Rinné's test positive) ; it indicates an absence of middle ear disease. When the middle ear or conducting apparatus is definitely diseased, the tuning-fork cannot be heard opposite the meatus after it has ceased to be heard when held on the mastoid (Rinné's negative).

Galton's Whistle is for testing the upward limit of audition of a patient. Diminution of audition for high-pitched notes occurs in old age and in incipient nerve deafness. *Paracusis Willisii*, "hearing better in a noise," is a characteristic of bilateral middle ear disease. Such patients can hear conversation better in a train

or omnibus. In boiler makers' and some other forms of nerve deafness the converse is true.

Inspection of the Ear.—Note should be made of any discharge and its character (see below), any pain or tenderness over the mastoid (see below), any eczema of the meatus, etc. To examine the *meatus* (which should be done first without a speculum) the auricle should be pulled gently upwards and backwards, the tragus being held forwards by a blunt probe. If a speculum is to be used, the auricle should be held between the middle and ring fingers of your left hand (for the patient's right ear), the speculum being inserted with your right hand inwards and slightly downwards and forwards. The speculum can then be held between the thumb and forefinger of your left hand. It facilitates examination to have a mirror on the forehead as in laryngoscopy (§ 119) to reflect the light from the side of the patient's head. The convexity of the floor of the meatus may be mistaken for an abscess or polypus. Cerumen is of dark colour and soft consistence. The membrana tympani may present indrawing (due to blocking of the Eustachian tube), congestion, thickening, or loss of lustre, atrophic areas, or perforations.

The Naso-Pharynx should be next examined. Note (1) the activity of the palatal muscles, (2) the Eustachian tubes and back of the nose by posterior rhinoscopy, and (3) the patency of each nostril.

The Patency of the Eustachian Tube is usually tested by inflation of the middle ear by *Politzer's* method. The nozzle of the rubber bottle is inserted into one nostril, and both nostrils are then held closed between the thumb and finger of the operator. The patient is then directed to swallow or to say " hic," and at the same moment the air from the rubber bottle is forced into the nose. Deglutition raises the palate and opens the Eustachian tube, and the air, having no other outlet, is forced into it. A tube connecting the ear of the patient with that of the operator will enable the latter to hear an audible " pick " if the middle ear is inflated, and this will reveal the patency of the Eustachian tube. A second point to note is the effect which inflation has upon the symptoms—the hearing, tinnitus, or pain. The hearing is temporarily improved in middle ear or Eustachian disease, made worse in nerve deafness. In *Valsalva's* method of inflation the patient pinches his nostrils firmly, and makes an expiration as if to blow his nose, but without allowing the air to issue. The *Eustachian catheter* is sometimes required to inflate for diagnosis or treatment. It is not a difficult operation, but requires a little practice. Pass it tip downward very gently along the floor of the nose to the edge of the hard palate, the patient being directed to breathe through the nose so that the soft palate may droop. Immediately the tip of the catheter has reached the edge of the hard palate turn it upwards and outwards, and it will enter the Eustachian orifice. It may be aided by the patient swallowing at the same time. The nozzle of Politzer's bag may now be carefully introduced, and inflation performed as before.

§ **618a. Causes of Deafness.**—Two kinds of deafness are recognised by aural surgeons ; **nerve deafness** due to lesions of the auditory nerve, and **obstructive deafness** due to some disease in the middle ear or auditory passages.

Diagnosis of Nerve Deafness and Obstructive Deafness.

Nerve Deafness.	*Obstructive Deafness.*
Diminution of bone-conduction.	Loss of air-conduction only, with negative Rinné.
Loss of hearing for very high-pitched tones.	Better hearing for very high than for very low tones.
Decreased hearing in midst of noise.	Increased hearing in midst of noise.
Hearing for conversation relatively better than for watch.	Hearing for conversation relatively worse than for watch.
Tinnitus occasional.	Tinnitus usual.

The **Causes of Deafness**, slightly altered from Dr. Dundas Grant's arrangement,[1] may be summarised as follows :

Nerve deafness.—*a. Coming on* GRADUALLY, WITHOUT EVIDENCES OF INTRACRANIAL DISEASE, *may be due to*—

1. Toxic causes—*e.g.*, quinine, salicin, tobacco.
2. Chronic anæmia, or congestion of the labyrinth.
3. Paresis from chronic concussion—machinery, guns, etc.
4. Hysterical deafness.

b. Nerve Deafness coming on GRADUALLY, WITH INVOLVEMENT *of other* NERVOUS STRUCTURES.

1. With facial paralysis would indicate either disease of the petrous bone (when there would be discharge of some kind) or gumma, tumour, or pachymeningitis within the skull.
2. With the sixth nerve or spinal accessory—basal pachymeningitis, or a tumour.
3. With hemiplegia of the same side—disease of the temporo-sphenoidal lobe or motor strands below.
4. With crossed hemiplegia—disease of the pons.
5. With hemianæsthesia—disease of the hinder part of the interior capsule.
6. With failure to understand spoken but not written words, without loss of speech (word deafness)—disease of the left superior temporo-sphenoidal lobe.
7. With locomotor ataxy or disseminated sclerosis—degeneration of the auditory nucleus.

c. Nerve Deafness of SUDDEN ONSET.

1. Sudden onset of deafness, with an apoplectiform attack, followed by recurrent vertigo, would indicate Ménière's disease (§ 529*a*).
2. With sudden hemianæsthesia or hemiplegia—hæmorrhage into the temporo-sphenoidal lobe, internal capsule or pons.
3. If dating from a mental or emotional shock—neurotic or hysterical deafness.
4. If dating from forcible syringing, loud noises, explosions, or blows on the head—concussion of the labyrinth. Fracture of the base may be attended by fracture of the capsule of the labyrinth.

d. Nerve Deafness of MODERATELY ACUTE ONSET.

1. Without pyrexia, nerve deafness may come on within a few hours in syphilitic disease of the labyrinth. Acute congestion and acute anæmia of the labyrinth coming on with nausea and giddiness are also causes.
2. With pyrexia, primary inflammation of the labyrinth may supervene with cerebral symptoms during or after various specific fevers, meningeal inflammation of the auditory nerve, or injury.

Obstructive deafness, or deafness due to disease of the middle ear or auditory passages rarely comes on (*a*) SUDDENLY, excepting from impaction of cerumen. If it comes on (*b*) ACUTELY, it is probably due to acute catarrh of the Eustachian tube.

CHRONIC OBSTRUCTIVE DEAFNESS OF SOME STANDING :

a. Without a History of Previous Discharge.

1. If the deafness dated from an acute naso-pharyngeal catarrh, tinnitus is not constant, inflation with Politzer's bag gives some relief, and on inspection, the tympanic membrane is indrawn, opaque, and thickened, the disease is the exudative form of chronic catarrh of the middle ear.
2. If the deafness had an insidious onset, tinnitus is a prominent symptom, inflation gives no relief, and on inspection the tympanic membrane is somewhat opaque, in parts thinned and in other parts thickened, the disease is the sclerotic form of chronic catarrh of the middle ear.

b. With a History of Previous Purulent Discharge.—The deafness is probably due to perforation or cicatrices resulting from suppurative inflammation of the middle ear, and inspection of the drum confirms this.

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[1] *Medical Annual*, 1895.

Combined Obstructive and **Nerve Deafness.**—In this condition it is sometimes a little difficult to make out the exact state of matters. However, such cases may be grouped into those with and those without discharge.

a. If there is a *history or presence of discharge*, the commonest condition which gives rise to it is a suppurative otitis media spreading to the labyrinth. In these circumstances we get signs of nerve deafness gradually supervening on those of obstructive deafness.

b. If there be *no discharge, past or present*, the most usual conditions are: 1. Disease of the cochlea or ankylosis of the stapes supervening on an old chronic catarrh of the middle ear. 2. If the history of nerve deafness precedes the obstructive deafness, the middle ear catarrh has supervened on the nerve deafness.

Pain in the Ear may be due to: 1. Otalgia, when there is no sign of local disease or defective hearing, and a reflex cause such as a bad tooth is present. 2. Disease of the external meatus, such as furuncle or eczema. 3. Disease of the middle ear, when there is deafness, some pyrexia, and examination reveals congestion of the membrane.

PAIN IN THE MASTOID REGION may be due to: 1. Mastoid neuralgia, which sometimes follows old mastoid disease. 2. Accompanied by redness, swelling, and tenderness—inflammation of the mastoid lymphatic gland, periostitis, or abscess. 3. Accompanied by deep throbbing, pain and constitutional disturbance, it may be due to internal mastoiditis, which is often consequent on chronic suppuration.

Pain more or less GENERALISED OVER THE HEAD, accompanied by PYREXIA, may be associated with the following diseases of the ear:

a. Acute Diseases.—1. Acute middle ear suppuration, which is relieved by outlet of pus; 2. mastoiditis; 3. acute meningitis; 4. pyrexia.

b. If associated with a *history of chronic suppuration* from the ear:—1. If the temperature is continuously high, it may be due to retention of pus, extradural abscess, or meningitis. 2. If the temperature oscillate, there may be pyæmia or sinus thrombosis. 3. If the temperature after an initial rise is normal or subnormal, and there are headache, slow pulse, and delayed cerebration, suspect abscess of the temporo-sphenoidal lobe.

Discharge from the Ear.—A STICKY OOZING discharge may be due to eczema of the meatus or condylomata. A HÆMORRHAGIC discharge may be due to vascular granulations or erosion of bloodvessels occurring with middle ear disease, or (rarely) vicarious menstruation in hysteria. An offensive SANIOUS discharge, with fungating granulations, acute radiating neuralgia, and enlargement of the neighbouring glands, is characteristic of malignant disease of the ear.

A PURULENT discharge (*a*) *which is or has been copious*, and associated with deafness from the beginning of the symptoms, is due to acute or chronic suppuration of the middle ear. When associated with chronic suppuration, it may be due to the presence of polypus, granulations, or cholesteatoma, caries of the malleus, incus, or temporal bone, disease of the mastoid antrum or naso-pharynx, or to constitutional causes, such as diabetes mellitus, tubercle, anæmia, or syphilis.

(*b*) A PURULENT discharge *which is not, and never has been, copious*, and deafness, which if present, did not supervene till an interval after the onset of symptoms, may be due to external disease of the ear, acute or chronic.

Tinnitus, or noises in the head, comprise, as Dr. Dundas Grant aptly remarks, "as many varieties of sensation as the patients' powers of description can make them." Tinnitus may be due to impacted wax or disease of the ear, when there is usually associated impairment of hearing and local signs. A *humming* tinnitus, worse on lying down and taking food and stimulant, is due to venous congestion; relieved by lying down and taking food is due to anæmia. Tinnitus taking the form of voices or music is auditory illusion, due to mental disturbance, and often originates in chronic disease of the organ of hearing. A *pulsating* tinnitus (1) checked by compression of the carotid atery is due to arterial congestion of the middle or external ear; (2) checked by compression of the vertebral arteries in the sub-occipital triangle—arterial congestion of the internal ear; (3) audible on auscultating head or ear—probably intracranial aneurysm.

Vertigo and its causes have been dealt with in § 511. Ménière's disease is described in § 529*a*. Pseudo-Ménière's disease may occur with middle ear catarrh.

Orientation, or, as it is sometimes called, equilibration, is the function of that branch of the eighth nerve which goes to the semicircular canals (compare § 503). Disorders of this branch alone are rare, and they cannot well be tested clinically. Disease of this branch or of the semicircular canals give rise to a sensation described by the patient as giddiness. Horizontal or vertical rotatory vertigo, depending upon which of the ampullæ or semicircular canals is involved, occurs in association with this condition.

The *Prognosis* and *Treatment* of these various symptoms depend mainly on the cause in operation. To deal with them individually would be beyond the scope of this work. Nerve deafness is not very hopeful. Any toxic or other cause in operation should, if possible, be removed, and weak galvanism may be tried. Obstructive deafness is, in a large proportion of cases, due to middle ear catarrh, which is more hopeful than nerve deafness. A certain amount of good may be done in chronic catarrh by regular inflation, which the patient can be taught to do himself, and regular inhalation of various remedies such as ammonium chloride. Acute middle ear disease requires prompt measures. Hot fomentations, leeches and incision of the tympanum may be necessary. Syringing with boracic lotion should then be ordered. If the case tend to become chronic, employ constant syringing with boracic lotion, varied with astringent solutions and powders, and inflation by the Eustachian catheter. Mastoiditis and other intracranial symptoms demand surgical interference.

§ **619.** The **Glossopharyngeal Nerve** (the ninth) is the sensory and motor nerve of the pharynx. It supplies the middle constrictor muscle of the pharynx (and the stylo-pharyngeus), and common sensation to the pharynx and the back of the tongue. In the terminal part of its course it contains the taste fibres from the posterior third of the tongue, which ultimately go to join the fifth (§ 615).

