Clinical methods : a guide to the practical study of medicine / by Robert Hutchison and Harry Rainy.

Contributors

Rainy Harry.
Hutchison Robert, Sir, 1871-1960.
Royal College of Physicians of Edinburgh

Publication/Creation

London : Cassell, 1912.

Persistent URL

https://wellcomecollection.org/works/amanp43f

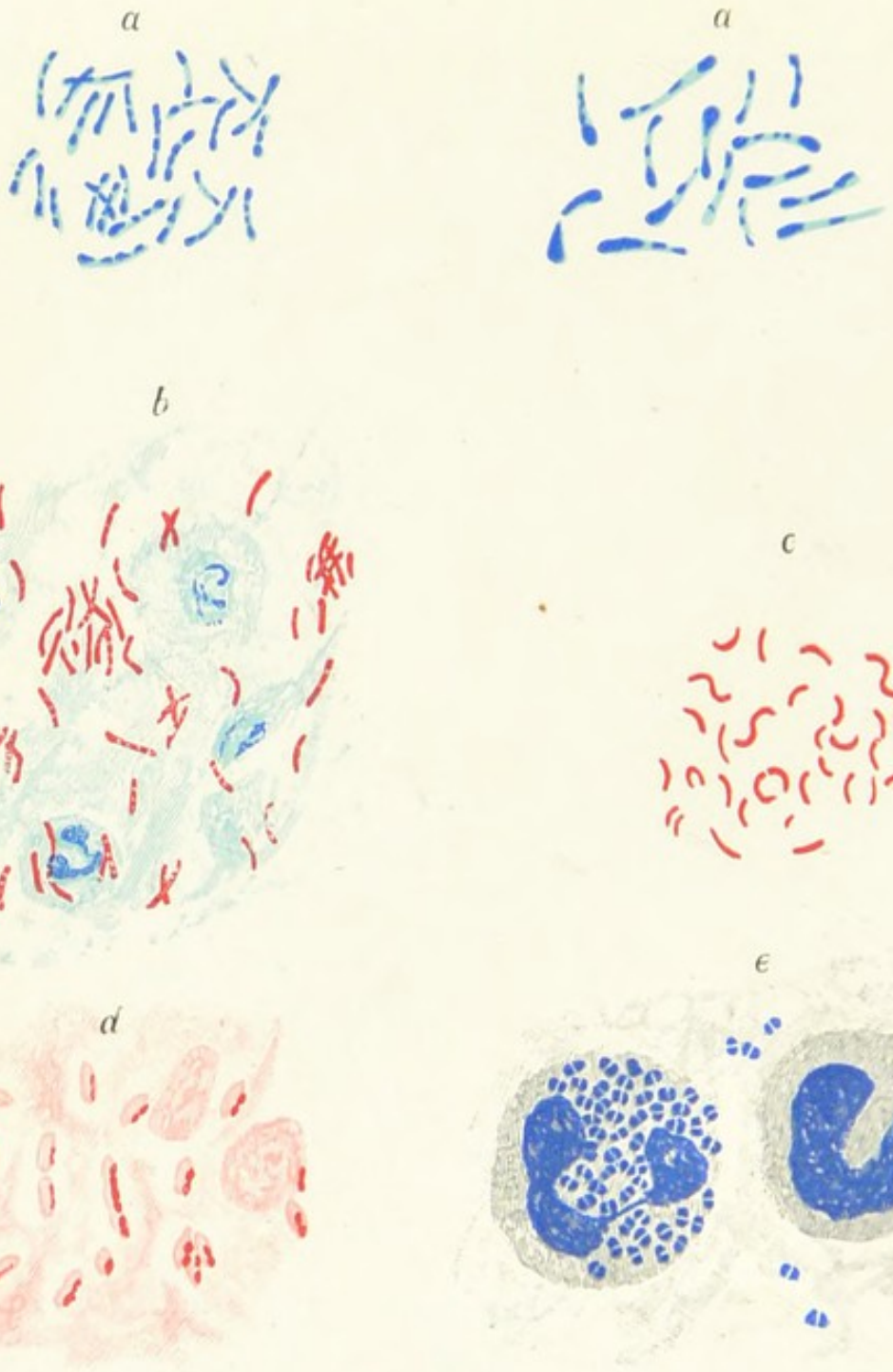
Provider

Royal College of Physicians Edinburgh

License and attribution

Clinical Methods

Hutchison and Rainy



BACTERIA.

(*After an original drawing by Richard Muir.*)

a, Bacillus diphtheriæ. Long forms. Twenty-four hours' growth; agar culture; stained watery methylene blue. × 1 000.

a', The same after five days' growth. Involuted forms

b, Bacillus tuberculosis in sputum from case of phthisis. Stained Ziehl-Neelsen. × 1,000.

c, Spirillum choleræ. Twenty-four hours' growth; agar culture; stained fuchsin. × 1,000.

d, Pneumococcus (Fränkel's) in sputum from case of acute pneumonia. Stained Ziehl-Neelsen, fuchsin; decolorized in weak acetic acid. × 1,000.

e, Gonococci in gonorrhœal pus. Stained thionin blue. × 1,000.

[*Frontispiece.*

Clinical Methods

A GUIDE TO THE PRACTICAL STUDY OF MEDICINE

BY

ROBERT HUTCHISON

M.D., F.R.C.P.

Physician to the London Hospital, and Assistant Physician to the Hospital for Sick Children, Great Ormond Street

AND

HARRY RAINY

M.D., F.R.C.P.Ed., F.R.S.E.

Assistant Physician to the Royal Infirmary, Edinburgh

WITH 13 COLOUR PLATES AND
146 FIGURES IN THE TEXT

Fifth Edition, Revised throughout

CASSELL AND COMPANY, LTD
London, New York, Toronto and Melbourne
1912

First Edition *September* 1897.
Reprinted 1898, 1899, 1900.
New Edition *March* 1902. *Reprinted October* 1902, 1903, 1904.
New and Revised Edition *September* 1905.
Reprinted February and November 1906.
New and Revised Edition *May* 1908.
Reprinted October 1908 *and November* 1910.
New and Revised Edition *May* 1912.

PREFACE TO FIFTH EDITION

In preparing the fifth edition of this work, the authors, whilst retaining the original plan and limits of the book, have thoroughly revised all the chapters, and in some cases have rewritten large sections of them, in order to keep abreast of developments that have occurred in nearly every branch of the subject since the last edition was published four years ago.

In doing so, they have received invaluable assistance from a number of their colleagues who possess special knowledge of the various departments, and they especially desire to acknowledge their indebtedness to Dr. P. N. Panton, with whose aid the chapter on the Blood has been practically remodelled throughout; to Drs. Lovell Gulland, Martin Flack, and Gordon Holmes, for much advice and help in the chapters devoted to the Respiratory, Urinary, and Nervous Systems respectively; and to Dr. James Ritchie, who has made himself responsible for the chapter on Bacteriology. In the chapters dealing with the Alimentary and Circulatory Systems the authors have also introduced numerous alterations, and the section on the Examination of the Gastric Contents is essentially new in its present form.

Wherever it was found desirable, old diagrams have been replaced by new ones, and the chapter on the Blood is illustrated by new Plates. The Plates illustrating regional topography, in the preparation of which the authors received the valued advice of the late Prof. Cunningham, have been retained unaltered from the last edition. The volume has been entirely re-set, a greater variety of type has been used, and the Index has been much amplified.

R. H.
H. R.

April, 1912.

PREFACE TO ORIGINAL EDITION

The title "Clinical Methods" probably describes the scope of this book better than any other. It is not intended as a treatise upon medical diagnosis. On that subject there is already a sufficiency of good works in existence. It aims rather at describing those methods of clinical investigation by the proper application of which a correct diagnosis can alone be arrived at. To every student when he first begins work in a medical ward the question presents itself: How shall I investigate this case? To that question the present work is intended to provide an answer. The first chapter deals, therefore, with the methods of case-taking in general, and includes a general scheme for the investigation of medical cases. The rest of the book is really an expansion of that scheme, each system being taken up separately, and the methods of investigating it described in detail.

A special chapter has been devoted to the clinical methods of examining children, as these differ in many respects from those employed in the case of adults. Chapters have also been added on the examination of Pathological Fluids and on Clinical Bacteriology, subjects which are daily growing in importance. The methods employed in the investigation of surgical, gynæcological, or obstetric cases do not fall within the scope of the work.

No effort has been spared to make the book thoroughly up to date, and it is hoped, therefore, that it will be found useful by those practitioners who may

wish to make themselves acquainted with the latest methods of clinical investigation. While the whole book has passed through the hands of both of us, yet each has made himself specially responsible for certain parts. Thus Dr. Rainy has written Chapters II., IV., VI., and XIV., the sections on the electrical examination of muscles and nerves, on the parasites of the alimentary tract, and on the microscopical examination of the urine. The rest of the work is from the pen of Dr. Hutchison.

In order to avoid burdening the text, but few references have been given to authorities and original sources. We should like, however, to take this opportunity of acknowledging the help which we have received from various friends. Amongst these are Drs. Alex. Bruce, R. W. Philip, G. Lovell Gulland, and John Thomson, who have helped us with criticism and advice in the preparation of Chapters IV. and IX., VI., V., and XII. respectively. We have also to thank Dr. Patrick Manson, Dr. Byrom Bramwell, Dr. J. Purves Stewart, and Prof. Symington for the use of specimens and illustrations, and Dr. T. F. Milroy for assistance in the revision of proofs. To Dr. R. J. M. Buchanan we are specially indebted for preparing the drawings illustrating the microscopical examination of the blood.

R. H.
H. R.

September, 1897.

CONTENTS

CHAPTER X

PAGE

EXAMINATION OF THE EYE, EAR, THROAT, AND NOSE

CHAPTER XI

THE LOCOMOTORY SYSTEM (BONES, JOINTS, GAIT)

CHAPTER XII

CHAPTER XIII

CHAPTER XIV

LIST OF COLOUR PLATES

CLINICAL METHODS

CHAPTER I

CASE-TAKING

THERE can be no question of the value of accurate and systematic case-taking. It trains the beginner in habits of thoroughness and exactness at the bedside, and ensures that no point of importance in the case is missed. To the more experienced clinician the systematic record of cases is of no less value. It gives to his experience a concrete embodiment, so that he can draw upon it at any future time by the comparison of new cases with old, and so enables him gradually to build up his clinical knowledge upon a sure foundation. When we come to the *method* to be pursued in taking a case, however, there is much divergence of opinion, and almost every clinical teacher has his own special plan. Nor is it of so much importance what particular method one adopts, provided one adheres to it. Every good method of case-taking should be both comprehensive and concise. It should be comprehensive, so as to be capable of being applied to every case and of covering all the points in it; it should be concise, so as to present all the important features of a case in as small a compass as possible. The question of conciseness is of very great importance. Nothing is more annoying than to be obliged to wade through a mass of verbiage in order to get at the chief facts of a particular case. The student should practise the art of focusing a case in such a way as to present its leading features in a few

sentences. For this purpose the writing of résumés of cases will be found a useful exercise. He should also avoid lengthy verbal descriptions as far as possible, especially where the facts admit of graphic representation. The use of outline diagrams will be found very helpful in this respect. Physical signs can be filled in on them by means of conventional symbols.

We have appended to this chapter a scheme of case-taking which meets all necessary requirements. At the same time, it must be used, like all such schemes, with some judgment and elasticity. All the points mentioned need not be minutely inquired into in each individual case. For example, if a patient be suffering from advanced cardiac disease, there is no use in writing a minute description of the state of his teeth. Yet that is the kind of error into which beginners not infrequently fall. Of course, it demands some experience to enable one to say what the points are which it is of importance to inquire into in any particular case, and at first one may sometimes be at fault; but the application of a little common-sense will ensure the avoidance of any gross blunders.

The "taking" of any case consists of two parts—

I. The interrogation of the patient.

II. The physical examination.

I. The Interrogation of the Patient

The object of the interrogation of the patient is to elicit information regarding his present illness and the state of his previous health and that of his family. The interrogation must be pursued with patience, the patient being allowed, as far as possible, to tell his story in his own words. Two good rules should be remembered—first, to avoid leading questions; and, second, never to ask the same question twice. The use of leading questions is only occasionally allowable,

as, for instance, when one suspects that he has to do with a case of malingering, when one may get the patient to assert the existence of contradictory symptoms, so confirming one's suspicions. It may also be necessary in dealing with patients who are stupid either by nature or as the result of disease. When one is trying to elicit what are known as "subjective symptoms," the use of leading questions may also be admissible. It is important to avoid asking the same question twice because to do so looks careless, and conveys to the patient the impression of taking but a languid interest in his case.*

We may proceed now to go more into detail regarding the questions which should be asked. In doing so, we shall consider first the questions which one has to put in every case—what one may call the *general interrogation;* and then we shall take up the questions which have to be put in examining cases of disease affecting the different systems or organs—this may be called the *special interrogation.*

1. **General interrogation.**—Begin by ascertaining the patient's name, age, occupation, and whether he is married or single. It is also of importance to note his exact postal address for purposes of future communication.

Two important questions then follow—(1) Of what does he complain ? † (2) How long have the symptoms been present ? Having thus defined his complaint and its duration, proceed to ascertain the chief facts in his history.

The most logical plan is to take the **family**

* When one has to deal with a patient who is very deaf it will often be found convenient to use a binaural stethoscope as a speaking-trumpet, the ear-piece being placed in the patient's ears, while one speaks into the chest-piece.

† It is a mistake to ask "What is the matter ?" as this lays one open to the retort that that is what the patient came to find out.

history first.* It is usually sufficient to inquire regarding the state of health or cause of death of the immediate relatives only—the parents, brothers and sisters, and, if the patient be married, of his own children, if any. These facts tell us whether he is predisposed by heredity to any particular disease.

One may then pass to his **personal history.** Here it is well to begin with what may be grouped together as the patient's environment, or *surroundings* and *habits*. This includes inquiry into (*a*) the exact nature of his occupation, and whether or not it exposes him to injurious influences ; (*b*) his home surroundings, their sanitary condition or otherwise ; (*c*) the amount of exercise he takes; (*d*) the nature of his food, and the extent of his indulgence in such articles as alcohol, tea, and tobacco.† Regarding alcohol, it is important to ascertain not merely how much—e.g. how many glasses of spirits per day—the patient takes, but how and when he consumes it—e.g. with or between meals. The kind of tobacco he smokes and the number of ounces consumed per week may need to be ascertained in some cases. (*e*) One should always ascertain, lastly, whether or not he has ever lived abroad, and, if so, in what part of the world.

The information thus acquired enables one to add to the tendencies to disease which the patient has inherited, those which he has acquired from his environment and personal habits.

One should take up next the question of the patient's **previous health.** Ascertain what illnesses he has had, when he had them, their duration, and

* This statement is perfectly true in theory, but in practice it is perhaps more convenient to begin with the history of the present illness, to pass from that to an inquiry into the patient's previous health, and thence to the family history.

† When inquiring into a patient's habits of life it will often be found advantageous, particularly in private practice, to ask him to give a brief account of a typical day.

whether or not his recovery from them was complete. It is usually necessary to inquire directly as to whether or not the patient has ever had syphilis. It is not sufficient to ascertain that he has had a sore; the question of secondary symptoms—e.g. rash—must also be gone into. If the patient denies syphilis, it may be necessary to ask whether or not he has ever been exposed to the risk of it, and whether or not he has had any other venereal disease. In the case of female patients, information regarding venereal disease should, as far as possible, be obtained indirectly, direct interrogation on the subject being employed only when a definite history is absolutely necessary for the elucidation of the case. The student need hardly be reminded that even in these circumstances his questions should be put as delicately as possible.

Having now ascertained the patient's inherited and acquired tendencies, and the seeds of disease which may have been sown in him by his previous illnesses, we are ready to acquire information regarding his present disorder.

Ask how and when it began, whether suddenly or gradually; what was the first thing he noticed wrong; what has been the order of appearance of his symptoms, and which are those that chiefly trouble him at the present time. Ascertain whether or not he has already been under treatment, and, if so, what has been done for him.

This exhausts the general interrogation, and includes the chief facts that have to be inquired into in every case.

2. The **special interrogation,** to which we have already referred, must be modified according to the particular organ which one believes to be affected and the nature of the disease of which it is suspected to be the seat. It is here that the student has most difficulty.

It is only by experience that one can tell what it is essential to ask in each individual case. In order to help the beginner, however, we have drawn up for his guidance a scheme of interrogation which he can pursue when he has reason to suppose that the patient's general symptoms point to an affection of any particular system or organ. Such a scheme is necessarily very far from complete, and may require to be supplemented in individual cases. Nor is one able in such a work as this to explain *why* such and such questions should be put in affections of this or that organ or system. The reasons for the questions the student will find out for himself in due time. Our present object is merely to help him in the interrogation of his earlier cases, so that he may not miss any important facts. The questions are to a considerable extent concerned with eliciting what are sometimes spoken of as "*subjective symptoms*"—i.e. the morbid sensations experienced by a patient as the result of the disease of some organ or system.

In making the notes, these, along with the other replies, should be entered under the special system to which they refer.

1. **Alimentary system and abdomen.***

* While the methods of gynæcological examination are beyond the scope of this book, yet it must not be forgotten that it is frequently necessary in purely medical cases to inquire regarding the menstrual function. The cases in which such inquiry may be required are too diverse to be enumerated here. It need hardly be said that the necessary questions should not be abruptly put to the patient, but should be delicately led up to. Having inquired regarding the regularity of the bowels, one may ask if the patient is "regular in her own health" or "regular in her unwell times." It will be remembered that in a majority of cases menstruation recurs every twenty-eight days, but the intervals may be longer or shorter according to the patient's habit. If menstruation has ceased, one must inquire how long it has been absent. Normally the cessation of menstruation, or menopause, should not occur till about the forty-fifth year. It is also necessary to inquire whether the patient is losing more or less blood than usual. This is spe-

(*a*) Symptoms point to an affection of the *stomach*. Inquire regarding—

Appetite.—Is it excessive, diminished, or capricious ? Does it increase on eating ? Does he suffer from thirst ?

Meals.—Arrangement of these ; the nature of the food. Does he eat between meals ?

Sensations referred to stomach.—Their nature, and where exactly they are felt. Their relation to the taking of food ; are they produced or relieved by it ? How long after food do they come on ? Are they specially influenced by different kinds of food ? Distinguish especially between "pain" and mere "sense of discomfort" or "fullness."

Vomiting.—Frequency and time of ; by day or by night ; in the morning or in the evening. Its relation to food ; is it only after food, or does it occur at other times ? Its relation to pain ; does it relieve pain or not ? Does patient strain and retch much, or does the vomited matter come up quite easily ?