Anatomy.—The ninth, tenth, and eleventh nerves arise from a longitudinal collection of cells in the medulla and floor of the fourth ventricle situated beneath or ventrally to the calamus scriptorius. The ninth arises from the upper end, the tenth from the middle and outer part, the eleventh from the lower end. The ninth is joined by ascending branches from the lateral column as low down as the fourth cervical segment, and the spinal part of the eleventh by branches as low as the sixth cervical segment. The spinal accessory is the only one which supplies voluntary muscles, and it should be remembered that it is the accessory portion of this nerve which supplies the larynx and palate through the vagus.

Paralysis of the Glossopharyngeal Nerve is practically never met with alone, and the exact limitation of its functions has never been defined. Paralysis would be indicated by (1) loss of sensation—*i.e.*, insensibility to tickling—of the upper part of the pharynx, and (2) some disturbance of deglutition. (3) The sense of taste in the posterior part of the tongue should be tested in suspected cases.

§ **620.** The **Vagus,** or **Pneumogastric Nerve** (the tenth) has widespread connections with the respiratory passages, heart, œsophagus, and (through the sympathetic) with all the abdominal viscera. It is also the motor nerve to the larynx, pharynx, and palate (by fibres derived from the accessory portion of the spinal accessory, eleventh). The involvement of the vagus proper or its nucleus (fortunately not very common) is evidenced by disturbances of the cardiac rhythm, slowing of the respiration, and perhaps by vomiting, hiccough, sighing, and yawning. The motor effects are evidenced in the palate, larynx, and pharynx.

Laryngeal paralysis has already been referred to in § 127. The crico-thyroid is supplied by the superior laryngeal, and all the other muscles by the inferior laryngeal branch of the vagus, both of which are really derived from the accessory portion of the spinal accessory nerve. Unilateral paralysis of the larynx need not produce any symptoms, and is nearly always due to some local lesion of a *laryngeal branch* of the vagus. Lesions above the level of the nuclei—*i.e.*, when situated in the cortex of *both hemispheres*, or in the descending motor tracts of both sides,

always produce bilateral paralysis (as in the case of the eyes), and this bilateral paralysis may be complete, or affect only the abductors (a dangerous condition, which impedes inspiration). A lesion in the cortex or motor tract of *one side* is unattended by laryngeal paralysis, since the opposite cortex can innervate both cords (Horsley and Semon).

§ **621.** The **Spinal Accessory Nerve** (the eleventh) at its origin consists of two portions. (*a*) The accessory portion rising from the medulla joins the vagus, and gives to it its motor functions for the larynx, palate, and pharynx (*vide* § 620). (*b*) The spinal portion arises from the anterior horns of the first four segments of the cervical cord, and supplies the sterno-mastoid and trapezius (which are also supplied by twigs from the cervical plexus). When the sterno-mastoid is paralysed, the patient is unable to turn his head to the opposite side. When the trapezius is paralysed, he is unable to shrug his shoulders. This portion of the nerve is often involved in obscure irritation producing torticollis.

The Soft Palate is now believed to be supplied from the accessory portion of the eleventh through the vagus and the pharyngeal plexus (Fig. 168). Paralysis of the palate is evidenced by (i.) insufficient elevation of one (or both) sides of the palate during phonation when the patient says " ah." Deviation of the uvula to one side is not a proof of paralysis. (ii.) Inability to pronounce words containing " g " and " b." (iii.) Regurgitation of fluids through the nose in advanced cases. As regards the *Causes* of paralysis of the palate (1) it is more often met with in diphtheritic peripheral neuritis than in lesions of the vagus or spinal accessory. (2) A lesion of the nucleus of the accessory portion of the spinal accessory nerve is met with in bulbar paralysis, and occasionally in amyotrophic lateral sclerosis and progressive muscular atrophy. It is also met with occasionally in other spinal lesions, such as tabes. (3) Lesions of the motor tract above the nucleus, a condition that may occur in hemiplegia due to a cerebral lesion.

§ **622.** The **Hypoglossal Nerve** (the twelfth) is purely a motor nerve to the tongue. Probably the orbicularis oris is also innervated from the *nucleus* of this nerve through the facial. One side may be paralysed in both supra- and infra-nuclear lesions, and is evidenced by the deviation of the tongue when it is protruded *towards* the paralysed side, owing to the unbalanced action of the tongue muscles on the healthy side. When the lesion is bilateral, the tongue is motionless. (1) Cortical and sub-cortical lesions are very often attended by unilateral non-atrophic paralysis and by hemiplegia. Articulation is only temporarily affected. Lesions producing this condition are mentioned under Hemiplegia. (2) Nuclear lesions give rise to atrophic paralysis of one or (more commonly) both sides of the tongue, and the mucous membrane is thrown into folds. In bilateral paralysis, speech, mastication, and deglutition are much impeded. It is generally part of bulbar paralysis (*infra*). Both nuclei are generally affected together by a degenerative lesion ; sometimes one is affected in tabes. (3) In unilateral paralysis of the hypoglossal the lesion is usually situated in the nerve trunk. Sometimes there is a triad of symptoms, as first described by Dr. Hughlings Jackson—unilateral hemiatrophy of the tongue, with paralysis of the palate and the larynx on the same side. Such cases are mostly due to a localised pachymeningitis of syphilitic origin involving the trunks of both the hypoglossal and the accessory portion of the eleventh. A fourth symptom is sometimes added—paralysis of the sterno-mastoid and trapezius from involvement of the spinal portion of the eleventh. Unilateral lesions of the hypoglossal nerve trunk alone are also met with in parotid and other tumours of the neck, and occasionally neuritis.

§ **623. Bulbar Paralysis** (or glosso-labio-laryngeal palsy) is a bilateral, progressive, atrophic paralysis of the lingual, labial, laryngeal, and palatal muscles due to slow degenerative changes affecting the nuclei of the twelfth and the accessory portion of the eleventh nerves. Its description comes suitably at the end of the cranial nerves where several paralyses clinically and anatomically associated have been referred to.

The first *Symptom* usually noticed is alteration of the speech, which becomes thick and indistinct, words being run into one another as though the tongue were too large for the mouth. In reality, however, the tongue becomes wasted, and, as in other atrophic paralyses, is the seat of fibrillary tremors. At first the lingual letters only are difficult, but by-and-by, as the soft palate becomes paralysed, words containing gutturals such as "ground" and "grub" are impossible to the patient. Gradually, as the tongue wastes, the mucous membrane is thrown into folds, and the patient is unable to protrude the organ. (2) The orbicularis oris is simultaneously involved. The patient cannot whistle, and by the falling of the angles of the mouth the expression becomes mournful. The other muscles of the face are unaffected, but he cannot inflate the cheeks, owing to the paralysis of the soft palate. (3) The vocal cords are also paralysed, and phonation, difficult at first, becomes by-and-by impossible beyond a meaningless grunt. (4) Owing to the affection of the soft palate and pharynx, swallowing becomes difficult, and fluids regurgitate through the nose, and the saliva, which the patient cannot swallow, dribbles out of the mouth over the edges of the protruding lower lip. Occasionally the disease is associated with descending sclerosis.

The *Diagnosis* in typical cases is not difficult. Certain combined lesions, such as that referred to under the Hypoglossal Nerve, may present a difficulty, but bulbar paralysis is always bilateral. *Myasthenia gravis* (§ 574) resembles it, but presents certain generalised symptoms. *Pseudo-bulbar paralysis* is due to bilateral disease of the cerebral cortex in the lower part of the ascending frontal convolutions or about the angle of the internal capsules, but this is much slower and often improves, and is not associated with atrophy of the paralysed muscles.

The *Prognosis* is extremely bad. The progress is slow, but sure, and a fatal issue invariably results, usually after a course of about one or two years, from complications or the extension of the disease to the vagus or other cranial nuclei.

The *Etiology* is obscure. The disease is practically confined to persons at or beyond middle life. It may occur in association with, or independently of, progressive muscular atrophy, and the morbid anatomy is probably identical with it. The occasional occurrence of an *acute* form of *bulbar paralysis* (due to acute softening analogous to acute anterior poliomyelitis) makes their identity more complete.

Treatment is unavailing, though antisyphilitic remedies may be tried in cases having a history of syphilis.

THE SKULL AND SYMPTOMS REFERABLE TO IT.

§ **624.** The **Skull** as the brain-case is related to neurology. Scars, exostoses, and the traces of syphilis may be found. The cranial deformities of hereditary syphilis and of rickets are given in the table in § 447 and in § 13.

The *circumference* of the skull is greatest at the level of the external occipital protuberance and glabella.

Average in	male adult	56 cms.
,, ,,	female adult	54 cms.
,, ,,	children aged 12 years	50 cms.
,, ,,	,, ,, 12 months . . .	45 cms.
,, ,,	infants new born	35 to 40 cms.

The average measurement of the naso-occipital arc in the male adult is 35 cms.

The microcephalic and macrocephalic skulls, facial asymmetry, narrow forehead, scapho-cephalic skull (with high vertex and narrow transverse diameter), protruding teeth, or a high narrow arch of the hard palate are all regarded as degenerate or neuropathic evidences, but they are not infallible signs.

External Landmarks of the Brain.—The chief cerebral structures to locate are the fissure of Rolando and the fissure of Sylvius. The *fissure of Rolando* is a most

important landmark, and may be found by drawing a line from the root of the nose to the occipital protuberance. Then mark a point ½ to ¾ inch behind the middle of this line, and draw a second line from that point downwards and forwards at an angle of 67 degrees ; the second line will lie over the fissure. This is conveniently done in practice with soft metal joined at the required angle. The fissure of Sylvius is found by drawing a line from the external angular process of the frontal bone to the occipital protuberance. The fissure starts at a point 1½ inch behind the external angular process. The horizontal ramus of the fissure lies beneath a line drawn from this point to the parietal eminence.

Sir William Macewen attaches importance to the percussion of the skull as an aid to the diagnosis of intracranial tumours.

§ 625. **Hydrocephalus** is a distension of the ventricles of the brain with fluid. It occurs in two forms : (*a*) Congenital or infantile ; and (*b*) acquired or secondary hydrocephalus.

(*a*) Typical cases of Congenital Hydrocephalus may be recognised by the shape of the head, which is enlarged and dome-shaped, projecting all round and beyond the bones of the face. The fontanelles are very wide, and Wormian bones may fill up the gaps between the cranial bones. The face in proportion to the size of the head seems to be extremely small. The orbital plates of the frontal bones are pushed downwards, so that there is exophthalmos, and the eyelids do not cover the sclerotics. The condition dates from birth. It may exist before birth sufficiently to obstruct labour, or it may be so slight as not to be noticed until the child is one or two years old. The circumference of the head of a child of four may reach as much as 25 or 30 inches. The general symptoms consist of bodily weakness and developmental delay, and backward mental condition, which shows itself in the slighter cases as a constant peevishness, and in the graver cases as idiocy. In some cases the intellect is quite normal. Spina bifida, talipes, encephalocele, and other developmental deformities may accompany hydrocephalus. The *Causes* of this form of hydrocephalus are not known.

In slight cases the *Diagnosis* may have to be made from rickets, in which there are (1) a bossed and square shape of the head, (2) a flattened instead of a dome-shaped vertex, (3) evidences of rickets in the other bones of the body.

Prognosis.—Most cases of congenital hydrocephalus die within the first five years of life. Moderate cases sometimes live until the twelfth year. Mild cases of hydrocephalus sometimes do not deteriorate further. The patient lives without other symptoms perhaps for the normal span of life.

(*b*) The symptoms of Acquired Hydrocephalus are less decided. They come on insidiously at any age, and are associated with those of the causal condition. The head does not enlarge if the disease supervenes after closure of the fontanelles, but there are symptoms of cerebral compression (§§ 547 and 551), and sometimes gradual blindness, due to optic neuritis.

Causes.—Acquired or secondary hydrocephalus mostly depends on some obstruction of the veins of Galen, such as that produced by a tumour at the base of the brain, or in the third ventricle, or the iter. It may also result from intracranial inflammation, especially post-basic meningitis (§ 549). Pressure on the middle cerebral or straight sinus, into which the venæ Galeni empty, has the same effect (§ 551).

The *Treatment* of congenital hydrocephalus is not hopeful. Drugs are powerless to promote the absorption of the fluid. In the milder cases the treatment resolves itself broadly into a general strengthening or tonic treatment. In other cases " lumbar puncture," similar to that successfully used in tuberculous meningitis, has been employed with a measure of success in hydrocephalus. A smooth, fine aspirating needle is introduced between the third and fourth lumbar vertebræ, a little to one side of the middle line, to the depth of 2·5 centimetres in children and 5 centimetres in adults. One to one and a half ounces have been removed at a time (Quincke). It is especially worthy of trial if pressure symptoms are present. The treatment of acquired hydrocephalus is not much more promising, unless the cause is removable.

CHAPTER XX

EXAMINATION OF PATHOLOGICAL FLUIDS AND CLINICAL BACTERIOLOGY

In this chapter the methods of obtaining various pathological fluids, how to examine them, and their characters, will be briefly described, and in conclusion, an epitome of the chief bacteriological data required for clinical work will be given.

§ 626. Methods of Obtaining and Examination of Pathological Fluids.—Fluids are obtained by puncture with an ordinary hypodermic needle or with exploring needles, which are made specially strong, and with somewhat larger calibre to permit of turbid fluid entering them. The needle must be boiled before use, and the skin thoroughly cleansed with soap and water, ether, and 1 in 30 carbolic acid. Cocaine or eucaine may be painted over the spot or injected in the vicinity, or the part may be frozen with the ethyl chloride spray.

The **pleural** cavity is best explored at the ninth space just behind the posterior axillary line, or at the site where localised dulness is present. Paracentesis thoracis is described in § 85.

Pericardial fluid is obtained by inserting the needle ½ to 1 inch to the left of the sternal margin in the fourth or fifth interspace. Unless we are certain that the pericardium is very full of fluid, it should never be attempted. Paracentesis pericardii has been described in § 38.

The **peritoneal** cavity may be explored for fluid in the middle line, to avoid bloodvessels, or at the side, above the anterior superior iliac spine. The puncture must be made over a dull area. **Paracentesis abdominis** for the removal of ascitic fluid is performed with an ordinary trocar and cannula. A many-tailed bandage is first placed under the patient, and the tails are tightened up one by one to maintain the abdominal pressure as the fluid drains away. Without this precaution the patient may suffer collapse from the rapid dilatation and congestion of the splanchnic area. Some advise a small preliminary incision through the skin, or the trocar may be thrust through the skin in the middle line. The fluid drains away through a long rubber tube into a pail by the side of the bed on the floor. The end of the tube should dip under an antiseptic. Before puncturing in the middle line care must be taken to ensure the bladder being empty. Southey's tubes may be employed instead of the trocar and cannula. They act as efficaciously though more slowly.

Lumbar Puncture is employed chiefly for diagnosis of the character of the fluid in increased exudation into the sub-dural space, and also for relief of certain forms of hydrocephalus. Children should be anæsthetised. For adults local anæsthesia alone is sufficient. Employ an antitoxin needle, the most convenient length of which is about 3 inches. The patient, if an adult, should sit up, leaning well forward, with the head low. Draw a line across the patient's back at the level of the highest margin of the iliac crests. This line intersects the vertebral column at the tip of the fourth lumbar spine. Having carefully sterilised the skin at about this spot, it is rendered anæsthetic by an ethyl chloride spray. The operator places his left index finger on the fourth lumbar spine as a guide, and then, with

the right hand, pushes in the needle about ½ inch below and ½ inch to the right of this spot. It should be inclined in an inward and upward direction. In the case of children, the best position is with the child on its left side, the back being bowed as much as possible. The fluid normally runs out drop by drop, but when under increased pressure, as in hydrocephalus and meningitis, it will sometimes spurt out; the intrathecal pressure may be roughly gauged by the rate of flow. For examination (p. 923) 5 c.c. are enough, but for treatment (p. 930) 10 or 15 c.c. may be removed.

How to Examine Pathological Fluids.—Fluid which has been withdrawn from the pleural, pericardial, or peritoneal cavity, or from a cyst, should be placed in a conical vessel, and allowed to settle, and its physical and chemical characters noted. The *Colour* should be observed, whether clear, turbid, or red, as with blood, or opaque from abundant cellular constituents, or excess of fat, as in chylous exudations. The *Reaction* is usually alkaline. The *Odour* tells little as a rule, except in the case of cysts which have been in the vicinity of the intestinal canal. The *Consistence* is usually watery; if viscid, mucin is present, as in a distended gall-bladder or an ovarian cyst. Clotting after standing indicates the presence of fibrin. The *Specific Gravity* must be noted. The *Deposit* must be examined under the microscope. Blood-cells, white and red, may be found, the former being more readily seen by the addition of acetic acid, which brings out the nuclei. Cancer cells may be found in malignant disease of the wall of the cavity, and are distinguished from epithelial cells by being large, and occurring in groups. The discovery of hooklets (Fig. 75, p. 379), or pieces of lining membrane of hydatid cysts, crystals of cholesterin, leucin or tyrosin, or various germs, or the amœba of dysentery, or actinomyces, may clear up a difficult diagnosis. Fatty acid crystals in groups favour the diagnosis of cancer.[1]

The *Chemical Examination* is then proceeded with, after filtering the fluid. Albumen, nucleo-albumen, and mucin are tested for as in urinary examination. For quantitative examination of albumen the fluid is first diluted to a specific gravity of 1,008, and acidified with acetic acid, then measured in Esbach's tube (§ 276). To detect urea, first remove any albumen by heating and filtering; then evaporate the fluid to small bulk, and test as in urine. To detect sugar, render the fluid slightly acid, boil, filter, evaporate to small bulk, and test as in urine.

When the fluid is suspected to come from a pancreatic cyst, it is tested to ascertain if it can digest albumen in an alkaline medium. The suspected fluid is added to milk. After the casein is precipitated, the Biuret test is tried. (A few drops of dilute solution of copper sulphate, and excess of caustic potash, causes a rose-red hue in the presence of peptones.) If present, it proves that the fluid has a peptonising power, and nothing except pancreatic fluid can peptonise in an alkaline medium. In cysts of some standing, the power of peptonising may be lost owing to the destruction of the trypsin.

Characters of the Pathological Fluids (table, p. 922). In the pleural, pericardial, and peritoneal cavities, inflammatory effusions (exudates) are difficult to distinguish from dropsical effusions (transudates). Cardiac dropsy is more albuminous than renal dropsy. A fluid containing more than 4 per cent. of albumen is probably of inflammatory origin; a fluid with less than 2½ per cent. of albumen is probably dropsical. Blood in distinct amount in these cavities is highly suggestive of cancer, but has sometimes been found with tuberculous disease of the pleura and with peritonitis associated with cirrhosis of the liver. In such a case, examination of the deposit, revealing cancer cells or tubercle bacilli, would settle the diagnosis. A few blood-cells, sufficient to give the fluid a rosy tinge, may occur with

[1] Simon, "Clinical Diagnosis," p. 466. London: Kimpton, 1896.

Table of Physical and Chemical Characters of Pathological Fluids.

Constituents, etc.	*Serous Exudation as in Ascites or Pleurisy.*	*Transudation.*	*Hydatid Cyst.*	*Pancreatic Cyst.*	*Ovarian Cyst.*	*Hydronephrosis.*	*Cerebro-spinal Fluid (Normal).*	*Distended Gall-Bladder.*
Colour.	Turbid yellow.	Clear yellow or greenish yellow.	Clear and colourless or opalescent.	Colourless. Often mixed with blood.	Variable—clear yellow to black.	Clear and watery.	Clear.	Clear or stained with bile.
Specific gravity.	Usually over 1018.	Rarely over 1015.	1006 to 1010.	Variable—often 1008 to 1010.	From 1002 to 1055 (low sp. gr. points to cyst of broad ligament).	1008 to 1020.	1006 to 1007.	Low.
Spontaneous coagulability.	Very marked. Clots in 24 hours or earlier.	Slight. May clot after long standing.	Absent.	Absent.	Absent. Consistence variable—watery to colloid.	Absent.	Absent.	Absent.
Chemical composition.	Alkaline. Much albumen, 4 per cent. or more (serum alb. and globulin). Small quantities { Sugar. Uric acid.	Alkaline. Albumen 2½ per cent. or less (serum alb. and globulin).	Alkaline. No albumen or negligible quantity. Much sodium chloride. Succinic acid.	Digests albumen in alkaline medium. Serum albumen present.	Alkaline. Albumen (serum alb. and globulin, and *metalbumen*).[1]	Traces of albumen, urea, and uric acid.	Trace of albumen and albumose. Small amount pyrocatechin, a reducing agent.	Mucin present; may contain bile.
Microscopic *Examination.*	Very little débris. { red blood cells. leucocytes. endothelial cells.	Even less débris than in serous exudation—similar constituents.	Characteristic hooklets, scolices, cyst membrane, hæmatoidin crystals.	Often cholesterin crystals.	Cylindrical ciliated epithelial cells, squamous cells, colloid concretions, red blood-cells, leucocytes, fat globules, cholesterin, fatty acid crystals, and hæmatoidin.	Sometimes renal epithelial cells, uric acid crystals.		
Remarks.	May be variable quantity of pus present. Occasionally it is red with blood as in cancer.[2]	May be sanguineous, and, rarely, chylous.	Diagnosis made by microscopic examination.	The ability to digest albumen is a positive test; but in very old cysts the power is lost.	Metalbumen and ciliated cells the diagnostic points.	Only diagnostic point is presence of renal cells, and these may be absent.	In meningitis the fluid is cloudy, with more albumen. Micro-organisms found aid diagnosis.	

[1] Metalbumen is thus tested for. Acidify fluid with acetic acid, boil and filter to remove other forms of albumen. Add to the filtrate 3 times its bulk of alcohol. Stand for twenty-four hours. Filter. Squeeze out precipitate and suspend in water. Filter. Filtrate has following reactions: (i.) On boiling it becomes turbid, but no precipitate falls; (ii.) on adding acetic acid no precipitate forms; (iii.) on adding acetic acid and strong sulphuric acid a violet colour is produced; (iv.) on adding acetic acid and pot. ferrocyanide the fluid is thick and yellow.

[2] Ascitic fluid usually shows the characters above described, "hæmorrhagic ascites," where blood is found in the fluid, occurs chiefly in cancerous peritonitis, and has been considered a fatal sign. Middleton reports cases of chronic peritonitis associated with hæmorrhagic ascites in alcoholics, who recovered.—*Lancet*, 1899, ii., p. 1286.

simple acute inflammation. The character of the cells in a pleuritic fluid may aid the diagnosis of the cause of the effusion (cyto-diagnosis); thus, an excess of lymphocytes points to tuberculous pleurisy; the predominance of polynuclear cells points to septic or other causes. The special characters of the various fluids and cysts are given in tabular form; but a few words should here be said about the cerebro-spinal fluid.

Cerebro-Spinal Fluid should be clear, colourless, containing a trace of albumen and albumose. Fehling's solution is reduced (? by pyrocatechin). When the fluid which runs out is turbid, it indicates meningitis of some kind, and cover-glass films should be made to study the organisms present. For microscopic examination it is convenient to centrifuge and collect the deposit (p. 929). There may be found the pneumococcus or the diplococcus intracellularis of cerebro-spinal meningitis or the diplococcus of infantile post-basic meningitis. The trypanosome has been found in sleeping sickness. In negative cases, where tubercle is suspected, a guinea-pig may be inoculated with the fluid. The characters of the cerebro-spinal fluid in the different forms of meningitis are shown in the following table:

Form of Meningitis.	*Appearance of Fluid.*	*Cells.*	*Films.*	*Culture.*
Tuberculous.	Clear.	Lymphocytes.	Tubercle bacilli.	Sterile.
Cerebro-spinal Fever. (*a*) Ordinary type.	Turbid, if early. Clear, if late.	Polymorphs, if early. Mixed, if late.	Diplococcus of Weichselbaum.	Diplococcus of Weichselbaum.
(*b*) Post-basic type.	,,	,,	Diplococcus of Still.	Diplococcus of Still.
Pneumococcal.	Turbid.	Polymorphs.	Pneumococci.	Pneumococci.
Staphylococcal and *Streptococcal.*	Turbid.	Polymorphs.	Staphylococci and streptococci.	Staphylococci and streptococci.
Syphilitic.	Clear.	Lymphocytes.	Nil.	Sterile.

For CYTO-DIAGNOSIS, or diagnoses by the cellular elements in the cerebro-spinal fluid, 5 c.c. should be centrifuged for five minutes. After the supernatant fluid has been poured away, take a scraping of the tube turned upside down, spread on a slide, fix by heat, stain with methyl blue, and mount. Normal cerebro-spinal fluid by this method will not show more than two or three lymphocytes in a microscopic field of a magnification of 400 diameters (say 1 or 2 per cubic millimetre), sometimes none, and no polymorphs. (1) In acute infective meningitis (brain or cord) excess of both polymorphs and lymphocytes is found. In cerebral abscess without involvement of the meninges there is no excess (Purves Stewart). (2) In tabes dorsalis and G.P.I. there is so large an excess of lymphocytes (20 or 100 per field as compared with 2 or 3) as to constitute a diagnostic feature. Thus may functional diseases of the nervous system be excluded.

§ **627. Bacteriological Examination.**—The subjects of serum-therapy and immunity have been dealt with in §§ 386 to 388. Here will be described in brief outline the methods of detecting and the appearance of the various microbes commonly met with in clinical work. For laboratory work the student must consult a special treatise.

The necessary APPARATUS consists of (1) slides and cover-glasses thoroughly cleansed, as described in the Examination of Blood (§ 395). (2) A pair of Cornet's forceps, made so that they hold the cover-glass *horizontally* when laid on a table. (3) Two platinum points fixed to the end of glass rods. (4) Sterile swabs (which

are said to be obtainable from any medical officer of health). To make them, twist a piece of cotton wool round the end of a stiff copper wire about 8 inches long. Place this in a test tube, and plug its mouth with cotton wool. The end of the wire should pass through the plug. The tube, with its contained swab, should then be sterilised by heat. (5) Stains—methylene blue, eosin, carbol-fuchsin, Gram's stain, and other reagents. (6) A good microscope, including a $\frac{1}{12}$ inch oil-immersion lens. (7) Wright's capsules (*vide* Fig. 108, § 387) are used for obtaining a sample of blood or serum for the Widal test. For bacteriological examination of the blood a sterilisable syringe is required. (8) A few tubes of nutrient media, such as gelatin, agar, and blood serum, and a small incubating oven are useful in many instances.