General characters of vomited matter.—Its amount and colour. Is there ever "coffee-grounds" vomiting ; is it ever sour and frothy ?

Eructations.—Presence or absence ; have they any taste ?

Flatulence.—Presence or absence ; after food only or between meals ? Relation to particular articles of food. Does it tend to escape downwards or upwards ?

State of the bowels.—How often are they opened ? Any special characters of the motions.

(*b*) Symptoms point to an affection of the *intestines*. Inquire regarding—

Diarrhœa.—Its frequency and its relation to meals or to special articles of food. Character of the motions. Has he ever passed any blood or slime ? Is there any straining or tenesmus during defæcation ? Is there any flatulence ?

Constipation.—What is his usual habit ? are the bowels opened regularly, and if so, how often ? How long since the last motion ? Has he ever noticed any grooving or flattening of the motions ? Does the constipation alternate with diarrhœa ? Has he any griping pain ? Has he had any vomiting ?

cially indicated in cases of anæmia. If the menstrual flow lasts for less than two or more than eight days, it is to be regarded as abnormal. The presence or absence of pain at the period is also a point of some importance. The age at which menstruation began, and the occurrence or not of intermenstrual leucorrhœa ("white discharge"), must sometimes be inquired into.

Pain.—Character ; persistent or intermittent. Where is it felt worst ? Is it relieved or aggravated by pressure ?

(*c*) Symptoms point to an affection of the *liver*—e.g. patient is jaundiced, or has pain in region of liver. Inquire regarding—

Pain.—Its site. Has he ever any attacks of very severe pain, coming on suddenly and lasting for a few hours ? If so, did the pain radiate, and in what direction ? Was there vomiting with it ? Was he yellow at all after it subsided ? Has he ever pain in the tip of the shoulder ?

Does he suffer from piles ?

Does he ever vomit blood ?

Has he noticed any change in the colour of the urine or fæces ?

Does his skin itch at all (if he is jaundiced) ? Inquire also regarding his digestion on the lines of the interrogation already laid down for affections of the stomach.

2. The symptoms point to an affection of the **circulatory system.** Inquire regarding—

A *family history* of gout, rheumatism, angina, apoplexy, or heart disease.

A personal history of rheumatic fever, St. Vitus's dance, scarlatina, or diphtheria. (If a child, ask also about sore throats and "growing pains.")

The following subjective sensations :—

Dyspnœa.—Has he to sit up in bed, or can he sleep lying down ? When does it come on ? *Præcordial pain* or distress ; its exact site and character ; does it radiate or not ? If so, in what direction ? *Palpitation :* its relation to meals, and to exertion. Does the heart give an occasional thump now and then ? *Sleep*, good or bad ; does he dream ? *Giddiness*, is it ever present, and when ? Is there any sense of undue exhaustion after bodily or mental work ?

Ask also for signs indicative of general venous distension –e.g. do the feet ever swell ? Has he any cough ? What is the state of the digestion ? Does his nose ever bleed ?

3. The symptoms and appearances point to an affection of the **blood.** Inquire regarding—

Family history of bleeders. Has he had any loss of blood ? Has he bleeding piles ? (If a woman—is menstruation excessive or diminished ?) What is the state of the bowels ?

Any possibility of lead-poisoning or malaria ?

Such subjective sensations as breathlessness on exertion ; headache ; giddiness.

Do the feet ever swell ?

4. The symptoms point to an affection of the **respiratory organs.** Inquire regarding—

Family history of bronchitis, asthma, phthisis, or "scrofula." The patient's occupation ; does it expose him to the inhalation of irritating fumes or particles ? Has he ever had large glands in the neck ? Does he sweat at night ? Is he getting thinner ?

Cough.—Its character and frequency ; when is it worst ? Does it pain him or not ? Does he ever vomit with it ?

Expectoration.—Its amount and general characters ; yellow or not ? Ever blood in it ? If so, is it only after severe coughing ? Is the blood bright and frothy or dark in colour ?

Pain in chest.—Is it aggravated by taking a breath ? Constant or not ? Where seated ?

Dyspnœa.—When is it felt ? If spasmodic, ask him to describe an attack.

5. The symptoms point to an affection of the **kidneys**—e.g. general dropsy—or **urinary passages**—e.g. pain in micturition. Inquire regarding—

Family history of Bright's disease, gout, or apoplexy.

Personal history of scarlatina, syphilis, lead-poisoning, prolonged suppurations, gravel or gout, and previous renal disease.

Has he any pain in the lumbar region ? Ever any attacks of acute pain shooting down into the groin ?

The following remote symptoms :—

Headache, vomiting, drowsiness, paralysis or fits, dimness of sight, dyspnœa.

Does the face ever look puffy in the morning ?

What is the state of the bowels ?

Inquire regarding micturition as follows :—

Urine.—Is it altered in amount ? Has he to rise in the night to pass it ?

Is it altered in colour ? Is it clear or turbid when passed ? Ever any blood in it ? If so, at what period of micturition is it present ?

Is there any increased frequency of micturition ? Is the increase by day or by night ?

Is there any pain during micturition ? Is it before, during, or after the act ? What is its character, and where is it felt ? Is it aggravated by movement ?

6. In **skin diseases.**

Inquire carefully into the patient's personal habits as regards diet, clothing, and washing. Ask if he has been taking any drugs recently. It may be necessary to inquire carefully regarding syphilis. Does the eruption itch ? If so, when is the itching worst ? Did the eruption appear all at once or in crops ? (Family history of gout ; previous history of rheumatism, anæmia, etc.)

7. The symptoms point to an affection of the **nervous system.** Inquire regarding—

A *family* history of mental disease, St. Vitus's dance, paralysis or fits.

The nature of the patient's work ; is he exposed to any poisons—e.g. lead, mercury, arsenic, naphtha, etc. Syphilis and alcohol should be inquired about with special care.

In cerebral cases it is often very important to inquire regarding discharge from the ear.

Should he complain of *fits*, the following questions should be asked :—

Age at first fit ? Any assigned cause ? Describe the first fit. When did the second occur ? What has been shortest and longest interval between the fits ? Are they more or less frequent now ? Do they occur in sleep or not ? Has he any premonition or aura ? What is its character ? How long before the loss of consciousness does it occur ? Is the onset sudden or gradual ? Are convulsions present ? Are they general or local ? Where do they begin and end ? Does he fall ? Has he ever hurt himself ? Does he bite his tongue, micturate, or defæcate during the fit ? Are there any after-symptoms, such as sleep, headache, automatism, or paralysis ? Is there any subsequent mental disturbance ?

If he complains of *paralysis*, inquire regarding—

Symptoms of heart disease, or chronic renal disease (*see* Circulatory and Urinary Systems). Had he any premonitory symptoms before the onset ? Has he any headache or vomiting ? Where is the headache situated ? Has he any giddiness or difficulty in walking ? (The method of eliciting other subjective symptoms of nervous disease is considered along with the investigation of the cranial nerves in Chap. IX., p. 409.)

8. The symptoms point to an affection of the **bones** or **joints.**

Inquire specially, in the family history, for tubercular disease, rheumatism, gout, or syphilis, and in the personal history for tubercular disease, previous manifestations of gout or rheumatism, for syphilis or gonorrhœa, and for any remote or recent injury (and in a woman for leucorrhœa or post-partum trouble).

If there be pain referred to a bone, ask whether it is worse during the day or during the night. If the pain be in a joint, ask whether it is present constantly or only when the joint is moved. Are there any starting pains at night ? Is the pain affected by weather ? Does the pain shift from one joint to another ?

If the patient be a young **child,** the following special questions should be put to the mother* :—

How many other children are there ? Any dead, and of what ? Where does patient come in the family ? Have there been any miscarriages ? If so, when ? Health of father's and mother's family ? Mother's health during pregnancy ?

Was this a full-time child ? Was the labour normal ? Was the child breast-fed : if so, how long ? If not, how was it fed ? What food does it get now ? Had it any rash after birth, or any snuffles ? When did it begin to get its teeth, and to walk ?

What is the usual state of the digestion and bowels ?

Inquire regarding previous illnesses : Fits (number and dates), attacks of diarrhœa, vomiting, sore-throat or bronchitis. Infectious diseases and ages at which they occurred (measles, whooping-cough, chicken-pox, scarlatina, etc.). Has there been any running from the ears ? If the child has a cough, inquire whether it has ever whooped, when the attacks are worst, and whether the cough is ever followed by vomiting.

The interrogation of the patient being completed, one proceeds to—

II. The Physical Examination

One investigates first of all, in every case, the patient's general state. This includes the general condition of his nutrition, the presence of any obviously

* In the absence of the mother, note from whom the history was obtained.

morbid appearances, and the other points considered in detail in Chap. II. One proceeds after that to the investigation of each system by itself. What system should be taken up first? As regards this, there are two possible methods. One may either take up the systems in one and the same order in every case, beginning, say, with the Alimentary, or one may examine first the system which is most affected. The latter is, on the whole, the better plan, provided always that one is able to tell which system it really is that is most diseased. The advantage of this method is that it gives most prominence to the most important part of the physical examination. Whichever plan the student elects to adopt, he may now proceed to the physical examination of the different systems in accordance with the instructions laid down in the following chapters, the results being noted in the order given in the scheme below.

Only one more point regarding case-taking remains to be emphasized, and that is the importance of noting negative as well as positive facts. It is often quite as essential, for example, to state that such a symptom as dyspnœa is absent as to record the fact of its presence. This is a point the importance of which is apt not to be fully appreciated by the beginner.

In conclusion, it need hardly be said that the examination should be carried out as gently as possible, all unnecessary exposure, exhaustion, or chilling of the patient being carefully avoided. If the patient be suffering from severe or acute disease, it may be advisable to postpone all physical examination other than that which is absolutely necessary for the diagnosis of his condition, or for guidance in treatment. It should also be borne in mind that when a patient is much exhausted, or suffering from serious disease of the lungs or heart, very dangerous and even fatal results may

ensue if he be thoughtlessly made to sit up in bed in order to have his chest examined.

CASE-TAKING SCHEME

I. Interrogation

Name. Age. Occupation. Married or single. Address. Date of coming under observation.

Complaint.

Duration.

Family history.*

Inquire regarding parents, brothers and sisters, and patient's own children. Note state of their health; or the cause of their death, with age at which they died.

Personal history.

Environment.—Nature of work and its surroundings. Hygienic conditions at home; habits as to exercise, food, tea, alcohol, and tobacco.

Previous illnesses or accidents (if any), with their time of occurrence, duration, and results.

Present illness.—Time and mode of its origin, the order in which symptoms appeared, and the chief symptoms which trouble patient now; treatment (if any) already employed.

II. Physical Examination

1. **Present state.**

General condition.—General state of consciousness and intelligence. Decubitus (if in bed), or attitude and gait (if up) (pp. 19 and 22). General state of development and nutrition. Expression of face; presence or absence of pallor, jaundice, cyanosis, dropsy, or trophic changes. Presence or absence of any special characters of the hands (p. 32). Glandular enlargements. Character of the respiration, and the presence or absence of cough. Take the temperature.

2. **Alimentary system.**

Subjective symptoms (*see* Special Interrogation, p. 7). Examine the *mouth* (including the teeth, gums, and tongue),

* *See* first foot-note on p. 4.

the *pharynx*, and *fauces* (pp. 46–50), and the *œsophagus* (with use of sound if necessary) (p. 51).

General inspection, palpation and percussion of the *abdomen* (pp. 54–64).

Stomach.—Palpation and percussion (pp. 66–69). Examination of test breakfast or vomit (pp. 82-92).

Intestines.—Investigation of (p. 80). Rectal examination if necessary (p. 80). Examination of fæces (p. 92).

Liver and *gall-bladder.*—Examination of, by palpation and percussion (pp. 70–74).

Spleen.—Examination of (p. 74).

3. **Circulatory system.**

Heart.—Subjective symptoms (Special Interrogation, p. 8).

Pulse.—Describe its rate and its rhythm. Compare the force of successive beats. Ascertain the state of the vessel walls. Note the blood-pressure during and between the beats. Observe the amplitude of the pulse waves. Analyse a complete beat of the pulse regarding rise, maintenance, and fall of pressure, and determine the presence or absence of secondary waves. Take tracings if the pulse is abnormal.

Inspection and palpation of *præcordia*, noting position and character of apex beat, presence or absence of epigastric pulsation or præcordial thrills, or of pulsation in the neck or at the base of heart.

Percussion of *heart* (p. 132).

(*a*) Upper border
(*b*) Right border } superficial, deep.
(*c*) Left border

Auscultation of *heart* (p. 148).

(*a*) At apex and a little internal to it.
(*b*) Tricuspid area at lower end of sternum.
(*c*) Aortic area.
(*d*) Pulmonary area and a little outside it.
(*e*) Between base and apex (3rd and 4th left costal cartilages).
(*f*) Veins and arteries of neck.

If a bruit is heard, note—

(*a*) Its time.
(*b*) Its character (musical, harsh, etc.).
(*c*) Its point of maximum intensity.
(*d*) Its direction of propagation.

4. **The blood.**

Count the red and white corpuscles (pp. 212–20). Esti-

mate the hæmoglobin (pp. 220–27). Examine the blood microscopically, making films if necessary (pp. 227–41).

5. Respiratory system.

Subjective symptoms (*see* Special Interrogation, p. 9).
Count the respirations and describe their character.
Inspection of chest, noting its shape, power of expansion, etc. (p. 248).
Mensuration of the two sides of the chest.
Palpation of chest (expansion and vocal fremitus) (p. 260).
Percussion of lungs anteriorly, laterally, and posteriorly (p. 265).
Auscultation of lungs in same order (p. 277), noting—
(*a*) Type of breath sounds.
(*b*) Character of vocal resonance.
(*c*) Presence or absence of accompaniments.
Sputum.—Note its naked-eye and microscopic characters (p. 295).

6. Urinary system.

Palpate the *kidneys* (p. 77).
Examine the *urine*—physically (p. 307), chemically (p. 321), microscopically (p. 377), making a note in *every* case of the following points :—
Quantity in twenty-four hours, colour, specific gravity, reaction, odour, general character of deposit.
Presence or absence of albumin, blood, sugar, and bile.
Microscopic characters of deposits.

7. Skin.

General colour ; presence or absence of pigmentation or eruption ; nature of " primary lesion " in eruption and of " secondary lesions," if present (p. 398).
Palpate the skin ; dryness, smoothness, thickness, elasticity. Character of subcutaneous tissues.

8. Nervous system.

Inquire regarding subjective symptoms (*see* Special Interrogation, p. 10).
Investigate state of—
(1) Intellectual functions (intelligence, memory, sleep, coma, delirium, speech, etc.) (p. 430).
(2) Cranial nerve functions (testing them in order) (p. 439).

(3) Motor functions (noting presence or absence of paralysis, or of abnormal muscular movements, and state of muscular nutrition) (pp. 478–90). Electrical reactions of muscles and nerves, if necessary (pp. 509–22).

(4) Sensory functions (including condition of sensibility to touch, weight, temperature, and pain, and the muscle sense) (pp. 491–96); presence or absence of abnormal sensations (p. 496).

(5) Reflexes :—
Superficial reflexes (p. 497).
Deep reflexes (p. 501).
Organic reflexes and sphincters (p. 507).

(6) Vasomotor and trophic changes. Tache or abnormal flushing. Localized pallor or blueness. Sweating (presence or absence in any locality). Joint changes. Changes in the nails, hair, or skin (abnormal pigmentation, eruptions, atrophies, etc.) (p. 509).

9. **The eye.**

Appearances seen on ordinary inspection of lids, conjunctiva, cornea, iris, etc. (p. 523).

Use oblique illumination and ophthalmoscopy, noting state of media, refraction, and characters of fundus (pp. 525–40).

N.B.—The fundus of the eye should be reported on in all cases of nervous disease.

The **ear.**—Examine pinna, meatus, and membrane (using speculum and inflation if necessary) (p. 540).

The **throat, nose,** and **larynx.**—Examine larynx (laryngoscopy) and anterior and posterior nares (posterior rhinoscopy) (pp. 545–53), noting any abnormalities.

10. **Locomotory system.**

Describe any changes in the bones or joints (p. 554).

Diagnosis

(Prognosis)

Notes of Treatment and Progress

(Daily notes in acute cases; in others make a note of progress every three days.)

State on dismissal.

If patient died, add notes of post-mortem (if held).

The following special scheme for cases presenting **mental symptoms** has been drawn up by Dr. Henry Head, and will often be found useful in medical wards :—

1. **General.**

The aspect of the patient as modified by the mental disturbance. General attitude and behaviour.

Any peculiarity in clothing. Does patient tend to strip himself or behave indecently ? Can he dress himself ? How does he take his food ?

General standard of intelligence. Can he read ? Can he write ? Can he amuse himself with pictures ?

Speech as modified by the mental state.

Is he destructive ?

Is he dirty in his habits ? If so, is it from inattention, or is he actively dirty ?

Masturbation, alcoholism, etc.

Is he cataleptic or rigid ? Does he make any rhythmical movements or sounds ? Restlessness or tremor of the hands ? Over-action of muscles of face ?

Does he sleep ?

Does he tend to wander about the room or house at night ?

2. **Sensory.**

Illusions of sight, hearing, smell, taste. Subjective sensations of touch based upon wrong interpretation of some actual sensation.

Hallucinations.

3. **Emotional.**

Exaltation.—Chattering, shouting, singing. Excessive sense of well-being. Restlessness or violence.

Depression.—Crying, sighing, moaning. Miserable feeling, either in attacks or continuous. Fear. Is the patient suicidal ?

Erotism.—Are the patient's statements coloured by an erotic tone ? Give examples.

Religion.—Is the patient's mental state coloured by an extravagantly religious tone ?

4. **Memory.**

Memory of intention, i.e. does the patient wish to say or do something and immediately forget the intention ? Does the patient misplace things ?

Memory of recent events.
Memory of remote events (e.g. events of childhood).
[If recent memory only is lost, try and find out when the break in memory occurs.]

5. Ideation.

Orientation.—Sense of time and space. Delusions of identity (i.e. does patient mistake those around him for his friends and associates before he entered hospital, or does he imagine they are famous or legendary persons?). Does he appreciate his surroundings, or does he imagine himself elsewhere than he really is? Does he invest the acts of those around him with a secondary or symbolic meaning? Does he describe actions he has performed, in themselves not impossible or improbable, which, however, did not actually occur? (e.g. when in bed with alcoholic paralysis does he describe the walk he took in the morning, the people he met, etc.?).

Coherence or incoherence of ideas.

Delusions of suspicion.—Continuous or only in attacks.

Delusions of persecution.—Action of unseen agencies, etc. (especially at night).

Delusions of grandeur.—Riches, power, bodily strength.

Delusions concerning his health or bodily state.

Fears (*unfounded*) in neurasthenia.

CHAPTER II

GENERAL CONDITION AND APPEARANCES

BEFORE commencing the physical examination, the physician may gather invaluable information from a more general survey of his patient. During the time occupied in asking questions, and even before it, the skilled eye and ear may detect much that has an important bearing on the case. Experience in actual clinical practice can alone educate to this, but some lines may be indicated along which to work.

One of the first things to observe is the **attitude of the patient as he lies in bed** (decubitus). In health a person lies in any manner in which he feels comfortable—sometimes on his back, sometimes on his side. He changes his position without much difficulty from time to time, and has no hesitation in altering his attitude if he slips from his pillows or feels otherwise uncomfortable. But the stress of disease will often confine his activity in narrow bounds. When fever has run high, or when some other cause has reduced the patient to extreme weakness and dulled his consciousness, he no longer makes any effort to secure a position of comfort, but *passively slips downwards* from his pillows in obedience to the law of gravity, and lies listless, flaccid, and silent even when the resulting attitude is such as to render the act of breathing unnecessarily exhausting.

Almost equally characteristic is the *lateral position* necessitated by some diseases of the viscera, and especially by those of the lungs and pleura. The two main factors in compelling this attitude are, first, the greater ease with which respiration can be performed on one

side than on the other; and, second, the fact that in certain positions the pain is rendered less acute, whilst in others it is aggravated. When these factors co-operate, it is easy to say which side the patient will choose. Thus, in pleurisy with much effusion, where the chief difficulty is the mechanical one of providing sufficient expansion for the uninjured lung, and where pain is slight or absent altogether, the patient will be found lying on the diseased side. If, however, pain be the prominent element, as occurs in the earlier stage of pleurisy, he will best secure easy respiration by lying in the position of least suffering. What this position will be it is not easy to predict, for the pain depends both on the amount of movement and the pressure exerted by the inflamed surfaces on each other. When the inflamed pleura is uppermost its movement is greatest, but its pressure against the chest wall is least; when it is lowermost the opposite is true; and so when movement is the chief cause of pain the patient will lie on the affected side, but when pressure exerts the greater influence, on the sound one. In either case, however, he confines himself to the selected side, and any change indicates an alteration in the state of the disease.

Another class of patients who prefer one side are those who have a cavity in the lung. When this cavity lies with its aperture below, the secretion flowing from it enters healthy bronchi, and by irritating them maintains a perpetual and most distressing cough. If, however, such a patient turns over, so that the cavity fills before its contents escape, a period of tranquillity is obtained, and though the cough eventually recurs, a larger quantity of secretion is promptly got rid of, and another period of rest secured. When, as frequently happens in phthisis, the secretion is tough and scanty, this symptom is inconspicuous.

Even in health many persons feel more comfortable on one side than on the other, and when ill will often continue to prefer the accustomed attitude ; hence the fact that the patient is repeatedly found on one side, although it suggests the propriety of being on the outlook for disease, does not always indicate its presence.

In cases where great demands are made upon the respiratory system, and especially when it fails to respond fully to such demands, the sufferer can rarely lie down in bed, but sits more or less erect and propped up with pillows. To this condition the name of **orthopnœa** has been given. It is common in advanced stages of heart, lung, and kidney disease, and its rationale is found in the fact that this attitude permits of freer use of the accessory respiratory muscles, whilst it leaves the diaphragm less impeded by intra-abdominal pressure, and perhaps, also, acts favourably on the intracranial venous pressure. When the abdomen is greatly distended the sufferer cannot flex his thighs without raising the abdominal pressure ; at the same time he prefers to sit up rather than to remain in bed, in order that the weight of the fluid may not hinder the descent of the diaphragm. In sitting up, however, he tries to avoid bending his thighs, and therefore he keeps well forward in his arm-chair, sometimes almost in a kneeling attitude, whilst he rests his head on a table placed before him. In such cases it is obvious that the removal of the ascitic effusion may afford unspeakable relief to the patient.

In **abdominal disease,** especially when the peritoneum is involved, the aspect is frequently characteristic. The patient lies on his back with a rigidity of attitude and shallow respiration which betoken the pain that any movement produces, whilst one or both legs are drawn up, according as the inflammation is limited to one side or has become more general.

In **colic** and **dysmenorrhœa** there is often great restlessness, which contrasts vividly with the fixed attitude of serous inflammation. In **renal colic** the patient tosses about and tries one position after another in futile search for a posture free from pain; whilst the less acute but more constant dragging pain of **renal calculus** produces a drooping of the shoulder on the affected side, which is most marked when the patient is erect, but may be present even in bed.

Patients who are attacked by **acute rheumatism** have a peculiar aspect of helplessness, the limbs lying motionless, the joints being swollen, stiff, and painful.

Various **diseases of the nervous system** produce characteristic attitudes; peculiarly important is that of meningitis, where the neck is bent backwards so that the head seems to bore into the pillow.

When possible, the physician should not only study his patient in bed, but should also see him up and walking. Many very characteristic attitudes, which are of the greatest value in forming a diagnosis, can only be observed when the patient is in the erect posture. Thus the forward stoop, the stiff neck, the tremor, and the fingers flexed at the metacarpal joints and working against the thumb as though engaged in making cigarettes, are as characteristic of paralysis agitans as is the festinant gait.

When the patient is **standing,** observe (1) the pose of the head; (2) the set of the shoulders; (3) the inclination at which the trunk is carried on the pelvis —thrown back in hypertrophic muscular paralysis, in pregnancy, and in massive abdominal tumour, often bent forward when abdominal pain is present; (4) the position of the arms and hands; (5) the outline of the lower limbs.

When the patient walks, any peculiarity in his **gait** must be observed. The more important types

of gait are described in Chap. XI., but the student must remember not only that alterations may be due to diseases of the muscular and nervous systems, but that the pain of a gouty toe, or of a blistered heel, or surgical conditions in the ankle-, knee-, and hip-joints, likewise produce characteristic effects.

At least a passing glance should be bestowed on the **dress.** Apart from insanity, where the patient's clothing is frequently dishevelled or grotesque, one may discover indications of a local or general change in his bulk, or his boots may wear unevenly in consequence of some abnormality of gait.

The **general development and nutrition** of the patient demand careful examination. In different types of men very considerable variations must be looked for, and various races differ greatly in breadth of chest. Age also is a factor which cannot be left out of the reckoning, and a proportion between height, girth, and weight that would be natural enough at 50 may be quite abnormal at 21. Recognizing, however, that variations must be expected in individual cases, there is still a certain general ratio between height, weight, and chest circumference which has been found to represent the average of a very large number of cases, and may therefore be taken as a rough standard, any wide divergence from which would call for special explanation. The table on the following page is one of several that have been compiled from very extensive statistics.*

Various attempts have been made, with partial success, to produce a formula which would enable the weight to be estimated when the height and girth are known. One of these, cited by H. Vierordt, is $W = \frac{H\,G}{240}$ kilogrm., where W stands for weight, H for

* Hutchinson.

TABLE OF RELATION BETWEEN HEIGHT AND WEIGHT

Height	Normal weight	Limits of deviation in excess or defect of this which are compatible with good health
ft. in.	lb.	lb.
5 1	120	∓ 24
5 3	133	∓ 27
5 5	142	∓ 28
5 6	145	∓ 29
5 7	148	∓ 30
5 8	155	∓ 31
5 9	162	∓ 32
5 10	169	∓ 34
5 11	174	∓ 35
6 0	179	∓ 36

height in centimetres, and G for girth in centimetres. If one translates the metric into the more usual British system, estimating the weight in pounds and the height and girth in inches, the formula becomes

$$W = \frac{H\,G}{17} \text{ lb.}$$

It is important to compare the chest girth taken at the level of the nipples * with that of the abdomen. If in a man below middle age the latter measurement is the larger, it either indicates an undue tendency to fat formation, which may at a later period impair his vitality, or it is due to intra-abdominal disease.

When these measurements have been made, the **nutrition** of the patient is observed. Under this head one notes whether the patient is too stout, is well nourished, or is emaciated. In health there is a fair quantity of subcutaneous fat, the muscles are of moderate size and firm texture, whilst those which have been called into special exercise in the ordinary occupation of the individual under examination may be markedly prominent, and the skin is elastic and

* In male subjects.

neither very moist nor very dry. When nutrition is perverted, the muscles become flabby, and the subcutaneous fat is increased so as eventually to become burdensome to its possessor; or emaciation sets in, owing to the balance between ingestion and excretion becoming deranged, and the waste of tissue exceeding its reparation. Emaciation is thus an important indication of many diseases, especially those which are accompanied with fever.

In estimating the state of nutrition the observer will take into account the general build of the patient —some are naturally small and slight, others are large and raw-boned; and one also meets with persons who, though possessed of little subcutaneous fat, have well-nourished muscles, whilst others, whose muscles are weak and soft, have an abundant supply of fat in the subcutaneous tissues.

Besides the nutrition of the patient, an attempt should be made to ascertain his **temperament** and, if present, his **diathesis,** since this often exerts a marked influence on the course of his illness. The analysis of temperament is beyond the scope of this work: the senior student will find valuable contributions to the subject in Hutchinson's "Pedigree of Disease."

To the trained observer the **expression** of the patient yields information of the very highest importance, and amongst the factors which determine expression the **eye** holds the foremost place. Some patients cannot look their doctor in the face, and this tendency to avoid catching his eye is important, as indicating a probability that the information they are about to give lacks truthfulness, and also that they are not to be trusted to obey the instructions which they receive. Sometimes the eyes are restless, following every movement of the attendant, as often occurs

in phthisis; at other times they stare vacantly into space, regardless of all that is passing around them—a condition well seen when the consciousness is growing dull. In exophthalmos the eyes are prominent, and show a ring of sclerotic above the cornea; or the prominence may be due to a high degree of myopia. In wasting disease or in profound collapse, such as is found in cholera, the sunken eyes and half-closed eyelids cannot fail to command attention. There are racial differences in the "set" and obliquity of the eyes, and by noting this feature something may at times be learned either of the heredity of a patient or of the tendency to reversion towards a lower type. The arch of the eyebrows may give a hint, where it is either excessive or too slight, of a proclivity to tubercular disease, and an even closer connection seems to exist between the strumous tendency and long, dark eyelashes, coupled with singularly clear sclerotics.

More detailed reference is made in a subsequent chapter to important abnormalities in the different structures of the eye, where the student will learn how the conjunctiva and sclerotic tell of tubercle and Bright's disease, of anæmia and rheumatism, of jaundice and of intemperance; and how the cornea foretells an early onset of senile changes in other organs by the appearance of an arcus senilis, or reveals the ravages of syphilis and struma; how the size and mobility of the pupils indicate the existence of disease in the nervous system, or the presence of aneurysm, or it may be only of synechiæ from an old iritis; and how the iris may contain a tubercular nodule, or be muddy and discoloured from iritis.

The **lower eyelids** are puffy and œdematous, especially in the morning, when the patient is suffering from Bright's disease; and a like appearance is often to be noted in patients who are suffering from very

severe paroxysms of cough. It is very characteristically present in children affected with whooping-cough. The eyelids may also be swollen and inflamed as the result of bug bites.

People look "dark under the eyes" when their digestion is out of order, or when fatigued, especially from want of sleep; often women are darker under the eyes during menstruation than at other times.

The **nose** has a sunken bridge in congenital syphilis; the tip is red in some cases of mitral regurgitation, in habitual drunkards, in females with chronic indigestion, and sometimes in purely local conditions. Undue mobility of the alæ nasi may be due to neurosis, or it may indicate obstruction to inspiration, and is in this respect very important to look for in infants. Young persons who suffer from adenoids, and to a lesser extent those afflicted with enlarged tonsils or chronic bronchitis, have pinched noses and open, fishy mouths. The pinching of the nose is due to falling-in of the alæ nasi where they lose the support of the nasal bones whilst the mouth is kept open to reduce the resistance to the entrance of air.

The **lips** are pale in chlorosis and other forms of anæmia; livid and blue in heart disease when compensation fails. A thick, short, and coarse-looking upper lip is often found in association with a phthisical tendency, whilst thin, mobile lips occur in persons of a neurotic temperament, and especially in female invalids whose constitutions are naturally weak, and who have not, nor can ever hope to possess, a large stock of vitality. Herpes on the lip is very often associated with inflammation of the respiratory tract, and is often an early and suggestive accompaniment of pneumonia. When it occurs in a patient who is obviously ill the chest should always be most carefully examined.

The **ears** are often ill developed in idiots, and sometimes in the insane develop hæmatomata. Of greater frequency is the occurrence of tophi in persons of gouty habit.

The **cheeks** give valuable information regarding the patient's health. In anæmia and aortic disease they are pale ; in hectic fever there is a bright circumscribed blush over the malar bones ; in the capillary engorgement of plethora they are ruddy and high-coloured, as they also are in many persons who lead an open-air life, exposed to all weathers ; in heart disease they are also high-coloured when back pressure has begun to tell on the systemic circulation, but the colour is of a bluish and cyanotic tint which cannot be mistaken for the rubicund cheeks of plethora. In unilateral chest inflammations, and particularly in pneumonia, the cheek corresponding to the affected lung may be flushed, but if the patient has been lying for some time on one side there is often a difference between the two cheeks, resulting from the pressure of the lower one upon the pillow, quite apart from the presence of disease.

The **form of the cranium** may also indicate some points of importance, to which reference is made in Chap. XI.

In addition to the appearance of individual features, the **general expression of the patient** must be noted. Is it animated, apathetic, or has it the absolute vacancy of unconsciousness ? Are there wrinkles on the face, or is it smooth ; or is one side smooth and the other wrinkled, as one sees it in unilateral paralysis of the seventh nerve ? Is the mouth drawn over to one side, and is there any other lack of symmetry between the two halves ? The expression may be characteristic of pain, or there may be a placidity resting on the features which gainsays the

assertion of a patient that his agony is most severe. A look of anxiety on a patient's face often presages serious illness at a time prior to the appearance of any other signs or symptoms which would suggest the gravity of the situation. Twitching of the face sometimes results from a nervous habit, at other times it is a symptom of definite disease, of which chorea affords a good example.

When **pain** is present, the various features are differently affected, according to its situation. Pain in the head, whether simple headache or of organic origin, causes the sufferer to frown; painful diseases of the chest and abdomen tend rather to affect the expression of the lower part of the face. These signs are of peculiar importance in the case of children who cannot describe their sufferings. Attempts have been made to associate certain lines which appear on the faces of sick children with diseases in special groups of organs. With the exception of a furrow which runs downwards from the ala nasi to curve round the angle of the mouth, and which is not infrequently present in cases of gastro-intestinal disorder, they are of little or no value for diagnosis.

The physiognomy of insanity is often highly characteristic, but descriptions of it must be obtained from special textbooks. In serious illness the nose often looks pinched, the eyes look sunken and lustreless, and the chin and malar bones sharp and prominent, owing to the loss of tone which the soft tissues have undergone.

Several types of expression have received special names. Of these the most important are the facies Hippocratica and the typhoid facies.

In the **facies Hippocratica** the skin is livid or pale, and opaque, the eyes are dull and sunken but remain open, the nose is sharpened, the temples

are hollow, the chin is sharp, the mouth is open through dropping of the lower jaw, the ears are cold and shrunken, and the cheeks drawn in. When this facies is associated with abdominal disease there is a red or livid ring around the eyes. The Hippocratic facies is a presage of impending dissolution. The **typhoid facies** is characterized by dull, lustreless eyes, tremor of the lips (with muttering delirium), and a blank, expressionless countenance. Associated with this are found a brown, dry tongue, a rapid pulse, a tendency to sink low in the bed, twitching of the tendons (*subsultus tedinum*), and a constant, purposeless picking of the bed-clothes.

The **state of the skin** where it is exposed must be carefully investigated. In the face we notice especially the **complexion.** This is dependent on two factors—the colour and the transparency of the skin. The most important abnormalities are pallor, yellowness, bronzing, an earthy tint, and a dusky bluish-red hue. **Pallor** occurs in various anæmic states, and also when the heart's action is greatly enfeebled, as in fainting or severe nausea. **Yellowness** may be due to pernicious anæmia, when the tint is pale lemon-yellow, which contrasts with the whiteness of the sclerotics ; or to jaundice, when the skin may be only faintly discoloured, or may be of a dark yellow colour, with excoriations from the scratching that results from the intense itchiness which the bile acids evoke. In jaundice the conjunctivæ and mucous membranes share in the coloration. **Bronzing** is found in Addison's disease, and affects both the skin and the inside of the mouth. An **earthy tint** is common in states of serious ill-health. It sometimes indicates a malarial history ; in other instances it is the result of syphilis or of cancer ; and in yet others it can be traced to an anæmic condition maintained by continual small losses

of blood, such as bleeding piles may involve. This tint is partly due to the altered state of the blood, partly to abnormal opacity of the skin. The **dusky tint** of embarrassed breathing and of advanced heart disease does not demand further notice here.

It is also important to search for **cutaneous eruptions,** some of which—measles and syphilitic rashes, for example—frequently appear first about the roots of the hair, whilst others have equally distinctive situations. Ulcers and scars should also be looked for. The colour and nutrition of the hair, and the dryness or moisture of the skin, must be noted; and if perspiration is present, its amount and situation. The perspiring brow of a rachitic child is very characteristic.

Reference has already been made to the **panniculus adiposus;** but, in addition to the presence or absence of fat, morbid conditions may lead to abnormal states of the cellular tissues. The chief of these is the presence of fluid or of air, the former being by far the commoner.

When fluid is present, the condition is that known as **dropsy,** and there are two varieties of this, which are sometimes described as "hydræmic" and "passive." In hydræmic dropsy, typical examples of which occur in sufferers from Bright's disease, the transudation does not first show itself in the most dependent parts of the body, but in other sites where laxity of the tissues favours its accumulation. Thus in chronic nephritis an early symptom is the œdema of the face, especially below the eyes, which comes and goes, being most noticeable when the patient first rises in the morning. In passive dropsy, however, which is typically present in those cases where pulmonary or cardiac disease produces a backward pressure in the veins, the swelling first appears at the ankles and over the

dorsum of the foot, and only gradually mounts to the legs, thighs, and trunk. When the venous obstruction is local, the dropsy is confined to the parts from which the return of blood is impeded. In this way one finds ascites resulting from cirrhosis of the liver, or œdema of an arm when the axillary glands are cancerous and constrict the axillary vein. Œdema of the whole upper part of the body may result from intrathoracic tumours ; the writer has seen it follow compression of the superior vena cava by an aneurysm. Dropsy may be recognized by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on pressure.