Method of Examination suitable for Clinical Work.—(1) With a looped platinum wire, which has been thoroughly cleansed by heating in the Bunsen burner or spirit flame, select a minute portion of the material to be examined, and smear this on the cover-glass with the end of the wire, or place another cover-glass on the top of the first, and slide them apart (see Blood, § 398). A thin film is thus formed. Take care not to touch the cover-glasses with the fingers. If the material be too thick, dilute it with a drop of sterilised water. (2) Let the film dry in the air, or by holding it high above the flame, and fix by passing it rapidly through the flame three times. (3) Put a drop or two of the selected stain (previously filtered) on the cover-glass, or float the cover-glass, film downwards, in a watch-glass containing the stain. The time of leaving the film in the stain depends on the stain employed, but in most cases one to three minutes is sufficient. (4) Rinse the cover-glass *thoroughly* in water, and dry by blotting paper or gentle heat high over the flame. After using the carbol-fuchsin stain, the cover-glass should be passed through a 1 in 4 solution of sulphuric acid (which decolorises everything except the tubercle bacillus and certain other microbes), then rinsed again in water and dried. The preparation may then be mounted in a drop of water and examined, or mounted in Canada balsam as a permanent preparation. A drop of cedar oil is placed on the cover-glass, and the oil-immersion lens employed.

Motile Organisms may be examined unstained, and their movement readily seen. When examining unstained preparations, remember to reflect the light with the plane mirror of the microscope placed in a slanting position, without the Abbé condenser. Place a drop of sterilised water on a cover-glass, and touch it with the platinum wire, which has previously been dipped in the material to be examined. The glass may be placed over the hollow of a hollow slide, thus forming the " hanging-drop "; but the movements can be as readily seen in the hollow or cell formed by drawing a ring of vaseline round the edge of the cover-glass.

Gram's Method.—Certain organisms possess the characteristic reaction that their stain is fixed by iodine. Place the cover-glass, film side downwards, in a solution of aniline-gentian-violet for five to eight minutes. This is prepared by adding 1 part of aniline oil to 20 of distilled water, shaking thoroughly, and filtering the mixture. To this add drop by drop a concentrated and filtered alcoholic solution of gentian violet, in the proportion of 1 part of aniline water to 9 parts of the stain. (2) Place the film in a solution made up of 1 gramme of iodine and 2 grammes of potassium iodide in 300 c.c. of water. This acts as a mordant, and fixes the stain in the microbe. The film becomes black. (3) Place the film in alcohol till it becomes a faint grey, everything except the microbe becoming decolorised. (4) Wash, dry, and mount as usual, or counterstain the other structures with eosin before mounting.

(*a*) The **Sputum** is examined for tubercle bacilli, the pneumococcus, the influenza bacillus, oïdium albicans of thrush, the bacillus of plague in cases of pneumonic plague, anthrax, the actinomycosis fungus, and the micrococcus tetragenus (see below). Pyogenic organisms are found when pus is present.

(1) The **Tubercle Bacillus** (Plate IV., Fig. 1) is slightly curved, measuring 2 to 5 μ long. Three go to the diameter of a red blood corpuscle. Often two lie

PLATE IV.

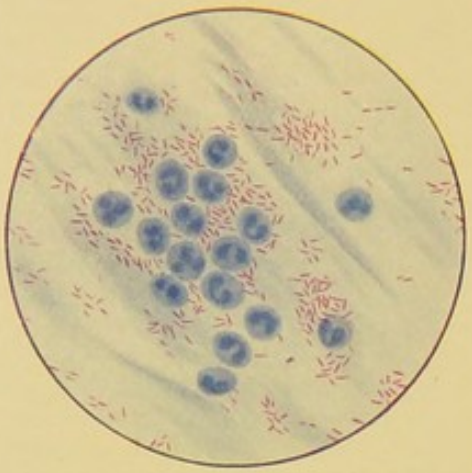

FIG 1.—TUBERCLE BACILLI.

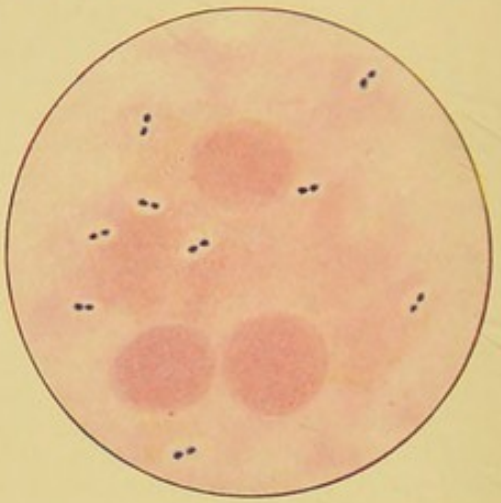

FIG. 2.—PNEUMOCOCCI.

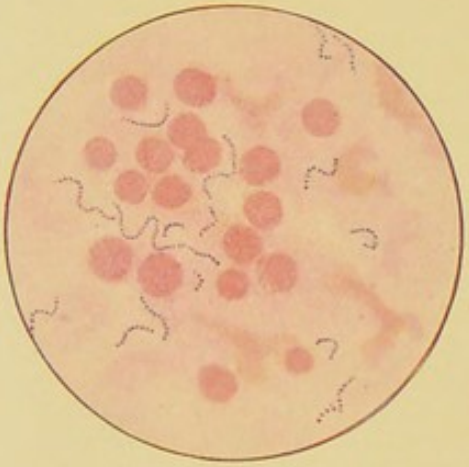

FIG. 3.—STREPTOCOCCUS PYOGENES.

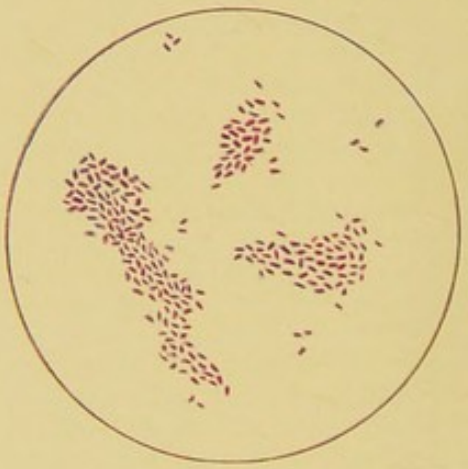

FIG. 4.—TYPHOID BACILLI.

together at an angle, like the letter V. Clear spaces resembling spores are often seen within them after staining. The tubercle bacillus is present in the *sputum* in all cases of active pulmonary tuberculosis. It is usually most plentiful in the thicker and most purulent part of the sputum. If hæmoptysis be present, select a dark red clot after the attack has subsided. Take the minutest portion possible, and press it between two perfectly clean cover-glasses. Separate these by sliding the one over the other, and fix the films thus obtained by passing them two or three times through the flame of a spirit lamp. When the bacillus is hard to find (as in cases where very few are present), boil about 100 c.c. of the sputum with double the amount of water, to which 6 drops of a 10 per cent. solution of caustic potash have been added. Add water from time to time, to allow for evaporation ; then set aside for twenty-four or forty-eight hours, or centrifugalise, and examine the sediment for the tubercle bacillus.

The tubercle bacillus may be detected by its characteristic staining reaction. In the Ziehl-Nielsen method, which is the best, carbol-fuchsin is dropped on the cover-glass film, which is then heated over a flame until the liquid steams ; or heat the stain in a watch-glass, and drop the cover-glass, film downwards, into it, and leave in for three minutes. Then decolorise in 33 per cent. or 25 per cent. sulphuric acid, and wash the glass in water. If a counter-stain be desired, in order to examine other elements of the sputum, leave the glass in a watery solution of methylene blue for two or three minutes. Then wash in water, dry, mount, and examine.

Other microbes which stain by the Ziehl-Nielsen method are the leprosy and the smegma bacillus. Leprosy bacilli occur in clumps, never isolated like tubercle. The smegma bacillus may readily be mistaken for tubercle in the examination of the urine. To distinguish it, place the cover-glass in absolute alcohol for three hours, then a quarter of an hour in chromic acid. Stain as usual, and wash in alcohol, after decolorising in acid. The smegma bacillus is thus decolorised.

(2) **The Pneumococcus** (Fraenkel's pneumococcus) (Plate IV., Fig. 2) is a coccus usually of lanceolate shape, often in pairs (diplococcus), sometimes in short chains. A capsule surrounds it, which is less deeply stained in stained preparations, and is absent in cultures. Sometimes more than two cocci occur within one capsule. They cause croupous pneumonia, but are also found in healthy saliva. Select a rust-coloured part of the sputum when the disease is at its height. It is found in the lungs in the red hepatisation state of the disease. The pneumococcus is easily *stained* by ordinary aniline dyes. It can also be seen by Gram's method, which distinguishes it from Friedlander's pneumo-bacillus (also found in pneumonic sputum), the latter being decolorised by Gram. By the carbol-fuchsin method the cocci should be deeply stained and the capsule a fainter red, or altogether unstained.

(3) The **Influenza Bacillus** (Pfeiffer's bacillus) is found in the sputum and nasal mucus of patients suffering from influenza. It occurs as a minute rod, not quite 1 μ long, lying in pairs or groups, and may be mistaken for diplococci. It stains with ordinary dyes. It may also be found in the blood at the acme of the disease.

(4) The **Micrococcus Tetragenus** occurs as cocci in groups of four in a capsule. It is found in the sputum coming from phthisical or bronchiectatic cavities. It stains with the ordinary dyes and with Gram's method.

(5) The presence of the **Actinomycosis** fungus is suspected when small sulphur coloured granules, visible to the naked eye, are seen in the sputum or in purulent discharge from tumours. Such granules under the microscope are seen to consist of a large number of threads radiating out from a centre like a fan, each thread having club-shaped ends. They stain well with Gram's method. The pus has a characteristic greenish yellow colour.

(*b*) A **False Membrane** occurring on the throat or palate must be examined for the presence of the **diphtheria bacillus.** Taking care not to touch the lips, cheek, or mouth inside, touch the suspected patch with a sterilised swab or brush, or remove a piece of the membrane with

a platinum loop or forceps. Transfer this to a sterilised test tube, or spread thinly on a cover-glass, fix, stain, and examine. Frequently so many micro-organisms are present that it is impossible to make out satisfactorily the diphtheria bacillus. In such cases cultures must be made by inoculating tubes containing blood serum or coagulated egg albumen with the platinum needle or swab. On such media, kept at body temperature, the diphtheria bacillus grows rapidly in characteristic colonies of punctiform spots which fuse into opaque, uneven streaks. The growths can be examined under the microscope within twenty-four hours.

The bacillus is known as the Klebs-Loeffler bacillus. It is of variable size and shape, average 2 μ long. It is straight or slightly curved. Some of the rods are knobbed, thicker at one end. Some stain uniformly, others irregularly. They lie in clusters, never in chains. There are no spores. Streptococci are frequently found with it, but are no index of the severity of the case. Loeffler's methylene blue is recommended as the most suitable stain, because by its use the characteristic beaded appearance or polar staining of the rods is well shown ; but any other basic stain may be used. It also stains by Gram's method.

Hoffman's *pseudo-diphtheria bacillus* resembles the true diphtheria bacillus, but it stains more deeply, and has not the polar staining which gives to true diphtheria its characteristic club shape. The best method of distinguishng the Klebs-Loeffler bacillus is by *Neisser's method*. There are two stains. The first consists of methylene blue, 1 gramme ; rectified spirit, 20 c.c. ; distilled water 950 c.c. ; and glacial acetic acid, 50 c.c. The second, or contrast stain, contains 2 grammes of vesuvin in 1,000 c.c. of distilled water. Immerse the film in No. 1 for four seconds ; rinse in distilled water ; immerse in No. 2 for four seconds ; rinse in distilled water ; blot and dry. Diphtheria bacilli will be stained brown with an occasional bright blue granule.

(*c*) **Pleural Effusion** may be examined after fixing and staining on a cover-glass or a slide, the latter giving a larger area for examination. Serous effusions show few or no germs : the diplococcus pneumoniæ and tubercle bacillus are almost the only germs which have yet been found in a serous pleurisy. Sometimes in tuberculous pleurisy no bacilli are found. The rheumatic diplococcus may be present, but as yet cannot be differentiated by clinical methods, only by inoculation of animals. The germs found in purulent effusions are the diplococcus pneumoniæ and the streptococcus and staphylococcus, and the tubercle bacillus.

(*d*) **Pus** obtained from any part of the body should be examined, microscopically (p. 924) or by culture, for the pyogenic organisms. Such are :

Gram-positive.	*Gram-negative.*
Staphylococci.	Gonococci.
Streptococci.	Pneumobacilli.
Pneumococci.	B. Coli communis.
Anthrax bacilli.	B. Typhosus.
Diphtheria bacilli.	B. Mallei (of glanders).
Tetanus bacilli.	Plague bacilli.
Actinomycosis.	Diplococcus Intracellularis.

(1) **Streptococci** (Plate IV., Fig. 3) occur in long chains.

(2) **Staphylococci** are large cocci occurring in small groups or bunches. Both are pyogenic organisms, and are found in pus in various situations. They stain with ordinary dyes and with Gram's method.

(3) The **Gonococcus** appears as a diplococcus, the paired cocci facing each other with concave surfaces. They are usually found inside the pus corpuscles lying in groups. They are most readily found in the pus coming from a gonorrhœal urethral, or conjunctival discharge. They stain with the ordinary dyes, and not with Gram's method. It is well to use a counterstain, such as eosin, which stains the corpuscles in which the gonococci lie.

(4) Buboes may require to be examined for the bacillus of **Plague** (bacillus pestis). The bacillus is short and thick, 2·3 μ long by 1·7 μ broad. It often lies in pairs, and so may be mistaken for a diplococcus. The pus discharged from buboes generally contains no bacilli.

The **Malignant Pustule** due to anthrax can rarely be shown to contain the anthrax bacillus. In doubtful cases examine the blood (see below).

The purulent discharge from a tumour caused by the actinomyces fungus contains tiny granules, which are described above under the Sputum.

(*e*) It is difficult to examine the **stools** for any particular organism because of the enormous number of germs present. Two, however, may be mentioned here—the Cholera Vibrio and the Amœba of Dysentery. Various kinds of bacillus coli may always be found.

The **Cholera Vibrio** (Koch's comma bacillus) is obtained from the mucoid masses in the choleraic stools, but in at least half the cases no definite result is gained by simple staining methods, and the organism can be recognised only by submitting it to a number of cultural and other tests. The vibrios are curved rods slightly shorter than tubercle bacilli. They lie in rows, end to end pointing in one direction. They stain with ordinary dyes, not by Gram's method.

The **Amœba of Dysentery** is a spherical body, sometimes of a pale greenish colour refracting the light strongly. It is 30 to 40 μ diameter, has a granular endoplasm and a clear ectoplasm, and is motile. The amœba should be looked for in the stools at once while the discharges are still alkaline. The glass slides, cover-glass, and the microscopic stage should be warmed. A drop of mucus from the stools, or a scraping from the wall of a tropical abscess is diluted with warm saline solution and placed on the slide. The amœba will then be seen actively moving. The amœba is easily killed ($\frac{1}{5000}$ solution of quinine arrests the movement), and if examined when the stools are acid, the amœba will have no movement.

(*f*) The **urine** may require to be examined for the tubercle bacillus, the gonococcus, the typhoid bacillus, and the colon bacillus. The tubercle bacillus is described under sputum, and the gonococcus under pus above. The diplococcus of rheumatism has been found in the urine, but requires experimental testing for its detection. The tubercle bacillus is sometimes very difficult to detect in urine. It is best to draw off the urine by a sterilised catheter to prevent the entrance of the smegma bacillus. Stand the urine for twenty-four hours in a conical glass, or centrifugalise it. Examine the sediment carefully, staining, etc., as with sputum.

The **Typhoid** bacillus (Eberth's bacillus) (Plate IV., Fig. 4) is short, thick, and mobile with rounded ends. It is 3 to 4 μ long, and 1 μ broad, and is flagellated.

Sometimes several rods lie end to end. It may be very difficult to distinguish from the bacillus coli communis, but is known by its characteristic cultures. It stains with ordinary dyes, and not with Gram's method. The typhoid bacillus is the cause of enteric fever, and occurs in the spleen, in the blood, in the rose-coloured spots, in the urine, and in the stools. However, one is not likely to succeed in finding them by microscopic examination except from culture growths, and expert knowledge is required to succeed in obtaining a pure culture.

The **Colon Bacillus** (b. coli communis) may also be found in the urine, causing cystitis, pyelitis, and pyelonephritis, apart from any other pathogenic germ. It has also caused suppurative peritonitis. It closely resembles the typhoid bacillus, but is somewhat shorter. It is motile, but has fewer flagellæ than the typhoid bacillus. It stains with ordinary dyes, not with Gram's method. It can only be distinguished by cultural and other tests.

g. The **Blood** may be examined for certain parasites—malaria, trypanosoma, filaria, kala-azar, etc. (§ 399). The pyogenic organisms (see table, p. 926), the typhoid bacillus, the rheumatic diplococcus, the anthrax bacillus, and the micrococcus of Malta fever have been found in the blood. The spirillum of relapsing fever is always present in the blood in the febrile periods. Kala-azar and some other microbes are difficult to detect unless a large quantity of blood is available, and for this purpose the blood may be drawn off with a hypodermic syringe from the spleen or liver.

In suspected cases of **Anthrax** examine the blood, as the anthrax bacillus is only rarely obtained from the pustule or local lesion, from which the disease spreads to infect the blood throughout the body. The bacillus is one of the largest, about 5·2 μ long. When cultures are examined, spores are seen inside and around the filamentous segments. It stains with the ordinary dyes and with Gram's method.

The **Micrococcus Melitensis** is the cause of Malta fever. It is a minute coccus ($\frac{1}{3}$ μ), motile, sometimes lying in chains. It can be obtained by puncturing the spleen with a fine sterilised hypodermic needle with due precaution, and withdrawing a little blood. This is a dangerous method, and the presence of the disease may be diagnosed by the agglutination tests. The coccus stains with the ordinary dyes, and not by Gram's method.

Widal's Serum Reaction.—This is the usual method of examination of the blood employed as a bacterial test in typhoid fever, but it may be used for other infections. It depends upon the fact that the patient who is the subject of an infection has in his serum an antibody (agglutinin) which causes the infecting micro-organisms to run into clumps, and renders them immobile. In typhoid fever the reaction is not present until after the tenth day of the disease, but persists afterwards for several months, or even years. To perform this test, blood is collected from the patient's finger by means of Wright's capsules (see § 387, Fig. 108), and allowed to stand. The serum separates out, and is used for the test. By breaking off the end of the capsule access is obtained to the serum, just as is done in estimating the opsonic index (see § 387). The test may be performed in the following ways :

(1) *The Hanging Drop Method.*—The serum is transferred to a watch-glass. A platinum wire loopful is placed on a coverslip, and to it are added 9 loopfuls of normal saline. The drops are then mixed so that the dilution is 1 in 10. A loopful of this is transferred to another coverslip, and to it 9 loopfuls of salt solution are added. The second serum dilution is 1 in 100. A loopful of a twenty-four-hour-old broth culture of typhoid bacilli is now put on to each of two clean coverslips. To the first a loopful of the serum, diluted 1 in 10, is added, making a dilution 1 in 20 ; to the second a loopful of the serum, diluted 1 in 100, making a dilution of 1 in 200. Each coverslip is inverted over a hanging drop chamber, the edge having been vaselined, to prevent evaporation, and the bacilli are watched under the high power of the microscope. Clumping should occur within half an hour with a dilution of 1 in 200.

(2) *Sedimentation Test.*—This is best performed by means of Wright's pipettes (see Fig. 172). A mark is made on the lower end of the pipette, about 1 inch from the extremity, with a soft aniline pencil. By means of the rubber cap normal saline is drawn up to the mark. This quantity is now blown out on to an ordinary glass slide by squeezing the cap, so that it forms a pool at the left-hand top corner. This process is repeated until six such pools, each containing an equal quantity of normal saline, are placed on the slide in two rows of three each. The patient's serum is now drawn into the pipette up to the mark, and this is now blown out again, and mixed with the saline in the first pool, giving a dilution of serum of 1 in 2. This mixture is drawn up to the mark and blown out into the second pool, giving in this case a dilution of 1 in 4. This is repeated for the third, fourth, fifth, and sixth pools, giving dilutions respectively of 1 in 8, 1 in 16, 1 in 32, and 1 in 64. The next step is to take an emulsion of a twenty-four hours' culture of typhoid bacilli. This is also drawn up to the mark on the pipette and added to the first pool, the process being repeated for all the other pools in turn. The pools now contain dilutions of serum of 1 in 4, 1 in 8, 1 in 16, 1 in 32, 1 in 64, and 1 in 128. Samples of each of these are now drawn up into the pipette. The 1 in 4 dilution is drawn up to the mark, and a bubble of air let in ; then the 1 in 8, then a bubble of air, and so on in turn the 1 in 16, 1 in 32, 1 in 64, and 1 in 128 dilutions, a bubble of air intervening between each. The pipette is then sealed, and stood aside in an upright position for half an hour. It is then examined by the naked eye. In the dilutions in which clumping has occurred a definite sediment will be seen to have settled at the bottom of the column, whilst if no clumping has occurred, in any particular dilution that dilution will be uniformly cloudy. If the reaction is *positive, the* 1 *in* 64 *dilution should clump definitely in half an hour.*

Fig. 172.—Wright's Pipette. Supplied by Parke, Davis and Co.

(3) Either of these tests may be employed, using emulsions of dead bacilli. Sir A. E. Wright has described[1] how the practitioner can carry about with him, without risk, dead cultures of both typhoid and Malta fever organisms, and thus readily diagnose the presence or absence of these diseases.

h. The **Cerebro-Spinal Fluid** may contain pyogenic germs, tubercle bacilli, the pneumonia diplococcus, the diplococcus of epidemic cerebro-spinal meningitis and that of posterior basic meningitis, and the trypanosoma, in all of which its examination is useful for diagnosis ; compare tables, p. 923. The cerebro-spinal fluid is obtained by lumbar puncture (pp. 921 and 923), and a film is made and fixed in the usual way, preferably after centrifugalisation. Pyogenic organisms, tubercle, the pneumococcus, and the trypanosoma have been described.

The diplococcus of epidemic cerebro-spinal meningitis is known as the **Diplococcus Intracellularis** of Weichselbaum. The cocci lie in pairs, within the pus corpuscles, thus resembling the gonococcus. It stains with ordinary dyes, and not with Gram's method.

The **Diplococcus of Posterior Basic Meningitis,** which occurs in infants, is found in the cerebro-spinal fluid, but is rarely found within the pus cells, and has not the lanceolate shape of the pneumonia diplococcus. It tends to be grouped in pairs, resembling sarcinæ or tetragenus. It stains with ordinary

[1] *Brit. Med. Journ.*, 1897, vol. i., p. 1214.

dyes, and not with Gram's method. It has more vitality than the diplococcus of epidemic cerebro-spinal meningitis, but is probably only a slightly modified form of that organism. The chief distinctions are a specific agglutination reaction and a different opsonic power of the blood.

i. For the **Spirochæte Pallida (Treponema Pallidum)**, the edge of the primary sore or other syphilitic lesion is gently scraped with the edge of a microscopical slide or sterile scalpel until it begins to bleed. The blood is then mopped off with some absorbent material until it is replaced by a drop of serum. A film of this serum is made and fixed in absolute alcohol. It may be stained by Leishman's method, but a better method is by Giemsa's stain. One drop of this stain is added to 1 c.c. of tap-water, which is then spread over the film, or the film may be floated face downwards in the stain. The staining takes from five to twenty-four hours. The excess should then be washed out by tap-water, and the film dried and mounted in the ordinary way. A description of the organism is given under Syphilis (§ 404, and Fig. 119).

Note on the Therapeutic Uses of Lumbar Puncture (p. 921).—Recently this method has attracted attention, not only for purposes of diagnosis (pp. 923, 930), but for treatment. The withdrawal of 5 to 15 c.c. of cerebro-spinal fluid relieves not only intrathecal but intracranial pressure ; probably it acts in other ways also—*e.g.*, by " flushing " the theca vertebralis. The fluid spurts out when the pressure is too high, and in some cases it may be allowed to run till it comes in drops.

1. In *tuberculous meningitis* it relieves the symptoms of cerebral compression, and cases of recovery have been recorded after repeated tapping. Possibly it acts in the same way as tapping in cases of tuberculous peritonitis.
2. In *epidemic cerebro-spinal meningitis* 10 c.c. may be removed, and 10 c.c. of a 1 per cent. solution of lysol introduced.
3. In many cases of *Coma* and *Cerebral compression*, from whatever cause arising, it is a useful palliative measure.
4. Inaccessible *cerebral tumours* so treated result in the temporary relief of the symptoms of cerebral compression ; not more than 5 or 6 c.c. should be withdrawn at a time, for fear of hæmorrhage into the tumour substance.
5. In *fracture of the base of the skull* cases of recovery have been reported.
6. Cases of *uræmic coma* or *convulsions* have been promptly relieved, and have ultimately recovered (McVail, *British Medical Journal*, 1903).
7. In cases of *tetanus* the antitoxin may be more advantageously introduced by lumbar puncture than hypodermically, and may be given with stovaine and morphine.
8. Cases of *strychnine-poisoning* have been treated by eucaine introduced in this way.
9. Anæsthesia of the lower limbs and trunk—*spinal anæsthesia*—is produced by the introduction of cocaine, stovaine, and novocaine by lumbar puncture after withdrawing a larger amount of fluid than that introduced.

FORMULÆ

(referred to as F. in the text)

OF USEFUL PRESCRIPTIONS.

The proportions given are those for one adult dose unless otherwise stated.