Localized œdema may be due to nervous causes, and is found in certain of the angioneurotic group of diseases.

Subcutaneous emphysema is not common, but when present it can be readily recognized by the crackling sensation which is detected on pinching the part affected.

The **hands** of the patient merit careful observation. Notice the strength of his grip as he shakes hands ; this often indicates improvement or the reverse with considerable accuracy. Their general shape should then be noted. Are they stunted, as in congenital cretinism, or "spade-like," as in myxœdema ? Are the joints large, as occurs in rickets and in persons of strumous diathesis ? Are they deformed as well, as occurs in rheumatoid arthritis, or swollen and painful, as in acute rheumatism ? Sometimes what looks like enlargement of the joints is really due to wasting of the surrounding tissues. When the patient is gouty, the finger-joints are often implicated, and nodules, known as **Heberden's knobs,** are formed. These must not be confused with **Haygarth's nodosities,** which are fibrous thickenings found in cases of

rheumatism.* Gout sometimes shows itself by producing a contraction of the palmar fascia that prevents extension of the fingers. In nerve disease the skin of the hand may undergo **trophic changes,** becoming thin and glossy ; or the vessels may be influenced by vasomotor disorders, and lead to redness or to a pallid and dead-looking state of the fingers. Nerve diseases also produce very characteristic movements or attitudes of the hand, as may be seen in athetosis, tetany, and lead palsy. **Tremor** of the hands is a frequent indication of disease. Among the conditions which produce it may be instanced paralysis agitans, multiple sclerosis, certain traumatic neuroses, Graves's disease, uræmia, insomnia, mercurial poisoning, alcoholism, abuse of tobacco, and senile degenerative changes. The methods of studying this symptom are detailed at p. 488. In ulnar paralysis the hand becomes deformed by over-extension of the first phalanges, combined with excessive flexion of the rest, so that a claw-like attitude is produced. This is known as the **"main en griffe."** When the muscles of the thenar and hypothenar eminences have undergone atrophy the hand becomes flattened, and thus somewhat resembles that of an ape. In acromegaly and in pulmonary osteoarthropathy there are very characteristic enlargements of the hands, which present a singularly massive appearance. The fingers become clubbed under conditions which produce chronic congestion of the peripheral veins, chief among such conditions being respiratory or cardiac embarrassment. Where the congestion is marked the finger-tips are blue and cold,

* Considerable difference of opinion exists as to what Heberden really described, but the terms are now generally used in the sense given in the text. Those who desire to consult the originals are referred to Heberden, "Commentaries on the History and Cure of Diseases," 2nd edit., p. 148 (London, 1803); and Haygarth, "A Clinical History of Diseases," Part ii. (Bath, 1805).

and the nails are much curved longitudinally. The nails exhibit longitudinal grooves in gouty persons, in whom also they are often singularly hard and brittle; whilst a transverse furrow is the record of some former interference with the nail's nutrition, and, in the absence of a local cause, points to some severe constitutional illness. Many persons, especially those of the so-called lymphatic temperament, have moist and clammy hands. These in women frequently indicate excessive leucorrhœal loss, and so aid in directing inquiry towards this subject. Even if the hand be not moist when the patient is seen, the fact that the colour has been sweated out of the palm of the glove may show that the tendency exists. In infants the movements or position of the hands and fingers will often direct an acute observer to the seat of disease.

The **neck** should always be inspected, and special note taken of any of the conditions described in the paragraphs that follow.

1. The state of the **lymphatic glands.** In syphilis the glands under the upper part of the trapezius are very frequently enlarged. In septic ear diseases the glands below the ear can often be readily felt. Where there are carious teeth, and where there is malignant disease in the mouth, enlarged glands can usually be detected near the angles of the jaw. In scrofulous persons enlarged tubercular glands occur in groups or in long chains beside the sterno-mastoid, and scars will mark the points where they have suppurated. In lymphadenoma the glands are enlarged and firmly matted together. If enlarged glands are found either in the neck or elsewhere, it is important to observe whether they remain firm and distinct, or become fused together, or whether fluctuation can be detected.

2. The **thyroid gland.** The existence of any swelling of this gland is important, and its effect on

the patient's respiration should be studied. Sometimes such enlargements exercise considerable pressure on the trachea ; at other times, particularly if the disease be malignant, the recurrent laryngeal nerves may become implicated. In cases where there is difficulty in determining whether a tumour is connected with the thyroid, much assistance may be obtained from the fact that the gland and any tumour which is connected with it move up and down with the larynx during deglutition.

3. **Unusual prominence of any muscle** or group of muscles in the neck should be described. Such prominence may be bilateral, as of both sterno-mastoids in emphysema, or unilateral, as in tonic wry-neck. A congenital sterno-mastoid tumour may be present, and, if unrecognized, may lead to much perplexity ; whilst various cysts, cold abscesses, or developmental abnormalities may be encountered. Their recognition, however, is rather a question for surgical diagnosis.

4. **Movements of the laryngeal box** are sometimes conspicuous, and may call for explanation.

5. **Rigidity of the neck** may be due to inflammation, to rheumatism, to disease of the spinal column, or to various nervous diseases, whilst spasmodic movements occur in clonic torticollis.

6. Any **bulging of the apices of the lungs** during a fit of coughing, or pulsations seen in the vessels, must be recorded, nor must the existence of aneurysm be overlooked.

7. **Boils and carbuncles** are very frequently situated on the back of the neck. As they are not infrequently present in cases of diabetes, they should direct the observer's inquiries to the urinary system, and sugar should be tested for.

8. Finally, with respect to the **general shape of**

the neck, it should be noted whether it is short and thick, or long and smooth, or "scraggy," or projecting forwards with a prominent larynx. The last form is common in persons with phthinoid chests.

The character of a patient's **respiration** is often of great service in reaching a diagnosis and a prognosis. Under the name of *extra-auscultation* Professor Wyllie has grouped together the various phenomena connected with respiration which are to be heard, apart from those revealed by the stethoscope, when standing at the bedside of the patient.* In the following scheme his classification is adopted:

CLASSIFICATION OF THE PHENOMENA OF EXTRA-AUSCULTATION.

I. Obstructive noises in the respiratory passages.

1. In the nose
   - *a.* Thickened mucosa, or accumulated secretion.
   - *b.* Paralysis of alæ nasi.
2. In the back of the throat
   - *a.* Nasal stertor. The soft palate strikes the back of the pharynx.
   - *b.* Oral stertor. The soft palate strikes the tongue, which has fallen back.
3. In the larynx
   - *a.* Swelling of cords
   - *b.* Paralysis or spasm of glottis.

   } Laryngeal stridor (almost invariably inspiratory).
4. In the trachea
   - *a.* Tracheal stridor (leopard growl).
   - *b.* Tracheal rattle (death-rattle).

   } (Occur during both inspiration and expiration).
5. In the bronchi
   - *a.* Musical sounds (wheezing).
   - *b.* Crepitant sounds.

II. Cough.

1. Duration. Single coughs, repeated coughs, paroxysms.
2. Quality. Resonant or toneless, moist or dry, suppressed or free.

III. Hiccough.

IV. Voice.

1. Volume. 2. Quality.

* *Edinburgh Hospital Reports*, vol. i., p. 48.

When the **respiratory passages are obstructed** the normal quiet respiratory sound is replaced by more or less noisy breathing. When the obstruction occurs in the nose, either from mucus in the meatus, or from thickening of the mucosa which covers the turbinated bones, or from paralysis of the alæ nasi, the breathing is sniffing or bubbling in character. When the soft palate is relaxed, and especially when it is paralysed, it prevents the free passage of air between the mouth and thorax, and produces a snoring or stertorous sound. When the rima glottidis is obstructed from any cause, such as spasm or paralysis of the vocal cords or œdema of the larynx, stridulous breathing results. If a polypus or other tumour lie between the cords, there may either be stridor or simply noisy breathing. The trachea may have its airway narrowed by pressure from the outside, as in cases of tumour and especially of aneurysm, when the breathing becomes growling; or mucus may obstruct the lumen, producing a rattling sound. The "death-rattle," which occurs when weakness and insensitiveness combine to prevent any effort at expectoration, is a typical example of the condition. Obstruction in the bronchi gives rise to wheezing and crackling sounds. Sometimes the respiration is sighing; Gairdner is inclined to think that this may indicate fatty degeneration or slight dilatation of the heart. An important division of dyspnœic conditions may be made according as the difficulty in respiration is felt during the inspiratory or the expiratory period. Most cases of obstruction of the air-passages are characterized by *inspiratory dyspnœa*, whilst many of the pulmonary causes of dyspnœa produce *expiratory trouble*. As a common example of the latter one may cite the prolonged expiration in a case of bronchitis with emphysema. The breathing may be characteristic of

diseases quite distinct from those of the respiratory system. Examples of this are found in the stertorous breathing of apoplexy, the hissing expiration of uræmia, and the dyspnœa or "air-hunger" of commencing diabetic coma, which affects both inspiration and expiration.

If **cough** be present, its character must be most carefully noted.* The first thing to observe in this connection is whether the cough consists of independent explosive expirations, or is paroxysmal in character. The former occurs in early phthisis, in granular pharyngitis, and in some forms of nervous irritation; the latter is often found in severe bronchitis, and is very typical in pertussis. One should also notice whether the cough induces pain or nausea, and whether its tone is resonant, or suppressed, or husky. In *common colds* the cough is at first short and dry, but as the quantity of secretion increases, the type becomes more paroxysmal, and the fit of coughing continues till the mucus is expectorated. In *bronchitis* the condition resembles that found in the last affection, but the paroxysms are more severe, and wheezing is often present. When due to *early phthisis*, the cough is frequent, short, and sharp, and is described as *dry* because there is no rattling of mucus associated with it. Later in the disease, when the caseous masses are breaking down, secretion is much more copious, and the cough becomes moist and paroxysmal. In severe cases actual vomiting may be induced. A *nervous cough* generally has the character of single, short, dry explosions, repeated at intervals, and a similar type

* The student will observe that two elements must be discriminated in a cough. The first of these is the *explosive element*, due to the sudden opening of the valve formed by the false cords and surrounding structures; the second is the *vocal element*, due to the rush of released air between the true cords (*vide* Wyllie, *loc. cit.*).

is produced by irritation of the peripheral nerves, whether the source of the irritation be found in a disordered stomach, or threadworms in the rectum, or be due to disease in the ear or to the discomforts of teething, or take origin in the nerves of the pregnant uterus. Local conditions in the *throat* may be the cause of most troublesome and persistent coughing, and a careful observer will not fail to look for granular pharyngitis when the patient complains of constant hawking, or for a relaxed and trailing uvula, more particularly when the cough starts the instant the patient lies down.

In *pleurisy*, *pneumonia* (associated as it often is with more or less pleurisy), and in *pleurodynia*, the cough consists of solitary dry, hacking, expulsive efforts, suppressed as much as possible to prevent unnecessary pain, but repeated frequently. In *laryngitis* and *croup* the cough may be simply noisy, but more often is either husky or stridulous. When the lumen of the trachea is encroached upon by a *mediastinal tumour* or an *aneurysm* there is generally a very resonant, brassy cough, aptly compared to the cry of a gander. When once heard, this is almost sufficient to clinch the diagnosis without further examination.

In *hysteria* the cough is often loud and barking, and gives the impression of being produced with the view of attracting attention. Such a cough is sometimes associated with hysterical aphonia. *Pertussis*, when it is fully developed, is distinguished by a most characteristic cough. There is first a long-drawn, almost stridulous inspiration, then a series of short, sharp, expiratory coughs, which follow each other with extreme rapidity. The face turns dark and the veins grow prominent, the child clings firmly to any support it can find, so as to give full play to the accessory muscles of respiration, and when at last the fit of

coughing ends it is followed by a long-drawn whooping inspiration. The severity of the paroxysm induces vomiting, and sometimes causes evacuation of the bladder and bowel.

Hiccough, which results from spasmodic contraction of the diaphragm, is a common enough disorder. It may be due to trivial causes, such as an attack of indigestion; but it also occurs, and that most persistently, in many serious illnesses, when the symptom may become one of considerable gravity. Thus, if it be met with in a patient whose kidneys are affected, and especially if the occurrence in such a case follow the passage of instruments to relieve stricture, there is ground for serious apprehension as to the issue.

The **voice,** as well as the cough, should be studied. The chief points to observe are its strength, whether it is clear or husky, or whether aphonia exists. The voice may be nasal either through habit or in consequence of obstruction in the upper airways. A distinction should be made between open and stopped nasal tones, the former resembling the sound produced when the mouth is kept shut during phonation, the latter that heard when one speaks whilst holding the nose.

Temperature.—The hand laid upon the skin gives a certain amount of information as to the temperature, especially if there be no perspiration; but a far more accurate guide is found in the **thermometer,** whose use should never be omitted.* In taking the temperature the following practical points must be attended to :—

1. The thermometer must be accurate and of good quality. To ensure accuracy, it should be compared

* Just because the hand takes account of moisture as well as of the actual temperature, it may convey information which the thermometer fails to impart—e.g. the "pungent" dry heat of early pneumonia is most characteristic.

with a standard instrument. In Britain this is done at Kew, and certificates are issued which state the error of each individual instrument. In process of time, however, and particularly if the thermometer has been recently made, molecular changes occur in the glass which tend to make the reading too high. Such changes are slight, and seldom attain a value of any clinical importance, though if great accuracy is necessary a fresh comparison should be made every two or three years. Also, if the bulb of the thermometer be made too thin, the glass will yield to pressure, and the patient may either purposely or accidentally compress it so much as to make the mercury reach to four or six degrees above the actual temperature.

2. The thermometer must be kept in position long enough to allow the mercury to reach the body temperature. Generally it is well to exceed the period which the instrument professes to require.

3. In adults the temperature is taken in the mouth or in the axilla ; in young children the thermometer should be placed in the fold of the groin, and the thigh flexed on the abdomen, or it may be inserted into the rectum. The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla, but in old people the mouth temperature is often too low, and less trustworthy than that of the axilla. When it is taken in the latter situation, care must be exercised to keep the part as free as possible from perspiration, both during the observation and for a few minutes before it. Moreover, the arm should be drawn to the side for a short time before the thermometer is inserted, that the skin may not have been chilled by exposure to the air.

4. Before inserting the thermometer, make an invariable rule of washing it in lotion or in cold water, and see that the mercury is well shaken down ; wash

it again before replacing it in its case. In Great Britain the Fahrenheit scale is used, on the Continent the Centigrade.*

The temperature *should be taken at fixed times*, twice daily when possible, and at shorter intervals when fever fluctuates or runs high. Times that are convenient, and that fairly represent the daily conditions, are 9 a.m. and 7 p.m. In health the temperature has a daily range of from one to two degrees Fahrenheit, being lowest in the small hours of the morning, and gradually rising to attain its principal maximum somewhere about 5 or 6 p.m. Age exercises a rather marked influence on the temperature. In children it varies greatly with their time of life, and trivial causes produce great fluctuations. On the average, it is about half a degree higher than in adults. In the very old it is also slightly higher than in middle life, unless the circulation is weak, when the temperature may be considerably lower.

In diseased conditions marked **deviations from the normal temperature** are often present. Temperatures may be classified as follows :—

Normal† . .	98°–99° F. or 36·6°–37·2° C.
Subnormal .	Below 98° F. or below 36·6° C.
Collapse . .	„ 96° F. „ 35·5° C.
Febrile . .	Above 99° F. or above 37·2° C.
Hyperpyrexia .	„ 107° F. „ 41·6° C.

By consecutive observations, taken at suitable intervals, it is easy to determine whether an abnormal temperature is constantly present, or only occurs at intervals. When the temperature rises quickly, the patient feels chilly in consequence of the incomplete

* For a comparison of the two scales, *see* Appendix 4, p. 625.

† The figures given are merely approximate. Wunderlich allows a rather wider range for normal temperatures than those stated above, and, taking the mean normal temperature at 37° C., would allow a range of from 36·25° to 37·5° C.

response of the vasomotor mechanism to the new conditions, and in marked cases **rigors** occur. If, however, the temperature remains continuously high, the rigor gives place to a feeling of heat, coupled with thirst, headache, and a rapid pulse. This is known as **pyrexia,** or fever. If after fever the temperature falls rapidly, or if during the fever the extremities are chilled, the patient suffers from **collapse,** when the pulse is small, the features are pinched, the skin is

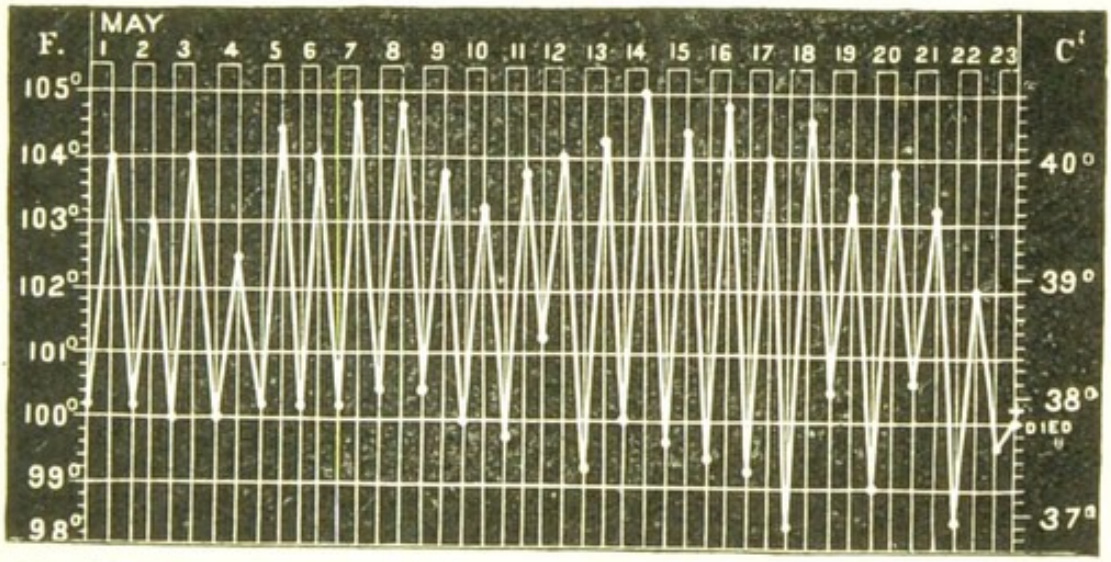


Fig. 1.—Remittent fever (hectic). Case of phthisis. (*After Finlayson.*)

moist with a clammy sweat, and the patient suffers from a sinking sensation and from nausea.