(1) Balneum Alkalinum.

Add two large handfuls (8 ozs.) of common washing soda to 30 gallons of water at 95° F. The patient remains twenty minutes in first bath, and the time is gradually increased up to forty-five minutes. Put to bed in blankets.

Valuable for chronic rheumatism—daily for six weeks. At first the pains are increased. Also useful for chronic eczema.

(2) Balneum Creol. vel Picis.

℞ Creolin ℥ss. to ℥ij. *or* Liquor Carbonis Deterg. . ℥j. to ℥iv. in 20 to 30 gallons of water, well stirred.

Useful for pruritus, prurigo, chronic eczema, and all itching affections.

(3) Balneum Sulphuris.

℞ Potass. Sulphurat. .	℥ij.
Acid. Acet. dil. (or vinegar) . . .	Oss.
Warm water . .	20 to 30 gals.

(4) Balneum Vaporis Hydrarg.

℞ Hydrargyri Subchloridi . . ℥ss.

Volatilise beside ½ pint of water for twenty minutes in a Lee's lamp. Seat the patient in a cane-bottomed chair. Tie the curtain round the neck, and put a blanket over the shoulders. Light the lamp, and place it beneath the chair. Leave the patient in from twenty to forty minutes.

(5) Shower Bath.

See that the shower is the required temperature[1] before placing the patient in the curtain, then turn on *suddenly*, because the chief therapeutic value of the shower is by its shock. It should not be given where marked heart disease exists. Valuable in hysteria and neurasthenia.

[1] Cold, 50° F.; tepid, 75° F.°

(6) Hot-Air Bath.

Remove the clothing and lay the patient in a blanket, adjust the wicker framework and cover it with three or four blankets, which should come up well under the chin. Light the torch under the chimney, and let it continue to burn until the patient perspires very freely. He may remain for fifteen or twenty minutes longer. The temperature inside the wicker work should be between 170° and 200° F. Valuable in renal disease.

Collunaria.—Nasal douches. Should be used at a temperature of about 100° F. Half a tumblerful to be injected along the floor of each nostril night and morning. The patient should be directed *not* to blow the nose forcibly immediately afterwards.

(10) Col. Pot. Chlor. Comp. (C. L. Throat Hosp.).

℞ Pulv. Pot. Chlor. .	.
Pulv. Boracis .	.
Sodæ Bicarb. .	. āā gr. xij.
Pulv. Sacch. Alb.	. ad ℥j.

To be added to half a tumblerful (℥v.) of water. Useful in all forms of chronic rhinitis.

(11) Collunarium Ac. Carbol. (vel Boracis).

℥j. of the Glycerinum Carbol. or Boracis (B.P.) to 10 fluid oz. of water.

(13) Enema Hazelinæ.

Hazeline and water . Of each 1 oz.

Administered slowly by means of a glycerine syringe for internal piles.

(14) Enema Nutriens.

(*a*) Peptonised milk, 2 to 4 oz.
(*b*) Strong beef-tea, 2 oz., and meat juice, 1 oz.
(*c*) One egg, 1 oz. brandy, 2 oz. strong beef-tea *or* 2 teaspoonfuls of meat extract; and 1 teaspoonful of Bullock's acid glycerin of pepsin; mix and divide into two enemata.

These may be administered alternately.

(15) Garg. Acidi Carboli c̄ Cocaina.

℞ Acidi Carbolici . . . ℥j.
Cocainæ Hydrochlor . . gr. viij.
Glycerini Boracis . . ℥ss.
Aquam Rosæ . . . ad ℥xij.

For acute pharyngitis and laryngitis.

(16) Gargarisma Acidi Tannici.

℞ Glycerini Acidi Tannici . ℥j.
Aquam . . . . ad ℥j.

For relaxed throat and to check bleeding after tonsillotomy.

(17) Gargarisma Boracis Compositum.

℞ Pulveris Aluminis .
Pulveris Boracis . . āā gr. vijss.
Tincturæ Myrrhæ . ♏v.
Mellis . . . . gr. x.
Aquam . . . ad ℥j.

(18) Gargarisma Chlorini.

℞ Potassii Chloratis . . ℥ij.
Acidi Hydrochlorici Fortioris . . . . ℥j.

Cork and set aside for five or ten minutes, then add—

Glycerini . . . . ℥iv.
Aquam . . . . ad ℥xij.

To be freshly prepared. A very prompt and efficacious remedy for scarlatinal and diphtheritic sore throat, hospital sore throat, and follicular tonsillitis. For children it should be applied with a brush every two hours.

(19) Gargarisma Potassii Chloratis.

℞ Potassi Chloratis . . ℥iss.
Aluminis . . . . ℥iss.
Aquam . . . . ad ℥x.

(20) Glycerinum Carbolici Fort.

℞ Acidi Carbolici . . . ℥iij.
Glycerini . . . . ℥j.

For applications in endocervicitis.

(21) Glycerinum Ferri.

℞ Liquoris Ferri Perchloridi . ℥iv.
Glycerini . . . . ℥iss.
Aquam . . . . ad ℥j.

To paint the tonsils in recurrent tonsillitis.

(22) Guttæ Auribus.

℞ Plumbi Acetatis . . gr. j.
Tincturæ Opii . . . ℥j.
Glycerini . . . . ℥j.
Aquam Rosæ . . . ad ℥j.

Used warm for inflammation or pain of the external auditory meatus.

Glyc. Ac. Carbol. (B.P.) answers the same purpose.

(23) Guttæ Dentibus (Dr. Gaye).

℞ Olei Caryophylli . . ♏v.
Etheris . . . . ♏xv.
Tincturæ Opii . . . ♏xx.
Glycerini . . . . ♏xx.

To drop in a painful hollow tooth, or to apply on cotton-wool. Washing out the mouth with warm carbolic lotion (1 in 100) often relieves toothache.

(24) Injectio Hydrarg. Hypoderm (Durham).

℞ Ammonii Chloridi . . gr. viij.
Hydrargyri Perchloridi . gr. xvj.
Aquam Destillatam . . ad ℥j.

Dissolve. ♏x. contain ½ gr. Hydrargyri Perchloridi. Inject ♏ii. to ♏v. into the muscle of buttock or shoulder every second, third, or fourth day.

Another formula consists of Hydrarg. Succinimide, 2%, with Cocain. Hydroch. ½ %; dose, 20 to 30 ♏. It is less painful and equally efficacious.

(25) Injectio Morph. cum Atropinâ.

℞ Atropinæ Sulphatis . gr. ½
Morphinæ Acetatis . gr. viij.
Sol. Ac. Carbol. (1 %) . ad ℥iv.

(♏ v. contain $\frac{1}{150}$ gr. and ¼ gr. respectively.)

(27) Injectio Pilocarpinæ.

℞ Pilocarpinæ Nitratis . . gr. v.
Sol. Ac. Carbol. (1 %) . ♏ 100.

Dose 2 to 5 ♏. Rapid diaphoretic. ♏j. useful to reduce arterial tension. Also used in nerve deafness, gr. $\frac{1}{10}$ gradually worked up to gr. ½ daily, resting for two hours afterwards (Field, *Brit. Med. Journ.*, 1889, vol. i., p. 471; and 1890, vol i., p. 1125).

(28) Injectio Strychninæ Co.

℞ Strychninæ Sulph. . . ½
Atrop. Sulph. . . . ⅛
Acidi Borici . . . 1
Acidi Carbol. (pur.) . . 1
Aquam Destillatam . ad 100.

♏v. as often as required, to relieve the prostration and bodily discomfort in vascular dilatation, such as occurs in neurasthenia and morphia craving.

(30) Linctus Communis.

℞ Oxymellis Scillæ . . ℥ij.
Syrupi Tolutani . . ℥ij.
Tincturæ Camphoræ Compositæ . . . . ℥j.
Aquam Destillatam . . ad ℥j.

Dose. — ½ a teaspoonful for bronchitic cough.

(31) Linctus Sedativus.

℞ Liquoris Morphinæ Hydrochloratis . . . ♏xl.
Acidi Hydrocyanici Diluti . ♏viij.
Acidi Hydrochlorici Diluti. ♏xvj.
Glycerini . . . . ℥iv.
Aquam Destillatam . . ad ℥j.

Dose.—℥i. Relieves the cough of phthisis. A teaspoonful in a wineglassful of water may be sipped every five minutes until the cough is relieved.

LINIMENTA.

(32) Linimentum Ammoniæ Co.

℞ Olei Terebinthinæ . . ℥x.
Liquoris Ammoniæ Fortioris . . . . ℥iv.
Saponis Mollis . . . ℥v.
Camphoræ. . . . gr. 80
Spiritûs Vini Methylati . ℥ij.
Aquam . . . . ad ℥x.

A stimulating embrocation, resembling Elliman's.

(33) Lin. Bellad. cum Chlorof.

℞ Lin. Belladonnæ, Lin. Chloroformi . . . āā ℥j.

Very valuable for lumbago and other rheumatic affections; sprinkled on lint. Do not cover with oil-silk, or it will blister. A very expensive liniment, which for hospital purposes can be made equally well with Lin. Terebinth instead of Lin. Chlorof.

(35) Lotio Alkalina Opiata (Fuller).

℞ Potassii Carbonatis . . gr. 200
Liquoris Opii Sedativi . ♏ 400
Glycerini . . . ℥x.
Aquam . . . . ad ℥x.

Apply warm to painful rheumatic joints.

(36) Lotio Calaminæ.

℞ Calaminæ . . . . gr. xx.
Zinci Oxidi . . . gr. xx.
Glycerini . . . . ℥ss.
Liq. Calcis . . . ℥iij.
Aquam Destillatam . . ad ℥j.

(37) Lotio Calcis cum Oleo (Carron oil).

℞ Acidi Carbolici . . . ♏x.
Liquoris Calcis . . .
Olei Lini . . . . āā ℥ij.

Mix well. Invaluable for burns.

(38) Lotio Capillaris.

℞ Tincturæ Cantharidis. . ℥ss.
Liq. Ammon. Fort. . . ℥ss.
Glycerini . . . . ℥ss.
Aquam . . . . ad ℥j.

Rub into the head night and morning for baldness.

(39) Lotio Creolin.

Creolin ℥j. to Oj. of water, to wash skin diseases, before applying ointments.

If the skin is dry and harsh, add Glycerin ℥ii. to Oi.

(40) Lotio Evaporans (vel Frigida).

℞ Liquoris Ammonii Acetatis
Spiritûs Vini Methylati . āā ℥v.
Aquam . . . . ad ℥x.

Local application for acute gout, sprained joints, or headache. Invaluable for insect bites and stings. N.B.—Must not be covered by oil-silk.

(41) Lotio Kerosinæ.

℞ Kerosene . . . . ℥ij.
Olive Oil . . . . ℥j.

For pediculi capitis.

(42) Lotio Plumbi cum Zinco.

℞ Lot. Plumbi Subacet. Dil. . ℥j.
Zinci Oxidi . . . gr. xx.
Glycerini . . . . ℥ss.
Aquam . . . ad ℥j.

Invaluable for acute eczema.

(43) Lotio Hyd. cum Acid. Carbol.

℞ Hyd. Perchlor. . . . gr. iss.
Acid. Carbol. . . . ♏xx.
Glycerini . . . . ℥ss.
Sp. Vini Rect. (or Meth.) . ℥j.
Aquam . . . . ad ℥j.

(44) Lotio Saponatus Kalinus (Hebra).

℞ Saponis Viridis . . . ℥j.
Spir. Vini . . . . ℥ij.

Dissolve by gently heating, filter, and add Ol. Berg. and Ol. Lav. aa ♏xv. For cleansing the head from Seborrhœa.

(45) Mistura Ammon. Chlor. Co.

Ammon. Chlor. . . . gr. xv.
Liquoris Morph. Hydrochlor. . . . . ♏v.
Aquam Chlorof. . . . ad ℥j.

4tis horis in facial neuralgia.

(46) Mist. Aperiens Effervescens.

No. 1.

℞ Sodii Bicarbonatis . . gr. xxx.
Sodæ Tartaratæ . . gr. xl.
Potassii Tartratis . . gr. xl.
Aquam . . . . ad ℥j.

No. 2.

℞ Acidi Tartaric . . . gr. xxx.
Aquam . . . . ad ℥j.

(47) Mistura Bromidi et Iodidi.

℞ Potassii Iodidi . . . gr. v.
Ammonii Bromidi . . gr. xv.
Spirit Ammon. Aromat. . ♏xv.
Syr. Aurantii . . . ℥j.
Aquam . . . . ad ℥j.

(48) Mistura Buchu Alkalina.

℞ Potassii Nitratis . . gr. v.
Potassii Bicarbonatis . . gr. xv.
Sp. Etheris Nitrosi . . ♏xx.
Tincturæ Nucis Vom. . ♏v.
Tincturæ Hyoscyami . . ♏xx.
Inf. Buchu . . . ad ℥j.

A valuable diuretic mixture. Also for cystitis and prostatic retention.

The Buchu infusion should be freshly prepared. The best way to administer this drug is to procure some ℥x. packets of the leaves; place a packet in a pint jug, previously warmed, add 1 pint of boiling water, stir, and, after standing one hour, decant. One pint to be taken in twenty-four hours.

(49) Mistura Calcis Chloridi.

℞ Calcis Chloridi . . . gr. xx.
Tincturæ Aurantii . . ℥j.
Aquam Chlorof. . . ad ℥j.

Valuable for *pruritus* from any cause.[1] Should be given three times a day after meals in gradually increasing doses. In *hæmorrhage*, uterine or pulmonary, should be given every two to four hours.

(50) Mistura Capsici.

℞ Tincturæ Capsici . . ♏iij.
Tincturæ Nucis Vomicæ . ♏v.
Spiritûs Chloroformi . . ♏xv.
Aquam Menthæ Piperitæ . ad ℥j.

For flatulence. An excellent "pick-me-up" for alcoholics.

(51) Carlsbad Mixture.

℞ Sodii Bicarbonatis . . gr. xv.
Sodii Chloridi . . . gr. v.
Sodii Sulphatis . . gr. xxx.
Magnesii Sulphatis . . ℥j.
Aquam Menthæ Piperitæ . ad ℥j.

A morning purgative draught for plethora, obesity, gout, and rheumatic gout.

(52) Mistura Cascarillæ Composita.

℞ Tincturæ Nucis Vomicæ . ♏v.
Tincturæ Scillæ . . . ♏x.
Oxymel Scillæ . . . ♏xx.
Infusum Cascarillæ . . ad ℥j.

Chronic bronchitis and emphysema.

(53) Mistura Diaphoretica.

℞ Spiritûs Etheris Nitrosi . ℥ss.
Liq. Ammonii Acetat. . ℥ij.
Aquam Camphoræ . . ad ℥j.

Diaphoretic and febrifuge.

(54) Mistura Digitalis Co.

℞ Tincture Digitalis . . ♏v.
Ammonii Carbonatis . . gr. iij.
Potassii Nitratis . . gr. v.
Tincturæ Nucis Vomicæ . ♏v.
Aquam Chloroformi . . ad ℥j.

For mitral disease with failing compensation.

(55) Mistura Diuretica.

℞ Potassii Acetatis . . gr. xv
Spiritûs Etheris Nitrosi . ♏xv.
Spiritûs Juniperi . . ♏xxx.
Decoctum Scoparii . . ad ℥j.

[1] The pathology of itching and its treatment by large doses of Calcium Chloride.—The *Lancet*, August 1, 1896.

(56) Mistura Ether Ammon.

℞ Sp. Ammoniæ Aromat. . ♏xx.
Spiritûs Etheris . . ♏xx.
Spiritûs Chloroformi . . ♏xx.
Aquam . . . . ad ℥j.

For cardiac failure. More efficacious if accompanied by hypodermic injection of Liq. Strychn. ♏iij.

(57) Mistura Expectorans.

℞ Ammonii Carbonatis . . gr. v.
Tincturæ Scillæ . . . ♏xv.
Spiritûs Etheris . . . ♏xv.
Tincturæ Strophanthi . ♏iij.
Infusum Senegæ . . ad ℥j.

For acute bronchitis in the second stage.

(58) Mistura Expectorans Infantilis.

℞ Ammonii Carbonatis . . gr. ½
Tincturæ Scillæ. . . ♏xx.
Vini Ipecacuanhæ . . ♏iv.
Aquam . . . . ad ℥j.

For a child one year old. Given every hour in broncho-pneumonia, it may cause emesis, which is beneficial.

(59) Mistura Ferri Laxans.

℞ Ferri Sulphatis . . . gr. ij.
Magnesii Sulphatis . . ℥ss.
Acidi Sulphurici Diluti . ♏iij.
Essentiæ Menthæ Piperitæ. ♏v.
Infusum Calumbæ . . ad ℥j.

(60) Mistura Filicis.

℞ Ext. Filicis Maris . . ℥j.
Syr. Zingib. . . . ℥j.
Tincturæ Quillaiæ . . ℥ss.
Aquam Chloroformi . . ad ℥iss.

(61) Mistura Hydrargyri Biniodidi.

℞ Potassii Iodidi . . . gr. v.
Liq. Hydrarg. Perch. . . ℥j.
Tincturæ Cardamomi Compositæ . . . . ♏xv.
Aquam . . . . ad ℥j.

(62) Mistura Olei Morrhuæ.

℞ Olei Morrhuæ . . ℥ss.
Liquoris Calcis . . ℥iij.
Liquor. Calcis Sacch. . ♏xxxvj.
Pulv. Trag. Co. . . gr. vj.
Olei Caryophylli . . ♏½
Olei Cassiæ . . ♏½

An agreeable cod-liver oil emulsion.

(63) Mist. Potas. Citratis Effervescens.

No. 1.

℞ Potassii Bicarbonatis. . gr. xx.
Aquam . . . . ℥j.

No. 2.

Acidi Citrici . . . gr. xv.
Aquam . . . . ℥ss.

An agreeable effervescing vehicle for quinine and other drugs.

(64) Mistura Ricini Calcis.

℞ Olei Ricini. . . . ℥ss.
Liquoris Calcis . . . ℥ss.
Tincturæ Quillaiæ . . ♏xv.
Syrupi . . . . ♏xx.
Olei Menthæ Piperitæ . ♏j.

℥i. every hour for diarrhœa and unhealthy stools in children.

(65) Mistura Sedativa Infantilis.

℞ Ammonii Bromîd . . gr. iv.
Tincturæ Belladonnæ . ♏iv.
Glycerini . . . . ♏x.
Aquam . . . . ad ℥j.

A harmless sedative for a child of one year old.

(66) Mistura Stomachica.

℞ Magnesii Carbonatis . . gr. x.
Sodii Bicarbonatis . . gr. xv.
Acidi Carbolici pur. . . ♏j.
Tincturæ Rhei Compositæ . ♏xv.
Infusum Calumbæ . . ad ℥j.

Tea-drinker's dyspepsia and pyrosis.

(67) Mistura Strychninæ.

℞ Liq. Strych. Hydrochlor. . ♏iij.
Acidi Nitro-Hydrochlorici Diluti . . . . ♏v.
Tincturæ Capsici . . ♏j.
Tincturæ Lavand Co. . ♏v.
Aquam . . . . ad ℥j.

The tonic for old age.

(68) Mistura Pro Tussi.

℞ Tincturæ Camph. Co. . ♏x.
Vini Ipecacuanhæ . . ♏v.
Oxymellis Scillæ . . ℥j.
Aquam Anisi . . . ad ℥j.

For chronic bronchitis.

(70) Nebula Alkalina

℞ Sodii Bicarbonatis . . gr. xij.
Boracis . . . . gr. xij.
Acidi Carbolici . . . gr. iv.
Glycerini . . . . ♏xl.
Aquam . . . . ad ℥j.

To be sprayed into the anterior nares for ozœna and ulceration.

(72) Nebula Zinci Chloridi.

℞ Zinci Chloridi . . . gr. x.
Acidi Hydrochlorici Diluti . ♏j.
Aquam Destillatam . . ℥j.

Sprayed into the anterior nares for chronic rhinitis; may be also used as a throat spray for chronic laryngitis. Gr. xx. to ℥i. cuts short an attack of acute laryngitis, and should be used as follows: Pull the tongue well forward and spray interior of larynx; apply cold compress to throat at night, and in the morning let patient inhale chloroformi ♏xx., Tr. Benzoin Co. ad ℥i. in a pint of boiling water every few hours. (See Vapores, p. 938).

(75) Pasta[1] Resorcin Comp.

℞ Resorcin . . . . ℥j.
Zinci Oxidi . . . ℥ij.
Pulv. Amyli . . . ℥ij.
Petrol. Moll. . . . ℥j.

Mix well. A valuable protective paste. For chronic conditions, Salicylic or Carbolic Acid (20 to 60 grs.) may be added.

(76) Pasta Sulphuris.

℞ Sulph. Precipit. . . . ℥j.
Zinci Oxidi . . . ℥j.
Silicious earth . . . ℥ss.
Adep. Benz. . . . ℥j.

A protective paste for chronic skin diseases.

(77) Pigmentum Acidi Chromici.

℞ Acidi Chromici . . . gr. 50
Aquam Destillatam . . ad ℥j.

To paint unhealthy ulcers.

(78) Pigmentum Acidi Salicylici.

℞ Acidi Salicylici . . . ℥j.
Extracti Cannabis Indicæ . gr. viij.
Collodii Flexilis . . . ℥vj.
Ætheris . . . ℥ij.

Paint on every night to remove corns.

(79) Pigmentum Cadis.

℞ Ol. Cadi . . . . ℥j.
Spiritûs Vini Meth. . . ℥j.
Sapo Mollis . . . ℥j.

For psoriasis of scalp and lupus erythematosus.

[1] Pastes are stiff ointments which act as protectives and drying agents.

(80) Pigmentum Creosoti.

℞ Creosoti . . . . ♏ij.
Acidi Salicylici . . . gr. xx.
Collodii . . . . ℥j.

Very efficacious for lupus erythematosus.

(81) Pigmentum Iodi Compositum.

℞ Iodi . . ℥j. } By
Acidi Carbol. Liq. ℥iv. } weight.

Rub together in a warm mortar and dissolve, warming if necessary. Resembles Costor's Paste. For intractable ringworm; apply once a week to the scalp with a hog's bristle brush.

(82) Plaster Mulls (Emplastra), with composition as follows, are obtainable at most large pharmaceutical chemists:

(*a*) Empl. Ac. Salicylic (5 to 15 %).
(*b*) Empl. Ac. Salicyl. (5 %), and Ac. Carbol (10 %).
(*c*) Empl. Ac. Salicyl. (10 to 50 %), and Creosote (20 to 50 %).
(*d*) Empl. Hydrarg. (10 to 30 %).
(*e*) Empl. Hydrarg. (20 %), and Ac. Carbol. (7½ %).
(*f*) Empl. Hydronaphthol (5 and 10 %).
(*g*) Empl. Ichthyol (5 and 10 %).
(*h*) Empl. Ichthyol (10 %), and Chrysarobin (10 %).
(*i*) Empl. Resorcin (10 and 15 %).
(*j*) Empl. Zinci Ox. (20 %), and Sulphur (1 %).
(*k*) Empl. Zinci Ox. (10 %), and Ac. Salicyl. (5 %).
(*l*) Empl. Zinci Ox. (10 %), and Ichthyol (5 %).

(83) Pilula Asafœtidæ cum Valeriana.

℞ Asafœtidæ . . . . gr. iij
Zinci Valerianatis . . gr. j.

Flatulence, "nervousness," and the manifestations of hysteria generally.

(84) Pilula Digitalis Composita.

℞ Pulveris Digitalis . . gr. j.
Pulveris Scillæ . . . gr. j.
Pilulæ Hydrargyri . . gr. j.

Valuable in cardiac dropsy, and as a diuretic in ascites. It is apt to salivate unless the bowels are acting regularly.

(85) Pilula Elaterini.

℞ Pulveris Elaterini Compositi . . . . gr. ij.
Extracti Hyoscyami . . gr. ¼.

$\frac{1}{20}$ gr. Elaterini in each pill. Diuretic and drastic pill for ascites.

(86) Pilula Ferri Alkalina (Blaud).

℞ Ferri Sulphatis . . . ℥j.
Potassii Carbonatis . . ℥j.
Pulveris Tragacanthæ . gr. xij.
Glycerini . . . . ♏x.

Misce bene ; fiant pilulæ, xxiv. Should be freshly prepared. Two a day, increased to eight, after meals.

(87) Pilula Ferri Comp.

℞ Ferri Sulph. . . .
Zinci Valerian . . . āā gr. j.
Ext. Aloes Barbadensis .
Ext. Nucis Vomicæ . . āā gr. ¼.

For the amenorrhœa and anæmia of hysterical girls.

(88) Pilula Colchicinæ.

℞ Colchicinæ . . . gr. $\frac{1}{60}$
Ext. Nuc. Vom. . . gr. ¼
Ext. Hyoscyami . . gr. ½
Ext. Gentianæ . . . gr. j.

Twice or thrice a day for acute gout.

(89) Pilula Hydrargyri Composita.

℞ Pilulæ Hydrargyri . . gr. j.
Pulveris Ipecacuanhæ . gr. ½
Pilulæ Rhei Compositæ . gr. ij.

May be given every night to reduce arterial tension.

(90) Pilula Podophylli Composita.

℞ Resinæ Podophylli . . gr. j.
Pulveris Ipecacuanhæ . gr. j.
Hydrargyri Subchloridi . gr. j.
Extracti Hyoscyami . gr. ij.

A useful liver pill in hepatic congestion.

(91) Pilula Aloini c̄ Ferro.

℞ Aloini . . . . gr. j.
Ext. Cascaræ . . . gr. ij.
Ferri Sulph. Exsic. . . gr. j.
Ext. Bellad Vir. . . gr. ¼
Ext. Nucis Vom. . . gr. ¼
Ext. Hyoscyami . . gr. j.

To make one pill or tabloid (coat with chocolate).

(95) Pulvis Alterativus.