There are three principal **types of fever**—the continued, the remittent, and the intermittent. When fever does not fluctuate more than about a degree and a half (Fahrenheit) during the twenty-hour hours, and at no time touches the normal, it is described as **continued.** When the daily fluctuations exceed two degrees, it is known as **remittent** (Fig. 1); and when fever is only present for several hours during the day it is called **intermittent.** In remittent fever the evening temperature is usually higher than the morning one, but in some cases, not infrequently in phthisis,

this type is *inverted*, and the "remission" occurs in the evening, whilst there is a morning "exacerbation." When a paroxysm of intermittent fever occurs daily, the type is said to be "**quotidian**"; when on alternate days, "**tertian**"; when two days intervene between consecutive attacks, "**quartan**" (Fig. 2). A "**double tertian**" is the name given to a daily fever when the paroxysms occurring on the first, third, fifth, and following odd days differ from those of

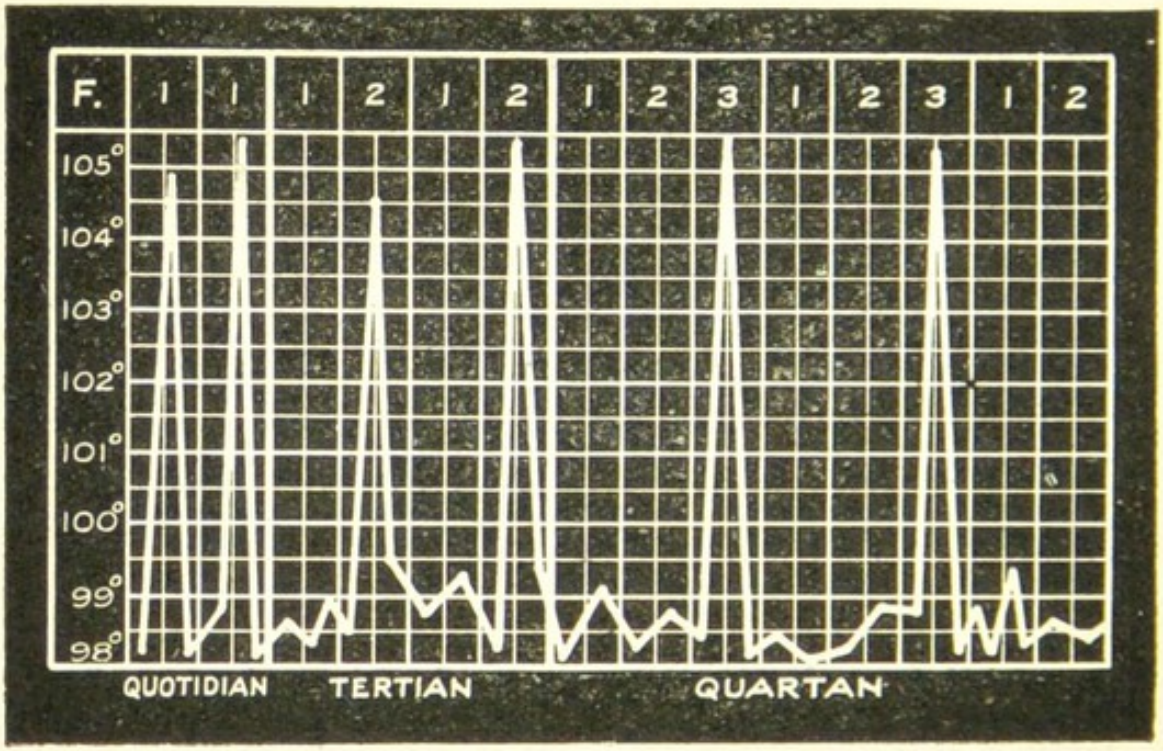


Fig. 2.—Intermittent fevers.

the second, fourth, sixth, and following even days in hour of appearance, in severity, or in character.

The **course of a fever** is divided into three stages—the initial or pyrogenetic, "*stadium incrementi*"; the stage of full development, or "*fastigium*"; and the stage of termination, or "*stadium decrementi*." When the fever ends rapidly it is said to resolve by **crisis** (Fig. 3); when gradually, by **lysis** (Fig. 4). Not seldom crisis is preceded by a

short but marked rise of temperature, accompanied in many cases by delirium ; it is sometimes followed by collapse.

In the study of any case of fever the points which require to be observed are whether the type is one of apathy and indifference, or of restlessness and twitching ; whether, and if so how far, the sensorium has been involved ; what the height of the temperature is, and what its course has been ; what are the rate and character of the pulse ; whether the skin is moist or dry, or exhibits any eruption ; and which of the viscera or secretions are characteristically affected. The explanation of these points may be found in works on medicine, but their true significance can only be learned at the bedside.

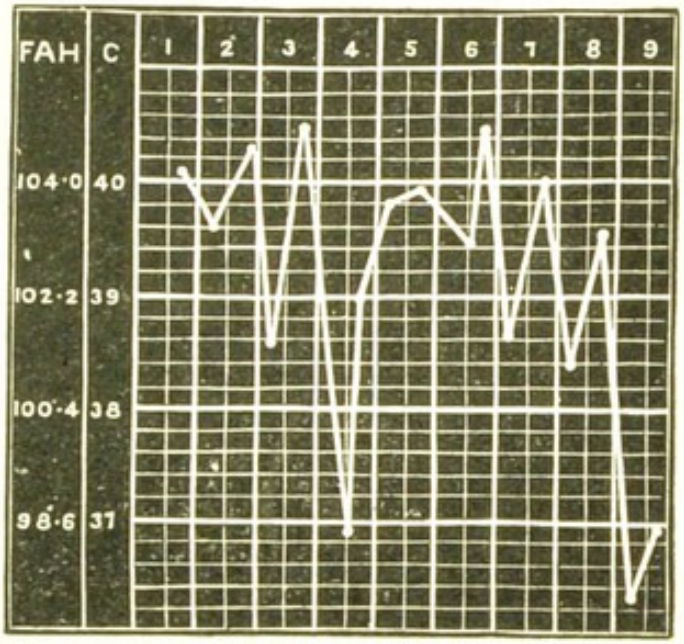


Fig. 3. Crisis. Case of lobar pneumonia. (*After Wunderlich.*)

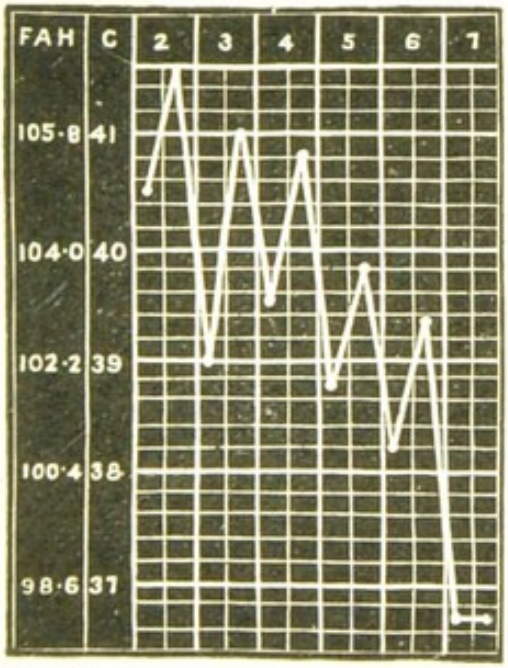


Fig. 4.—Lysis. Case of broncho-pneumonia. (*After Wunderlich.*)

CHAPTER III

THE ALIMENTARY SYSTEM AND ABDOMEN

I. THE MOUTH, THROAT, AND ŒSOPHAGUS

The Mouth and Throat

The mouth.—For the examination of the mouth the patient should be placed facing a good light. If artificial light be used it should be thrown into the mouth by means of a reflector. A piece of candle wrapped in blotting-paper and held in front of a bright spoon forms a good extemporized light and reflector.

The lips.—Note the colour of the lips. They are blue in cyanosis, pale in anæmia. Note the presence of any crusts, fissures, or ulcers. The lips should be everted in order to permit of an examination of their inner surfaces. Herpes of the lips is often seen in inflammatory conditions of the air-passages and lungs, especially in croupous pneumonia.

The teeth.—The **temporary teeth** are cut in the following order :—

First.—The two lower central incisors, sixth to eighth month.
Second.—The four upper incisors, eighth to tenth month.
Third.—The lower lateral incisors and all the front molars, twelfth to fourteenth month.
Fourth.—The canines (upper first), eighteenth to twentieth month.
Fifth.—Posterior molars, at two to two and a half years.

The **permanent teeth** appear as follows :—

First molar at six years.
Central incisors at seven years.
Lateral incisors at eight years.

Bicuspid (anterior) at nine years.
Bicuspid (posterior) at ten years.
Canines at eleven to twelve years.
Second molars at twelve to thirteen years.
Third molars at seventeen to twenty-five years.

The following table shows the relations of the permanent and the temporary teeth :—

		M.	C.	I.	I.	C.	M.	
TEMPORARY	Upper	2	1	2	2	1	2	20
	Lower	2	1	2	2	1	2	

		M.	BI.	C.	I.	I.	C.	BI.	M.	
PERMANENT	Upper	3	2	1	2	2	1	2	3	32
	Lower	3	2	1	2	2	1	2	3	

The presence of any irregularity or defect or carious disease in the teeth should be noted. It should be observed whether there is any exposure of their roots, or whether they are surrounded with tartar. Grinding of the teeth leads to bevelling of their edges ; this is especially found in young children. The presence of "Hutchinson's teeth" is important as affording evidence of congenital syphilis. In this condition the two central upper *permanent* incisors are at a higher level than the adjoining teeth ; they are rounded in section and slope inwards below, they are broader nearer the gum than at the crown, so as to be peg-shaped, and they present a semilunar notch at their ends. They are usually discoloured as well. In the same condition the molars tend to be dome-shaped.

The gums.—Their colour should first be noted. In lead poisoning a blue line can often be observed running along the gum near the insertion of the teeth, and especially, perhaps, on the gum between the teeth. In copper poisoning a greenish line can sometimes be seen in a similar position. The gums may be swollen and spongy in scurvy. They are sometimes retracted or show ulcerations or hæmorrhages. Ulcers and

hæmorrhages may also be observed in the buccal mucous membrane in various conditions.

The tongue.—Ask the patient to protrude it. Note if it is put out in a straight line. Observe its size and shape, whether broad or pointed. Look for tremulousness of the whole tongue and for fibrillary twitching of it. Note in the dorsum (1) its colour : is it pale, red, or discoloured ? (2) Is it dry or moist ?

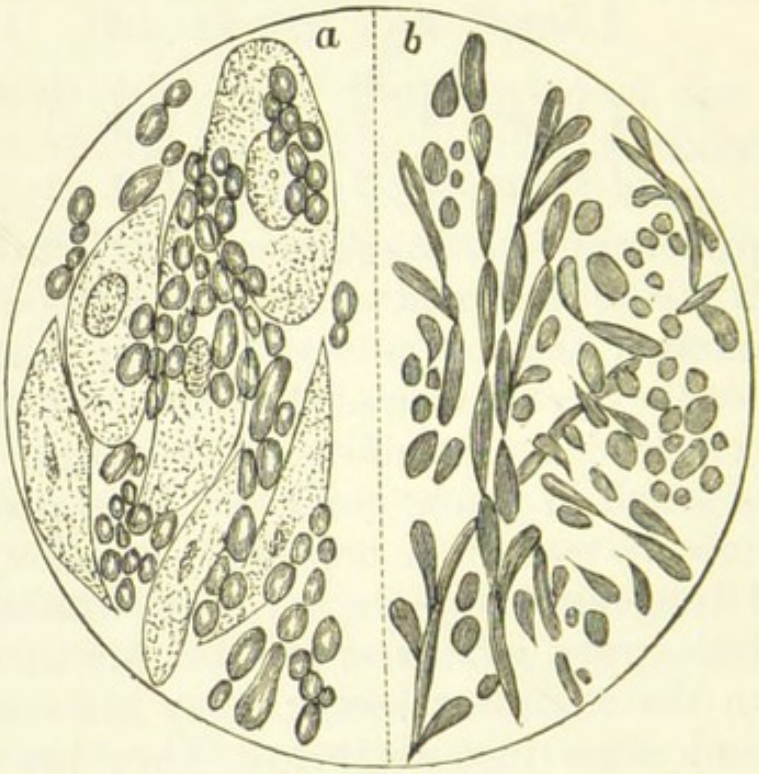


Fig. 5.—*a*, Scraping from a patch of thrush ; × 800. *b*, Culture of *Saccharomyces albicans* (*Monilia candida*)—the fungus of thrush ; × 800.

(3) The presence or absence of fur ; the colour and distribution of the latter should be noted. (4) The character of the papillæ. (5) The under surface of the tongue—a small ulcer on the frænum is sometimes seen in persistent coughing, and particularly in whooping-cough. Lastly (6), observe the edges of the tongue. Look for ulcers, indentations of the teeth, etc., on them.

The presence of **thrush** may sometimes be observed on the surface of the buccal mucous mem-

brane, especially in children. It presents the appearance of small white points or patches raised somewhat above the surrounding surface, which is sometimes redder than normal. Patches of thrush are very apt to be mistaken for small milk curds. They may be distinguished by the fact that milk curds can be easily detached, while thrush patches can only be removed with difficulty, and when removed are apt to leave behind a raw surface. To search for the fungus (*Saccharomyces albicans*) a small piece of the patch should be scraped off and examined in a drop of glycerine. A quantity of epithelial débris, along with bacteria and leucocytes, will be seen, and mixed up with these the filaments of the fungus. These consist of long but unequal segments, each usually possessing a refractile nucleus at each end (Fig. 5).

The palate, fauces, and pharynx.—Introduce a tongue depressor, and note first the general colour of the soft palate, fauces, and pharynx ; observe any abnormal degree of pallor or redness. The yellow tinge of jaundice often lingers long on the soft palate, and in commencing measles a patchy redness can be made out very early in the same situation. Note the presence of any ulcers or mucous patches on the palate, fauces, or tonsils. Look carefully at the **tonsils,** noting any enlargement of them. Yellowish or greyish points or patches may sometimes be seen on their surface. Try whether these can be wiped off, leaving a sound surface, as is the case with accumulated follicular secretion, or whether removal leaves behind a raw surface, as happens with the false membrane of diphtheria. Note always whether or not the soft palate and uvula show any similar spots or patches. Next look at the **pharynx.** The presence upon its surface of a number of flat adenoid swellings, somewhat resembling sago grains, is so

common as to be almost a normal appearance. In granular pharyngitis these are much increased. A few dilated venules can also be frequently observed. Note the presence of any pus or excess of mucus on the surface, and the existence of any ulceration. In retropharyngeal abscess the posterior wall of the pharynx is bulged inwards. Sometimes this can be more easily made out by palpation.

The breath.—The character of the breath may be noted at this stage. If it be offensive, ask the patient to breathe out first through the nose only, and then through the mouth, and observe whether the odour is present on both occasions or not. This affords an indication as to whether the source of the odour is in the nose or mouth only, or whether it is lower down than either. If the odour proceeds from the nose, make a rhinoscopic examination (p. 550), looking especially for the presence of a foreign body or for evidence of atrophic rhinitis or other local disease. Bad teeth, ulcerations of the gums or mucous membrane, and enlarged tonsils, accompanied by retention and decomposition of secretion in their follicles, are the commonest sources of offensiveness in the mouth.

In gangrene of the lung the breath has a putrid smell. In bronchiectasis, also, it has a peculiarly offensive odour only to be recognized by experience. Fetor due to pulmonary conditions is best brought out by asking the patient to cough.

Slighter degrees of offensiveness may be due to gastric disorder or to prolonged constipation.

In uræmia the breath has a urinous or ammoniacal odour. In diabetes it is sweetish, like new-mown hay. In cases where diabetic coma is impending, the odour becomes ethereal. Various drugs—e.g. turpentine, creosote, paraldehyde, etc.—impart their characteristic odours to the breath, while in the case of patients who

are taking bismuth a garlicky odour can often be observed. Iodides produce a peculiar fetor.

The Œsophagus

Special anatomy.—The œsophagus is from 9 in. to 10 in. long. It begins opposite the cricoid cartilage, and ends opposite the 9th dorsal spine. It is crossed by the left bronchus between the 4th and 5th dorsal vertebræ.

Exploration of the œsophagus.—This is done in cases in which there are signs of stricture. It is best carried out by means of a stomach-tube. The latter should never be passed, however, unless one has first excluded the possibility of the existence of an aneurysm. It should also be avoided in cases where there has been any recent hæmatemesis.

It is best to use a long, red rubber stomach-tube, No. 20 or 21 in the English scale. It should be at least a yard long—not too thin in the wall, rounded at the end, and with at least one large and bevelled eye. Before being used it should be thoroughly cleaned, and then dipped in hot water; oil is unnecessary.

If the pharynx be very irritable, it may be anæsthetized with cocaine, either by painting or by giving the patient a pledget of wool to suck, soaked in a 5 per cent. solution, and directing him to keep the saliva at the back of the mouth. After the lapse of five minutes, one can proceed to pass the tube.

The patient should be sitting up, with the head slightly bent forwards. His mouth is open, but the tongue *not* protruded. The physician grasps the tube in his right hand, and passes it back in the middle line to the posterior wall of the pharynx. It is not usually necessary to introduce a finger into the mouth in order to guide the tube, as there is no real danger of entering the larynx. The patient is then told to

swallow, and the tube is "paid out" until it reaches the stomach, or until it is permanently arrested. Whilst this is going on the patient should be told to breathe freely through his nose.

During this manipulation one has to look out for the following :—

1. *Pain* on passing the tube; its site should be noted.

2. The presence of an *obstruction*. If the tube be arrested, one must not conclude all at once that a stricture exists. Frequently the tube is seized by a muscular spasm of the œsophagus. On waiting for a moment, however, this always passes off. If a permanent obstruction be discovered, one has to attempt to localize it. The commonest sites for a stricture are—(*a*) At the entrance; this is 6 in. from the incisor teeth. (*b*) Where the œsophagus is crossed by the left bronchus; this is 8 in. to 9 in. from the teeth. (*c*) At the cardiac orifice; this is about 17 in. from the teeth. It is interesting to note that these are also the positions in which some normal narrowing of the œsophagus exists.

3. One has to look for *diverticula*. The existence of such should be suspected when the tube passes very regularly at one time, but is obstructed at another.

The presence of any blood on the tube after its withdrawal is an indication of the presence of ulceration. Sometimes, also, fragments of new growth can be detected in the eye of the tube.