℞ Hydrargyri cum Cretâ . gr. j.
Pulveris Cinnamoni Compositi . . . . gr. j.
Pulveris Rhei . . . gr. ij.
Magnesii Carbonatis . . gr. ij.

Dose for a child one or two years old.

(96) Pulvis Guaiaci Composita.

℞ Pulveris Guaiaci Resinæ
Sulphuris Precipitati .
Magnesii Carbonatis . āā gr. xx.

℥i. to ℥iii. every evening. A valuable alterative for gout and rheumatism; and for sciatica (gr. xx., t. d.).

(97) Pulvis Mirabilis.

℞ Bismuth. Carbonatis . . gr. v.
Sodii Bicarbonatis . . gr. v.
Pulv. Rhei . . . gr. j.
Pulv. Nucis Vomicæ . . gr. ½
Pulv. Cinnamon. Co. . . gr. 1½.

To be taken before meals. For dyspepsia.

(98) Pulvis Scammonii cum Hydrargyro.

Pulveris Scammonii. . gr. iij.
Hydrargyri Subchloridi . gr. j.
Pulveris Zingiberis . . gr. ij.

Dose for a child two years old. An aperient powder for thread-worms.

(99) Asthma Powder.[1]

℞ Pulv. Lobeliæ Inflatæ. .
Pulv. Stramonii . .
Pulv. Hyoscyami Exot. .
Pulv. Foliæ Belladonnæ .
Pulv. Corticis Cascarillæ .
Black Tea . . .
Pulv. Potassii Nitratis . āā viij.

Break up and mix thoroughly all the ingredients excepting the potassium nitrate. Dissolve the potassium nitrate in as small a quantity of water as possible, mix it thoroughly with the broken leaves, and dry the whole thoroughly. May be inhaled while smouldering, smoked in a pipe, or made up into cigarettes.

(100) Tabloids.

One of the chief objections to medicines in the form of tabloids appears to be their insolubility, and the possibility of their not passing into the stomach until some considerable time after they are swallowed. Dr. J. S. Bristowe ("Clinical Lectures and

[1] Formula kindly supplied by Dr. James Ross, Liverpool.

Essays on Diseases of the Nervous System") narrates a case in which small tabloids containing morphia were found in the folds of the œsophagus after death, and I have met with similar cases. A draught of water or some food should always follow their administration.

(101) Unguentum Calam. cum Oleo.

℞ Calaminæ . . .
Zinci Oxidi . . . āā gr. x.
Olei Lini . . . ℥ss.
Adeps Benz . . . ℥j.

A soothing ointment. Useful for pruritus.

(102) Unguentum Creolini Comp.

℞ Creolin . . . . ℥j.
Unguenti Hyd. Ammon. . ℥iij.
Sapo Mollis . . . ℥j.
Pet. Mollis . . . ad ℥j.

For psoriasis or chronic eczema (in certain stages).

(103) Unguentum Metallorum.

℞ Unguenti Hydrargyri .
Unguenti Plumbi Carbonatis . . .
Unguenti Zinci Benzoati . āā ℥iv.

For irritable or acute eczema.

(104) Unguentum Petrolati Comp.

℞ Hydrargyri Ammoniati . gr. x.
Liquoris Carbonis Detergentis . . . . ℥ss.
Paraffini Mollis . . . ℥j.

A mild tar and mercury ointment, useful in many skin diseases.

(105) Unguentum Salicylici et Carbolici.

℞ Ac. Salicylici . .
Ac. Carbolici . . āā gr. xxx.
Vaselin . . . ad ℥j.

Stimulating ointment for chronic skin affections.

(106) Unguentum Sulphuris Co.

℞ Sulph. Sublimat. . . gr. xxx
Acid. Carbol. . . . ♏ viij.
Sapo Mollis . . . ℥ss.
Adipis Benz. . . . ad ℥j.

For acne. Should be rubbed in night and morning. In obstinate cases Sapo Mollis ℥ii. to ℥iii. and more sulphur may be added.

(110) Vapores (Inhalations).

Directions.—A teaspoonful to be added to a pint of boiling water, to be inhaled for five minutes every night and morning from a narrow-necked jug or suitable inhaler. In this way use Tr. Benzoini Co. as an expectorant and local sedative in bronchitis and laryngitis; Tr. Iodi as a stimulant in chronic catarrh. Ext. Lupuli will allay irritability of mucous membrane. Ol. Eucalypti, Terebene, Creosote, Ol. Pini Sylvestris, may all be employed in the same way (strength ♏ 40 to Oi.), and certainly produce alterative effects in chronic catarrh if persevered with for several weeks.

If intended for Eustachian medication the following directions should be observed: About six times in the five minutes well fill the mouth with steam, close the nostrils with the thumb and forefinger, shut the mouth and expire forcibly, so as to drive the vapour towards the ears.

(111) Vapor Carbonis (Anti-Catarrhal Smelling-Salts).

℞ Acidi Carbolici . . gr. xxx.
Ammonii Carbonatis . ℥j.
Carbonis Pulveris . . ℥j.
Olei Lavandulæ . . ♏xx.
Tincturæ Benzoini Compositæ . . . . ℥ss.

A dry inhalation for catarrh. Boracic acid, finely powdered, may be used as a snuff.

(112) Vapor Siccus.

℞ Olei Pini Sylvestris . . ℥j.
Olei Eucalypti . . ℥j.
Tincturæ Benzoini Compositæ . . . ℥ij.
Creosotum . . . ad ℥j.

Ten or fifteen drops to be placed on the sponge of an oro-nasal inhaler previous to use.

(113) Nauheim Baths.

This treatment is commenced with weak saline baths at a temperature of 92° to 95° F., consisting of 1 pound of common salt, and 1½ ounces of calcium chloride to every 10 gallons of water. These should be given every other day for a week, the patient remaining in the bath six minutes. The strength is then gradually increased to 3 pounds of salt and 4½ ounces of calcium chloride for every 10 gallons of water, and the patient remains in the bath for twenty minutes, with the tempera-

ture lowered to 85° F. if he can bear it. In a fortnight or more effervescing baths are employed. In every 10 gallons of water dissolve 2 ounces of sod. bicarb., and add 3 ounces of hydrochloric acid just before the patient enters. Gradually increase the strength to 8 ounces of sod. bicarb. and 12 ounces of ac. hydrochlor.

It is simpler to employ "Sandow's Tablets" and powders, which contain the ingredients for the baths specially prepared in a convenient form for ready use.

Treatment extends over five weeks. The effervescing baths are ordered according to the discretion of the physician, and in severe cases it is sometimes unsafe to employ them at all.

(114) Resistance Exercises.

(Referred to in the Treatment of Heart Disease, p. 89.)

These exercises, as practised at Nauheim, Hesse-Nassau, Germany, comprise a series of movements, each, as it is performed, being gently resisted by the nurse or operator. They are never to be repeated twice in succession. The patient, while performing them, should be carefully observed, and if any symptom of interference with the circulation or respiration (*e.g.*, breathlessness, change of tint about the lips or cheeks, dilatation of nostrils, contraction of corners of mouth, moisture on the forehead, yawning) appears, the movement must be at once stopped and a rest taken. During the movements the patient should breathe regularly, and it may be advisable that he should count in whispers, so as to achieve this end. The attire should be perfectly free.

The resistance is accomplished by the operator placing the palmar surface of his hand on the aspect of the patient's limb towards which the movement is directed, or, in the case of body movements, on that aspect towards which the movement is being carried out. While undergoing the course of exercises, the diet must be liberal, but little importance is attached to the nature of the food, so long as it is plain and nutritious. The movements must always be followed by a period of rest.

The movements must always be begun very cautiously—*i.e.*, the simpler ones first—and with every care to avoid even the appearance of fatigue. Each exercise can be varied considerably by the operator (1) by modifying the *degree of resistance*, and (2) the *speed*—the slower the movement, the greater the strain.[1]

(*a*) Resistance exercises, as carried out by Dr. Schott, consist of a series of 19 movements, given in an anatomical series, from which the physician, after studying them carefully, can select and arrange a certain number, according to the nature and progress of the case from day to day. All excepting 6 to 8 and 12 and 14 can be done with the patient in the recumbent position, though not to the fullest extent.

(1) The arms are stretched out in front of the body at the level of the shoulders with the palms meeting each other. The arms are then carried outwards laterally in line with each other, and thereafter are brought back to their first position.

(2) The arm and hand are placed in the fully supinated position, hanging down, and the forearm is flexed upon the arm, without any movement of the latter, until the fingers touch the shoulder; thereafter the arm is extended to its original position. This movement is carried out first with one arm and then with the other.

(3) The arms, hanging down, are supinated and raised outwards until the thumbs meet over the head, after which they are brought back to their original position.

(4) The fingers of the hands, flexed at the first phalangeal joints, are pressed together in front of the lowest part of the body, and the arms are raised until the hands are above the head, after which they are brought back to their original position.

---

[1] A number of nurses of both sexes have now been trained by Dr. J. Fletcher Little to carry out this treatment in England, and also to administer the baths, the ingredients of which can be obtained from Buchner, 149, Houndsditch, E.C.

(5) The arms, hanging in the position of "attention," are raised forwards parallel to each other until they are elevated to a vertical position, and are then brought back to the position from which they started.

(6) The body is bent forwards and then brought back to the erect position, the knees not being moved. (Inquiry should be made of the patient for any sensation in the head, and, if such is present, the exercise must be stopped.)

(7) The body is rotated without any movement of its feet, first to one side, then to the other, and finally back to its original position.

(8) The body is bent laterally as far as possible, first to one side, then to the other, and afterwards restored to its original erect posture.

(9) This is a movement precisely similar to No. (1), except that it is carried out with the fists clenched.

(10) The arms are moved in the same way as in exercise No. (2), but the fists are firmly clenched.

(11) The arms, starting from the position of "attention," describe a circle by moving forwards and upwards until they are raised vertically. Each palm is then turned outwards, and the arms descend backwards to their original position.

(12) The arms, starting from the position of "attention," are moved upwards and stretched backwards as far as can be done without bending the trunk, and are then brought back to their original position. (For this movement the patient should be facing a looking-glass for the attendant to watch his face.)

(13) The patient, standing with the feet side by side, and supporting himself by leaning with one hand upon any object, flexes the opposite thigh as far as it is possible, and afterwards extends it until the feet are again side by side. Thereafter, leaning upon the other hand, he carries out a similar movement with the other thigh.

(14) The patient, leaning as in the last exercise, first bends the whole lower extremity of one side, kept extended, as far forwards as possible, then backwards as far as he can, and afterwards brings it beside the other. A similar movement thereafter is carried out with the other leg.

(15) Supported by leaning both hands in front on the back of a chair, the patient flexes first one leg and then the other upon the thigh as far as he can.

(16) Resting on one hand, the patient raises the extended opposite lower extremity outwards as far as possible, and then brings it beside its fellow. A similar movement is then carried out with the other limb.

(17) The arms, held horizontally outwards, are rotated forwards and backwards at the shoulder-joint, the operator clasping the patient's wrists and resisting.

(18) The hands, held in the extended position, are first bent backwards and then forwards as far as possible, after which they are brought back to their original position.

(19) The feet, held in their ordinary position, are first bent downwards and then upwards as far as possible, after which they are brought back to their original position.

(*b*) Resistance exercises, as given in Nauheim by Dr. Groëdel, and arranged by him in progressive order, beginning with the simplest and least tiring movements.

GROUP I.—1. Flexion and extension of the fingers.
2. Flexion and extension of the hand.
3. Flexion and extension of the feet.
4. Flexion and extension of the elbow.

GROUP II.—1. Pronation and supination of the arm, with the arm fully extended.
2. Abduction and adduction of the arms (to shoulder level only).
3. Flexion and extension of the knees.
4. Arms raised in front of the body and returned (to shoulder level only).

GROUP III.—1. Arms, held horizontally, are brought forwards and backwards.
2. The lower extremities, fully extended, are turned outwards and inwards (sitting).

GROUP III.—*continued.*

3. Complete abduction and adduction of the arms (the hands meeting above the head).
4. Abduction and adduction of the legs (sitting).

GROUP IV.—1. Arms extended, raised in front of body and up above head, then returned.

2. Flexion and extension of hip, with flexed knee (sitting or lying).
3. Arm hanging at side of body is moved backwards and forwards like the pendulum of a clock.
4. Flexion and extension of the trunk.

GROUP V.—1. Arms extended and fists clenched; arms raised in front of body to shoulder level, and brought back.

2. Flexion and extension of thigh, patient standing, and supporting himself on a chair with opposite hand.
3. Flexion and extension of the head.
4. Rotation of the trunk.

GROUP VI.—1. Flexion and extension of the hip, with extending knee (sitting or lying).

2. Rotation of the head.
3. Trunk bent sideways and back.
4. Abduction and adduction of the extended lower extremities, patient standing and supported by a chair.

GROUP VII.—1. Sawing—the arm, being held as in the act of sawing, is moved forwards and backwards and returned to original position.

2. The lower extremities moved forwards and backwards like the pendulum of a clock (patient standing).
3. Raising of the trunk (lying down).
4. Flexion and extension of the hip (lying down).

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The principal reference to each subject is in black type.

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