Auscultation of the œsophagus.—This is done in order to note the presence or absence of the sound produced by swallowing. In order to auscultate the œsophagus in the neck, the stethoscope should be placed at the left side of the trachea. In the upper part of the thoracic course of the œsophagus (as far as the 6th dorsal vertebra) it should be placed just to

the *left* of the dorsal spines, and below this just to their *right*. The patient is directed to take a mouthful of water, and to retain it until told to swallow. When he swallows, one hears a noise similar to that heard in one's own ear on swallowing saliva. The higher up one listens, the louder is this sound. If an obstruction be present, the sound is either not heard at all below that point, or it is greatly delayed.

II. THE ABDOMEN

Anatomy.—The natural lines on the surface of the abdomen are (1) the linea alba; (2) the lineæ semilunares; (3) the lineæ transversæ.

The **linea alba** is often selected as the site of puncture in tapping the abdomen. The structures lying behind it, from above downwards, are (*a*) the left lobe of the liver, extending to about three fingers' breadth below the ensiform; (*b*) part of the stomach, unless when empty; (*c*) the transverse colon, reaching as low as the umbilicus; (*d*) coils of intestine covered by omentum; (*e*) the bladder when distended, and the uterus when pregnant.

The **linea semilunaris** runs from the lowest part of the 7th rib to the spine of the pubes. It is about 3 in. from the umbilicus, but lies farther out when the abdomen is distended. The gall-bladder lies just to the outer side of the linea semilunaris of the right side.

Of the **lineæ transversæ,** one is opposite the umbilicus, another at the ensiform, and a third midway between these points.

In addition to these markings, the abdomen has been artificially divided into **regions** by means of vertical and horizontal lines.* The vertical lines are

* In describing the regions of the abdomen we have followed Quain. We should like, however, to point out that for clinical purposes the *interspinous line*, drawn between the two anterior superior iliac spines, has many advantages over the bi-iliac.

drawn upwards from the mid-point of Poupart's ligament on each side. The transverse lines are (1) the infracostal, drawn across horizontally at the level of the lowest points of the 10th costal cartilages, and (2) the bi-iliac, between the most prominent points of each iliac crest. Nine regions are thus marked off in three vertical rows. Those in the middle row are, from above downwards, the epigastric, umbilical, and hypogastric, and in each lateral row we have the (right or left) hypochondriac, lumbar, and iliac regions. The contents of these regions are exhibited in the table on the next page.

The *umbilicus* is $1\frac{1}{4}$ in. to $1\frac{1}{2}$ in. above the level of the bi-iliac line, and lies opposite the upper part of the 4th lumbar vertebra. Its position is far too variable for it to be a trustworthy landmark.

The *aorta* bifurcates about $\frac{3}{4}$ in. below and slightly to the left of the umbilicus, the *iliac arteries* running in a line drawn from that point to the middle of Poupart's ligament.

The *cœliac axis* arises at a point $4\frac{1}{2}$ in. to 5 in. above the umbilicus, and the *renal arteries* about an inch lower than the cœliac axis.

The transpyloric plane is often used as a guide in the examination of the abdomen. It is defined as lying midway between the suprasternal notch and the upper border of the symphysis pubis. It usually lies about halfway between the xiphisternal junction and the umbilicus, and it corresponds posteriorly with the lower border of the 1st lumbar vertebra.

General Examination of the Abdomen

The patient should be lying on his back in a good light. The abdomen is exposed by turning down all the bedclothes except the inner sheet. The night-shirt should then be drawn up, and, lastly, the sheet

Contents of the Abdominal Regions

Right Hypochondriac	Epigastric	Left Hypochondriac
Most of R. lobe of liver.	Part of R. lobe of liver. Whole of L. lobe of liver (usually). Gall-bladder.	Part of L. lobe of liver (sometimes).
Hepatic flexure of colon.	Part of stomach, including orifices. 1st and 2nd parts of duodenum.	Part of stomach. Splenic flexure of colon.
Part of R. kidney	Pancreas and upper end of spleen. Parts of the kidneys. Suprarenals.	Tail of pancreas and most of spleen. Part of L. kidney.
Right Lumbar	**Umbilical**	**Left Lumbar**
Part of R. kidney Ascending colon.	Part of R. and sometimes both kidneys. Most of transverse colon. 3rd part of duodenum.	Part of L. kidney (sometimes). Descending colon.
Part of ileum (sometimes).	Coils of jejunum and ileum. Part of mesentery and great omentum. Part of stomach.	Part of jejunum.
Right Iliac	**Hypogastric**	**Left Iliac**
End of ileum. Cæcum and vermiform appendix.	Coils of ileum. Upper part of rectum and sigmoid loop. Bladder in children and (if distended) [in adults. Gravid uterus.	Coils of jejunum and ileum. Sigmoid flexure.

folded down a little above the level of the pubes. These details are of especial importance in the case of female patients. Before beginning the examination of the abdomen, make sure that the bladder is empty. If necessary, a catheter must be passed.

Inspection of abdomen.—Look first at the general contour of the abdomen. Is it of normal fullness, is it swollen or protuberant, or is it sunken or retracted? If there be any bulging, note if it be general or local. General fullness, it has been epigrammatically remarked by Professor Wyllie, may be due to "fat, fluid, or flatus." If one were to venture to improve upon this, it would be to add "fœtus" as a possibility in the case of women. It must further be remembered that a *new growth* may also be a cause of general abdominal tumidity. The mode of distinguishing these conditions will be considered when we come to ascites. In general bulging it should be noted whether the distension is most marked in the antero-posterior or in the transverse diameter.

If the bulging be merely local, observe in which zone it is situated. Is it above or below the level of the umbilicus, and in which of the abdominal regions is it most marked? Lastly, note if there is any movement to be seen in the swelling, either along with or independently of respiration.

Pulsation in the epigastric region is a phenomenon which may be noticed on abdominal inspection, apart from any bulging in that region. The causes of it are (1) distension of the right ventricle (*see* p. 120); (2) venous pulsation of the liver; (3) aortic pulsation. The last is a condition which is very frequently observed in nervous subjects, especially women. The cause of it is obscure. It may be distinguished from right-ventricle pulsation by being situated somewhat to the left of the middle line, by the

fact that it can usually be traced downwards towards the bifurcation of the aorta, and by its being not quite synchronous with the apex beat, but somewhat delayed. (4) Transmitted pulsation from a tumour overlying the aorta. (5) Aneurysmal. This is, perhaps, the least common cause of epigastric pulsation. The pulsation in this case is expansile, a fact which can best be elicited by placing the narrow ends of two stethoscopes one on each side of the swelling, and observing if the other ends move apart at each impulse.

The **movements of the abdominal walls** should be studied. Normally, they bulge during inspiration, and fall in again during expiration. In paralysis of the diaphragm the reverse holds true; sometimes the paralysis is unilateral, in which case one side of the abdomen will move naturally. Cessation of movement of the abdominal walls is a valuable sign of peritonitis.

Sometimes **peristaltic waves** are visible through the abdominal wall. This is especially apt to be the case in chronic intestinal obstruction. The coils of intestine above the constricted part then stand out prominently. From this a definite "pattern" of abdominal tumidity results, depending on the site of the obstruction. If, for example, there be a constriction at the ileo-cæcal valve, the distended coils of small intestine may often be observed standing out in the centre of the abdomen one above the other, so as to form a "ladder pattern." On the other hand, if the obstruction be low down, say in the sigmoid flexure, the pattern of tumidity is one in which the periphery of the abdomen is chiefly affected. A dilated stomach may also stand out as a prominent tumour in which peristaltic waves are visible. The direction of such waves should always be noted. If

absent, they can often be elicited by flicking the surface with a wet towel, or by merely sharply tapping it with the finger. Peristaltic waves in the stomach run from left to right; those in a distended transverse colon, from right to left. This may sometimes be of diagnostic value.

Attention should next be paid to the **surface of the abdomen.** In great distension the surface is smooth and glossy. *Striæ* (white lines in the epidermis) should be looked for; they indicate former distension. Note any *distension of the surface veins*, and endeavour to ascertain in what direction the blood in them is flowing. In obstruction of the inferior vena cava the inferior epigastric veins are full from the establishment of a collateral circulation. In such cases also a large *lateral vein* can be seen running up about the midaxillary line, and thus establishing a communication with the tributaries of the superior vena cava. In portal obstruction a number of distended veins may often be seen radiating from the umbilicus. To this appearance the term "caput Medusæ" has been applied. It is due to establishment of a connection between the portal and parietal veins by means of the round ligament. *Pigmentation* of the abdominal wall is sometimes important. Along the middle line it forms the linea nigra—one of the signs of pregnancy. Note the appearance of the *umbilicus*. Is it depressed, level with the surface, or bulging? Is there any excoriation around it? Lastly, one should never omit to look at the usual sites for any evidence of hernia.

Palpation of the abdomen.—The patient should be on his back, with the knees drawn up, and the shoulders a little raised. He should be told to keep the mouth open and to breathe quietly, or his attention may be diverted by conversation. The

hand of the physician must be warm. Ordinary palpation should be performed with one hand only. In order to gain the confidence of the patient's abdominal muscles, the hands should be allowed to rest for a moment on the surface of the abdomen before palpation is actually commenced. Each region should be palpated systematically. Poking with the finger-tips should be avoided, the best movement being a gentle one from the metacarpo-phalangeal joints. During expiration the receding abdominal wall should be followed by the fingers, and a gentle rotatory motion of the finger-tips may then be carried out. It often enables one to feel the deeper structures better than one can do by simple pressure. In examining the lateral regions of the abdomen, bimanual palpation is often of service. The physician should sit or kneel by the bedside. One hand is placed posteriorly in the interspace between the last rib and the crest of the ilium. The other is placed over the abdominal wall in front. The posterior wall is then pushed up against the hand in front, so that any structure lying between the two hands can be distinctly felt. The secret of the method consists in keeping the front hand as still as possible. This procedure is of special value in the examination of the kidneys.

The first thing to notice in palpation of the abdomen is the degree of tension of the walls and of *resistance* experienced. Normally, the abdomen has an elastic or doughy feeling, only to be learnt by practice. In disease the resistance may be increased. It should be observed whether this increase is general or local. General peritonitis produces a great increase in the resistance from a reflex contraction of the muscles of the abdominal wall. Local increase in resistance is very frequently due to localized peritonitis, and is often of great diagnostic value. Palpation of the normal

abdomen is painless. If tenderness be elicited, its exact extent and point of maximum intensity should be noted. Anything in the nature of a tumour should be carefully felt for. In doing this, confusion is apt to be brought about by the recti. The thickening produced by parts of these may easily simulate a tumour. If this source of fallacy be suspected, try if the fingers can be got under the edge of the muscle, and feel if it thickens as the patient raises himself in bed.

If it be decided that a tumour is really present, one has first to determine whether it is situated inside the abdomen or in the abdominal wall. Try, therefore, to move the abdominal wall from side to side over the tumour. If the growth be intra-abdominal, this can usually be done without difficulty, unless it has contracted adhesions to the parietal peritoneum. Try also to grasp the tumour and to make the fingers meet, as it were, under it. This can usually be accomplished in the case of tumours situated wholly in the abdominal wall.

Supposing the tumour to be intra-abdominal, the first question to be settled is—Where is it growing from ? and, especially, is it coming up out of the pelvis, or is it truly abdominal ? To decide this the edge of the hand should be pushed back about an inch below the umbilicus, and in the direction of the prominence of the sacrum. One can then feel whether the tumour is passing down into the pelvis or not. The size and shape of the tumour should next be noted, and the nature of its surface—whether smooth or nodular. The presence or absence of fluctuation should then be investigated.

The *mobility* of a tumour is a very important point to determine. The directions in which it can be moved should be noted, and whether it is influenced

by respiration. The latter is a point of special value. Tumours connected with the liver and spleen move freely with respiration, and so may those of the stomach. Tumours of the kidney may be slightly movable. Those connected with the other abdominal organs do not move with respiration at all unless they have contracted adhesions.

In palpating the abdomen, the existence of *splashing* or *gurgling* at any points should be looked for. Splashing is often found over a dilated stomach, but is only of diagnostic value if it can be elicited some hours after the swallowing of food. Gurgling is produced by the passage of gas and fluid through constricted parts of the alimentary tract. It may thus be felt at the pylorus, especially if stenosed, or over strictures of the intestines.

Finally, the *umbilicus* should be examined. In malignant disease of the liver the umbilicus often becomes early infiltrated, and this sign has proved of great diagnostic value. The infiltration can often be recognized by its producing a "mooring" of the umbilicus—just as a scirrhus of the mamma does of the nipple.

In obscure cases of abdominal disease, palpation in the knee-elbow position, and under an anæsthetic, should never be omitted.*

Percussion of the abdomen.—This should be carried out in the same manner as will be described for the chest. In abdominal percussion the "flicking" method is extremely serviceable in detecting

* Examination of the patient in a hot bath has been recommended as of even greater value than the use of an anæsthetic in obscure abdominal cases, besides being safer. The patient gets into the bath at 100° F., and the temperature is rapidly raised by the addition of very hot water until 110° F. is reached. Complete relaxation of the abdominal wall is usually brought about at this temperature in five or ten minutes, but in some instances one may require to go up to 120° F. before the desired result is obtained.

slight degrees of dullness—e.g. in making out the lower edge of the liver. The forefinger of the left hand is placed firmly on the abdomen, the palmar aspect uppermost, and is sharply "flicked" with the middle finger of the right hand. Percussion of the normal abdomen yields a resonant note throughout, except in the regions of liver and splenic dullness, or over a full bladder. The percussion pitch of the hollow viscera depends on two chief factors—(*a*) the depth of the air space; (*b*) the tension of the containing wall.

As these two factors are of almost equal importance, and as each of them varies greatly in the same viscus at different times, the reader will readily understand that it is a mistake to dogmatize about the relative pitch of the note yielded by the various hollow viscera. The presence of free gas in the peritoneal cavity causes the normal liver and spleen dullness to disappear.

If any abnormal dullness be detected, the chief point to be determined regarding it is whether it is constant in position or shifts with alterations in the position of the patient. This will be more fully discussed when we come to speak of ascites.

Hydatid cysts yield on percussion a special kind of vibration called the "*hydatid thrill.*" To elicit it, three fingers should be placed over the cyst, and the middle one firmly percussed, the percussing finger being allowed to rest for a moment after each stroke. An "after-thrill" will then be experienced in the two adjacent fingers. It should be added that the sign is absent in about half the cases of hydatid cyst.

Auscultation of the abdomen.—Auscultation is not of much service in the examination of the abdomen. It is best to carry it out by means of a binaural stethoscope. In the region of the stomach

one may listen for swallowing sounds, bubbling and splashing (to be described later), or for abnormal conduction of heart sounds. Elsewhere one may hear friction sounds—from the presence of lymph on the surface of the peritoneum. One may listen over aneurysms to detect a bruit, and over suspected enlargement of the uterus for the presence of the uterine souffle or fœtal heart sounds. The latter are best heard (in normal presentations) at a point midway between the umbilicus and the left anterior superior spine.

Complete silence over the abdomen or any part of it, indicating cessation of intestinal peristalsis, is sometimes of value as a sign of peritonitis.

In cases of general abdominal swelling, **measurement** should never be omitted, as it affords a valuable index of the progress of the case. The circumference may be taken at the level of the umbilicus or at the point of maximum distension.

The examination of cases which are believed to have fluid in the peritoneal cavity, or **ascites,** calls for special consideration. In cases in which the fluid is sufficient to cause general distension, the conditions for which one is apt to mistake it are, as we have seen, fat in the abdomen and abdominal wall, gas in the intestines or free in the peritoneum, and new growths. Fluid gives, of course, a dull note on percussion. The dullness is not always absolute, however, owing to the transmitted resonance of subjacent bowel. When the fluid is free and not sufficient to fill the whole abdomen, its upper limit is more or less horizontal, but may show irregularities owing to the fluid running up into "bays" between coils of bowel.

Free fluid is also distinguished by the fact that it shifts its position with that of the patient. If he be turned over on his side and time given for the intestines

to float up, it will be found that the uppermost flank is now resonant, while the height of the dullness on the lower side has risen. If the fluid be very small in amount, it is a good plan to turn the patient on to his hands and knees. A dull area then appears around the umbilicus from accumulation of fluid there.

The "*transmitted thrill*" is another important physical sign of fluid in the peritoneum. It is elicited by placing one hand over the lumbar region of one side, the patient being on his back, whilst the opposite lumbar region is sharply tapped with the fingers of the other hand. A distinct impact will be felt to pass from one hand to the other. As a not dissimilar impulse is apt to be transmitted through the abdominal wall, especially if fat, it is always well to get an assistant to place the edge of his hand firmly in the middle line of the abdomen while percussion is being made. This damps down any vibrations transmitted by the wall. Where the amount of fluid is large, the vibrations are visible as well as palpable. On the whole we consider that the results of simple percussion afford the most trustworthy evidence of the presence of ascites.

Fat is to be distinguished by taking the abdominal wall between the hands and pinching it up. *Gas* is distinguished by the results of percussion. Of *new growths*, ovarian tumour is, perhaps, most liable to be mistaken for ascites. An ovarian tumour, however, causes an antero-posterior bulging of the abdomen, while in ascites the bulging is mainly lateral. In ovarian tumours, also, the dullness is central, and does not change with the position of the patient; in ascites the chief dullness is in the flanks, and it shifts, as we have seen, when the patient is moved. Lastly, in ascites the umbilicus is flat or bulges out, while in ovarian tumours it is drawn upwards.

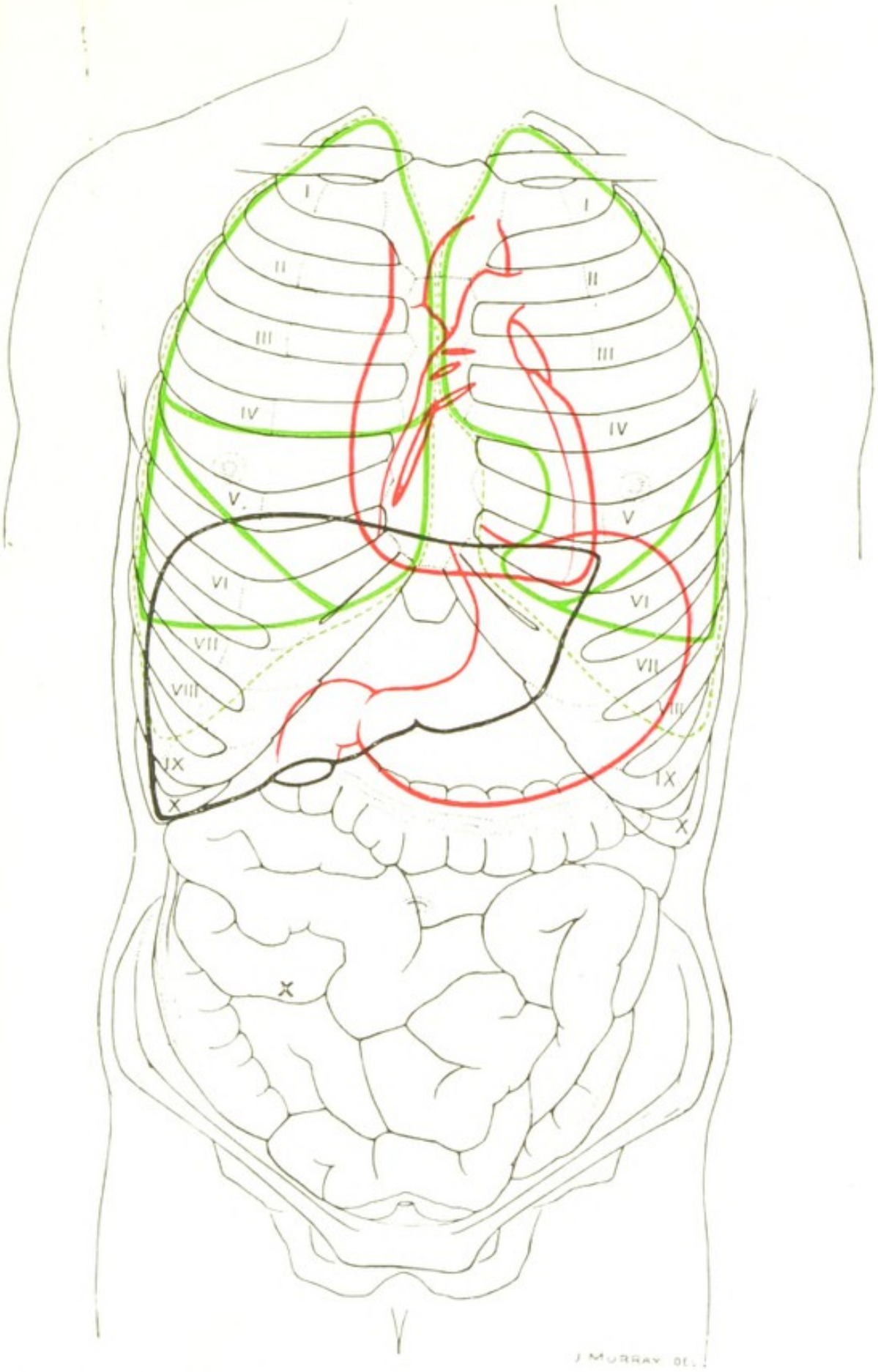


Plate 1.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE FRONT. Scale: 1 = 5·6.

III. THE ABDOMINAL VISCERA

One may now pass to the examination of the viscera in the abdomen, beginning with the stomach.

The Stomach

Special anatomy (Plates 1, 4).—The stomach is situated in the left hypochondriac and the epigastric regions. Its cardiac orifice lies behind the 7th left costal cartilage, 1 in. from the sternum and 4 in. from the surface. The pyloric orifice is surprisingly close to it, being about one full hand's breadth below the base of the xiphisternum and one finger's breadth to the right of the middle line. It passes considerably farther to the right, however, when the organ is distended. It is usually under cover of the liver. About two-thirds of the stomach is under cover of the ribs, the fundus reaching, in ordinary circumstances, as far up as the 5th rib in the mammary line. Hence it is somewhat behind and above the apex of the heart. Only a small part of the body of the stomach and of the pyloric region is in contact with the anterior abdominal wall. The exact position of the greater curvature varies. *Under normal conditions it should never be lower than the level of the umbilicus; usually it is considerably higher.*

It is difficult to determine the exact dimensions of the stomach, owing to its position, to the varying amount of gas it contains, and to the proximity of the transverse colon.

Recourse is frequently had to *inflation* of the stomach with gas in order to overcome some of these difficulties. The best method of inflating the stomach is to pass a stomach-tube provided with a glass mouth-piece. One distends the stomach either by blowing down the tube or by connecting it with a Higginson's

syringe or with a bicycle pump. One can clamp the tube when a sufficient degree of distension has been attained.

Inflation may also be carried out by causing carbonic acid gas to be produced in the stomach by the interaction of tartaric acid and bicarbonate of soda. In order to inflate the organ to its normal dimensions without over-distending it, 15 gr. of tartaric acid should be given in a cachet, followed immediately by 30 gr. of bicarbonate of soda in solution. If the stomach be dilated, however, three times these quantities or even more may be required.

A moderate degree of inflation can also be brought about by causing the patient to drink a tumblerful of soda-water. If the stomach be then percussed with the patient sitting up, the fundus will be found to be resonant from its distension with gas, whilst the lower limit of the greater curvature can be determined from the dullness caused by the water.

Inflation should never be practised if there has been any recent bleeding from the stomach or if one has reason to suspect the existence of a gastric ulcer. It should also be avoided in feeble subjects and in those in whom the heart is in any way embarrassed.

Inspection of the stomach region is included in the general examination of the abdomen (p. 56).

Palpation of the stomach. — Note any *tenderness*, and define its point of greatest intensity. Examine for *tumours*. The commonest of these is a pyloric new growth. Tumours of this region are characterized by their great mobility. They may be felt in, or pushed into, any region of the abdomen. Lastly, try for *splashing*. To make out this, sit at the left side of the patient with one hand over the left lower ribs behind; with the other placed over the front of the stomach, make short, sudden dipping

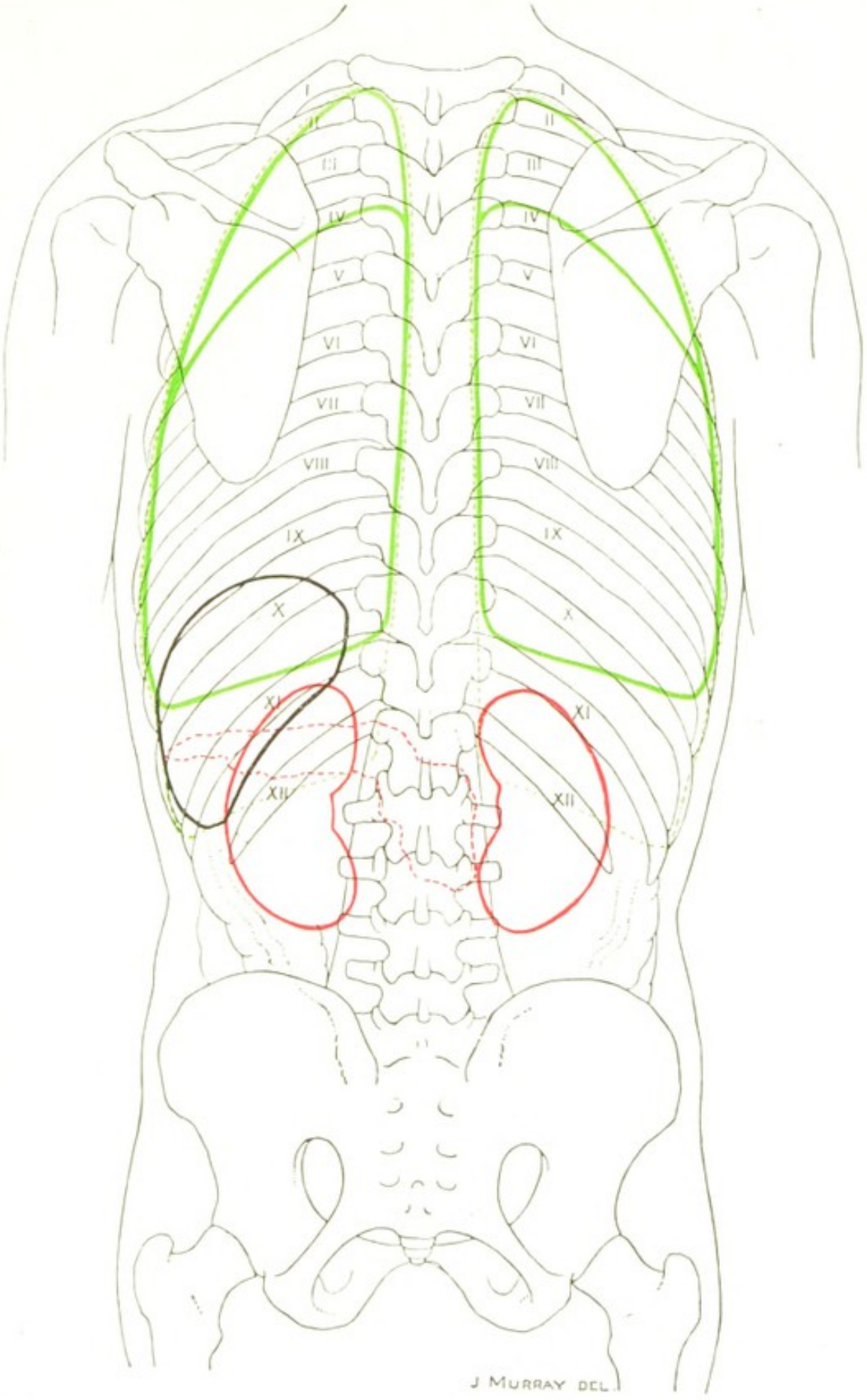


Plate 2.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM BEHIND. Scale : 1 = 5·6.

movements. If "splashing" be elicited it will be partly heard and partly felt.

Distinct splashing elicited three hours after a meal, especially if it can be made out below the level of the umbilicus, is very suggestive of a dilated stomach.*

Percussion of the stomach.—Three boundaries of the stomach can be made out by percussion: (1) between stomach and lung; (2) between stomach and liver; (3) between stomach and colon.

The last is that which it is most important to determine, and should be examined first. It is by no means always easy to define the lower border of the stomach exactly. This is because the transverse colon may give an almost identical note with it. Use light percussion. The "flicking" method succeeds very well here. Begin low down near the pubes, and percuss upwards just to the left of the middle line. The lower border of the stomach should be reached at least a finger's breadth above the umbilicus.

The limit between the lower edge of the lung and the upper part of the stomach is made out in a similar way. It is better to percuss from stomach to lung. The usual line of demarcation between the two runs in a slightly arched manner from the 6th costal cartilage in the parasternal line to the 9th rib in the midaxillary line. The area of stomach resonance which is bounded above by this line and by the anterior edge of the spleen, and below by the left costal margin, is called **Traube's space**. It covers that portion of the

* It should be remembered, however, that a splash may be elicited over even a normal stomach shortly after a meal containing much fluid, especially if the abdominal wall be thin; and care should be taken not to mistake a splash produced in the transverse colon for a stomach splash. The distinction between splashing in the stomach and colon may, in cases of difficulty, be rendered easier by auscultating over the upper part of the stomach when the splashing is elicited (cf. auscultatory percussion of stomach).

stomach which is in direct contact with the chest wall. We have seen that the fundus of the stomach extends above this under cover of the lung as high as the 5th rib in the nipple line. It cannot, however, be satisfactorily percussed out.

The demarcation between the stomach and liver is made out by percussing lightly from the stomach towards the liver margin. It is not of much importance in diseases of the stomach.

We would repeat that for the diagnosis of dilated stomach the position of the greater curvature is of most importance. If this be found to be below the umbilicus, while the lesser curvature is in its normal position, then the existence of dilatation is certain. It is confirmed if the stomach note extends much across the middle line towards the right.

The position of the boundaries between stomach and lung and stomach and liver depends as much upon the condition of the lung and liver as upon the stomach itself. Thus in fibroid contraction of the lung more of the stomach is exposed than is normal, and its area is therefore apparently increased. Cirrhosis of the liver may produce a similar apparent extension of stomach area. On the other hand, effusions into the pleura or enlargements of the liver cover up the stomach and cause a diminution in its area of resonance. Hence in the former condition Traube's space is much diminished (*see* p. 270). The possibility of a dislocation of the whole organ downwards must never be lost sight of. In such a case the outline of the lesser curvature may often be seen standing prominently out, especially when the organ is inflated. If the distance between the greater and lesser curvature is more than 10 cm., dilatation exists.

The **combined percussion-auscultation method** often gives valuable aid in mapping out the

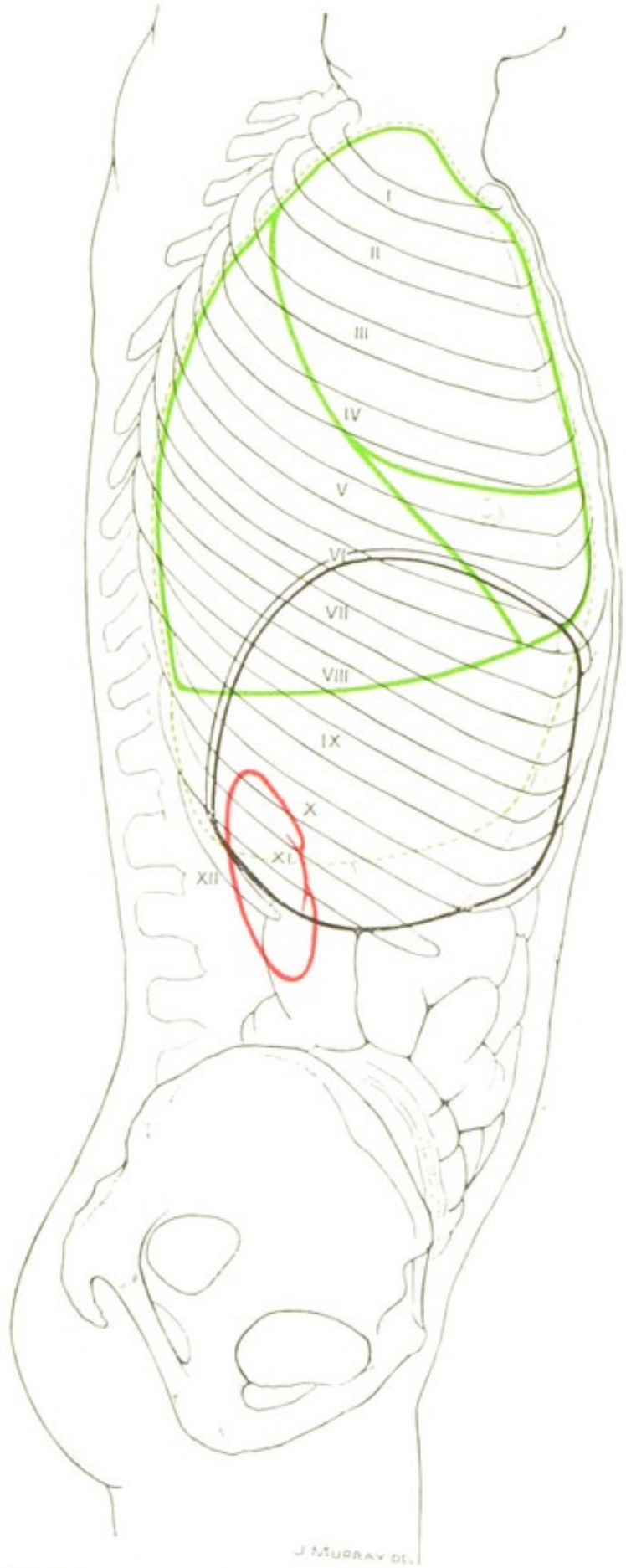


Plate 3.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE RIGHT SIDE. Scale : 1 = 5·6.

stomach.* It depends upon the fact that vibrations set up in the stomach, or any other gas-containing organ, resound all through it, and can be heard through a stethoscope placed over any part of it. To carry out this method proceed as follows: Place the end of a binaural stethoscope as nearly as possible over what one believes to be the mid-point of the stomach, i.e. a little below the xiphisternum. Then make *light* scratching movements on the skin along lines radiating from this point towards the borders of the stomach. So soon as the edge of the area of stomach in direct contact with the abdominal wall is passed, the characteristic note produced by the vibrations set up in the organ cease. If the characteristic stomach note be not obtained at first, the stethoscope should be moved downwards in the direction of the umbilicus. Previous inflation, though not essential, greatly facilitates the carrying out of the method.

Auscultation of the stomach area may detect peritoneal friction over it, or the crackling due to the bursting of fermentation bubbles in the interior. Heart sounds and murmurs are sometimes heard loudly over the stomach as over a resonating chamber. The deglutition sounds are of no diagnostic value.

The chemical investigation of the stomach is considered in Section IV. (p. 84).

The Liver

Special anatomy (Plates 1, 3).—The liver lies chiefly in the right hypochondrium. Its left lobe extends across the epigastric region, but does not pass more than 2 in. to the left of the sternum. Above, the liver reaches almost to the nipple; below, it extends to

* It is rarely possible to make out the actual size of the stomach by this method, but it delimits accurately the area in contact with the anterior abdominal wall, and from this valuable inferences can be drawn.

the costal margin. The lower border passes obliquely upwards from the 9th right to the 8th left costal cartilage, crossing the middle line somewhat above the mid-point between the base of the xiphoid and the umbilicus.

The gall-bladder is situated just internally to the 9th right costal cartilage, and immediately to the outer side of the right rectus muscle.

Inspection of the liver is of little value. Any visible swelling, fullness, or pulsation should be noted. The edge of the liver can sometimes be seen when the organ is enlarged. It forms a sharp line which moves up and down with respiration.

Palpation of the liver.—The lower edge should first be felt for. In order to do this, place the hand flat on the abdomen, with its edge towards the costal margin and just to the outer side of the rectus muscle, the reason for going so far out being to avoid the upper septum of the rectus sheath, which is apt to be mistaken for the lower edge of the liver. Then depress the edge of the hand slightly so as to push up a fold of skin, and ask the patient to take a long breath. If the edge of the liver be palpable, it will be felt to ride over the edge of the hand. Trial, of course, must be made at different levels before it is decided that the edge cannot be felt. The edge of the liver cannot, or can only very rarely, be felt in health. It moves down from two-fifths to three-fifths of an inch with inspiration. The character of the edge should also be noted —whether it is smooth or irregular, thickened or sharp. If in doubt whether what is felt be really the liver edge, feel for the fissure of the gall-bladder, and, towards the middle line, for that produced by the round ligament.

The **surface of the liver** in the epigastrium should then be felt in the usual way. Any tenderness should be noted, and whether it is localized or general.

The character of the surface should be made out. Is it smooth, as in waxy disease, or nodular, as in carcinoma? In the latter condition the centres of the nodules will often be found to be umbilicated. Care must be taken not to confound little irregularities which are frequently present in the upper parts of the recti with irregularities on the surface of the liver. Liver friction (due to perihepatitis) can sometimes be felt. It is usually best made out over the posterior surface of the organ between the vertebræ and the midaxillary line.

Heaving pulsation of the whole organ can best be appreciated by placing one hand over the lower right ribs behind and the other over the organ in front.

Percussion of the liver.—The following table shows the normal percussion limits of the liver (Fig. 21, p. 136):—

		Middle line	*Mammary line*	*Midaxillary line*	*Scapular line*
Upper limit.	Deep dullness.	Blend with heart dullness.	4th space.	7th space.	9th space.
	Superficial dullness.		6th rib.	8th rib.	10th rib.
Lower limit.		Hand's breadth below base of xiphoid.	Costal margin or somewhat above or below it.	10th space.	Blends with kidney dullness.

Procedure.—The patient should be lying down for percussion of the anterior and lateral aspects; sitting up or standing for the posterior aspect.

To make out the deep dullness, use heavy percussion—two fingers, if necessary. Begin high up—say about the 2nd rib—so as to get a good lung note, and percuss down from rib to rib till impairment is

detected. Then repeat the process, going from space to space instead of from rib to rib. Percuss in this way down the mammary, midaxillary, and scapular lines.

The upper limit of liver dullness in the middle line cannot be distinguished from the heart dullness. To map it out, draw a straight line from the apex beat to the angle where the right edge of the heart and the deep liver dullness meet. To make out the upper limit of superficial dullness, percuss lightly down the same lines. The upper limit of liver dullness forms an almost horizontal line around the chest.

In defining the lower edge of the liver, use very light percussion, and pass upwards. The "flicking" method does well. Another good plan is to percuss with three fingers of the right hand held in a row. Very slight degrees of dullness can often be more easily detected by this device.

The exact position of the lower edge of the liver is extremely variable. Usually it coincides with the costal margin in the mammary line. It may be considerably above or below this, however, without there being any pathological change in the organ. Its position in the middle line is also very variable. As a rule, it is situated about a hand's breadth below the base of the xiphoid.

In percussing the surface of the liver where it is not covered by lung, it should be observed that the organ has a certain degree of *resistance* or *resilience*. The normal amount of this can only be learnt by practice. If the organ be enlarged or congested, its resistance to percussion is increased owing to its being more firmly pressed against the chest wall.

The liver may be displaced, enlarged, or diminished.

Displacement may be either upwards or downwards. Upward displacement may occur from tumours, etc., in the abdomen pushing the liver up.

Downward displacement may be brought about by dilatation of the right ventricle of the heart, right pleural effusion, or emphysema of the lungs, or, more rarely, by new growths below the diaphragm. When the liver is dislocated downwards, the rounded upper surface of the left, and part of the right, lobe can usually be made out crossing the epigastrium. A displaced liver also does not move freely with respiration, while a liver which is merely enlarged does.

One must distinguish between real enlargements and diminutions of the liver and those which are apparent only.

Thus, *enlargement* of the liver may be simulated by consolidation of the base of the right lung, or by effusion into the right pleura. Downward enlargement may be simulated by accumulation of fæces in the transverse colon.

An hydatid cyst in the liver often produces an enlargement of the organ upwards rather than downwards.

Diminution of the liver may be simulated by the organ being covered up by an emphysematous lung, or by the colon passing up between it and the abdominal wall. The latter is a rare condition. It should be suspected if the lower limit of liver dullness varies very much at different points.

The **gall-bladder** is examined by palpation and percussion. It cannot be *felt* unless distended. It may then form a smooth, pear-shaped swelling, situated just to the outer edge of the right rectus muscle. It can be moved freely from side to side round a point opposite to the 9th costal cartilage. It also moves with respiration. Sometimes gall-stones can be felt in it. If there be many of these they produce on palpation a sensation resembling that produced on feeling a bag of nuts.

On *percussion*, a distended gall-bladder forms a dull area, projecting from the liver dullness towards the umbilicus, but usually continuous with it. Sometimes, however, the transverse colon comes to lie across the neck of the gall-bladder, so as to separate it from the liver. When this occurs, diagnosis of the tumour is apt to give trouble. To this point we shall recur when we come to the examination of the right kidney (p. 77).

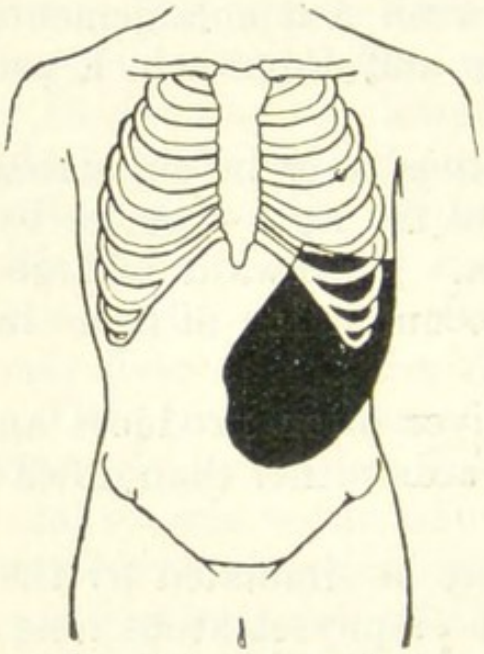


Fig. 6.—Enlargement of spleen.

The Spleen

Special anatomy (Plates 2, 4).—The spleen lies in the left hypochondrium. It is bounded above by lung, elsewhere by stomach and intestine. Its lower end rests upon the costo-colic fold of peritoneum. It lies along the 9th, 10th, and 11th ribs, being partially separated from them by the diaphragm and lower edge of the left lung. Its upper end is opposite the 9th dorsal spine, and reaches to about $1\frac{1}{2}$ in. from the middle line. Its lower end comes as far forward as the midaxillary line.

Inspection of the spleen.—If much enlarged, the spleen may form a visible tumour in the left side of the abdomen, which moves with respiration (Fig. 6).

Palpation of the spleen.—This is really the most important method of investigating the spleen. If one can exclude dislocation, then a spleen which is palpable may safely be pronounced to be enlarged; and it is never safe to diagnose enlargement of the spleen unless it *is* palpable.

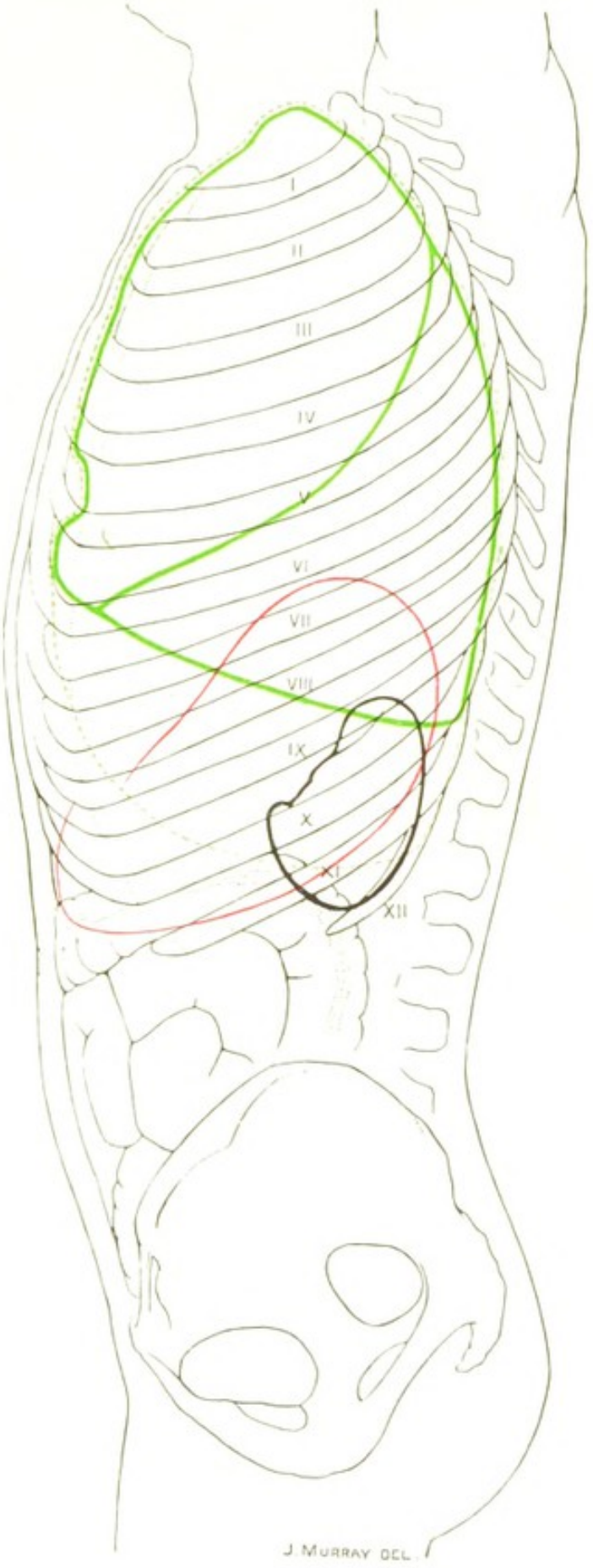


Plate 4.—VISCERA OF THORAX AND ABDOMEN, AS SEEN FROM THE LEFT SIDE. Scale : 1 = 5·6.

There are two methods of feeling for the spleen :

1. Stand or sit at the left side of the patient as he lies on his back. Keep the hand flat on the abdomen, and depress the fingers a little, so as to push up a fold of skin near the left costal margin opposite the 10th cartilage, and get the patient to take a long breath. The edge of the enlarged spleen will be felt to come up against the finger-tips, and to ride over them, as it were.

2. Go to the right side of the patient. Place the fingers of one hand behind in the space between the ends of the 10th and 11th ribs. Place the other hand over the left hypochondrium, with the fingers slightly tucked in under the edge of the costal arch. With the posterior hand tilt the spleen forwards while the patient inspires. The edge of the organ will then be felt against the fingers of the other hand.

The edge of the spleen is sharp and usually quite smooth. Notches can often be felt in it, but by no means invariably. It is important to note (1) that the anterior border of an enlarged spleen is always directed downwards and inwards, and (2) that there is always a slight space between the posterior edge of the spleen and the erector spinæ, into which the fingers can be dipped. Occasionally the spleen enlarges upwards only. This may happen where the costo-colic fold is abnormally well developed, and keeps the organ up. For the detection of such a condition one must have recourse to percussion.

Sometimes, on the other hand, when the spleen gets very large it pushes down the costo-colic fold and then drops down itself. A spleen which at one time crossed the middle line may cease to do so by dropping down in this way, though the organ is really larger than ever.

Percussion of the spleen.—The anterior part of the spleen can be defined whilst the patient is lying on his back. For the posterior part one of two positions

is advisable—he may either sit up, with his left hand supported on the top of his head, or he may be semi-prone, resting chiefly on the right scapula, with the left arm behind the head. He should not be altogether on his right side, else the spleen falls away too much from the surface of the body. The organ is most sharply defined when percussed with the chest either in full inspiration or full expiration. It is least clearly made out when the chest is in an intermediate position.

The limits of the normal spleen cannot always be defined by percussion. It may be borne away so much from the surface, owing to extreme arching of the diaphragm, or it may be so covered up by lung, that it is impossible to be sure of its exact limits.

Procedure.—Define the *anterior* edge by percussing lightly along the 10th rib, beginning near the costal edge. The splenic dullness (absolute) should be reached at the midaxillary line.

The *lower edge* is defined by percussing lightly upwards along the posterior axillary line or slightly behind it. The lower edge of the spleen should be reached about the lower border of the 11th rib.

To make out the *upper* and *posterior* borders heavier percussion is required. Percuss vertically downwards about midway between the posterior axillary and scapular lines, beginning at about the level of the angle of the scapula. The lung note will become impaired at the upper edge of the 9th rib, indicating that the upper limit of the spleen has been reached.

The *posterior border* is defined by percussing along the 10th rib, beginning near the middle line. The splenic dullness is reached at about 1½ in. from the vertebral spines. This border is not always easy to make out.

By joining together the different points defined

above, an oval area will be mapped out, which measures about 3 in. in its long diameter and 2 in. transversely.

Extension of splenic dullness may be *simulated* by effusions into the left pleura, or consolidation of the base of the left lung; by the presence of fluid in the stomach, or of fæcal accumulation in the colon.

Auscultation over the spleen may be practised to detect the existence of friction. The latter occurs in perisplenitis and over the surface of splenic infarcts.

The Kidneys

Special anatomy (Plates 2, 3).—Each kidney lies partly in the epigastric, partly in the hypochondriac region. The right kidney lies partly in the lumbar region as well. As regards their relation to the anterior abdominal wall, the kidneys are higher up than one is apt to suppose. The lower end of the right kidney is fully 1 in. above the umbilicus, the left is about ½ in. higher. The lower end of each is about 3 in. from the middle line.

As regards their posterior relations, about one-third of each kidney lies above the last rib. The upper end of the right kidney is at the level of the 11th dorsal spine, whilst its lower end reaches to about 1 in. above the iliac crest. The left kidney is about ½ in. higher.

Palpation of the kidney. Procedure.—The patient must be on his back with the knees slightly flexed, and the shoulders raised on a firm pillow. The lumbar region must be quite flat on the couch, not arched forward. Sit or kneel beside the patient on the side to be examined. Place one hand upon and below the last rib behind, the other immediately below the costal margin in front. It is best to have the right hand in front when examining the right kidney,

and the left hand in front when examining the left. The posterior hand should press the loin forwards, while the other hand pushes the anterior abdominal wall backwards, upwards, and outwards. The kidney will then be felt between the two hands if it is at all enlarged or displaced. Even in health (provided the patient be not too fat) the lower part of the organ can often be felt.

Should this manipulation not succeed, the patient must be asked to take a long sighing inspiration. If the front hand follows up the receding abdominal wall as the air leaves the chest, the kidney will be caught between the two hands.

The kidney moves very slightly with respiration. An exaggeration of this normal mobility, so that the organ slips up and down like a pea in a pod, constitutes *movable kidney*. This must be distinguished from *floating kidney*, in which the organ moves freely forward so as to " float " towards the anterior abdominal wall, as well as moving vertically and laterally.

A floating right kidney is very apt to be mistaken for a distended gall-bladder, and vice versa. The shape, size, and consistence of the tumour may be apparently identical in the two cases. One point of distinction is that while a distended gall-bladder can be temporarily pushed back from the abdominal wall, yet it always tends to spring forward again; it is therefore always in evidence. It is not so with a floating kidney; the latter often disappears for a time, and can only with difficulty be got hold of again. Another point of distinction is that a kidney can be pushed down towards the pelvis and held there even during forcible expiration, whilst the gall-bladder moves upwards again during the expiratory act. The different relation of the colon to the kidney and to the gall-bladder should also be remembered.

An enlarged left kidney may be mistaken for the spleen. The points of distinction are: (1) The spleen has a sharp edge. The edge of the kidney is *always* rounded. The existence of a sharp edge in any abdominal tumour excludes the kidney at once. (2) There is no space between the posterior border of the kidney and the erector spinæ, as there is in the case of the spleen; and (3) the colon lies between the kidney and the anterior abdominal wall, but not over the spleen (Fig. 7).*

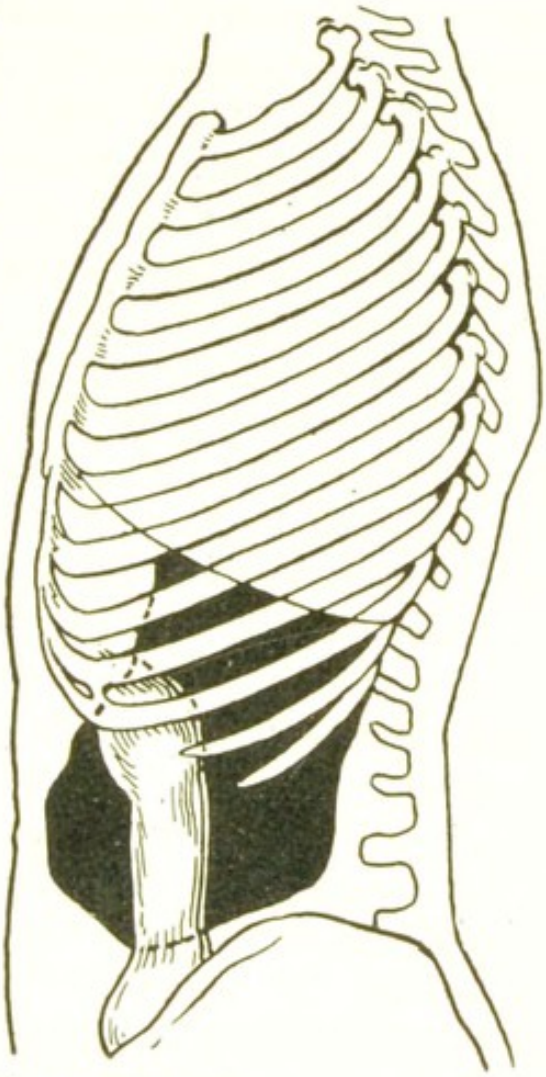


Fig. 7.—Showing colon crossing a tumour of the kidney. (*After Sahli.*)

It is impossible to determine the size of the kidney by means of percussion.

An enlarged kidney tends to bulge forwards. Perinephric abscesses, etc., bulge backwards.

The Intestines

Special anatomy.—The small intestine occupies chiefly the umbilical and hypogastric regions; the large intestine, the peripheral zone of the abdomen. The ileum joins the colon at a point 2 in. internal to, and somewhat above, the right anterior superior iliac

* If one is in doubt whether the colon lies in front of the tumour or not, it is often a help to inflate the large bowel by means of a rectal tube connected with a Higginson's syringe or bicycle pump.

spine. The apex of the cæcum corresponds to a point a little to the inner side of the middle of Poupart's ligament. The base of the vermiform appendix usually lies opposite a point $1\frac{1}{2}$ to 2 in. from the anterior superior spine along a line drawn from that spine to the umbilicus. This is sometimes called McBurney's point, because he has shown that in the majority of cases of appendicitis that is the point of maximum tenderness as determined by the pressure of one finger.

The splenic flexure of the colon lies behind the stomach, the hepatic lies under cover of the liver. The former is at a somewhat higher level than the latter. The transverse colon passes across the abdomen in a slightly curved direction, the lower part of the curve reaching to about the umbilicus.

Examination of the intestines by **inspection** and **palpation** has been described earlier in the present chapter under the general examination of the abdomen (p. 56).

Percussion of the intestines. — The notes yielded by the small and the large intestine cannot be satisfactorily discriminated. The combined percussion-auscultation method can be used to map out the colon in the same way as for the stomach (p. 68). One should place the stethoscope near the splenic or hepatic flexure, and percuss close to it. Then one may begin at what is presumably beyond the periphery of the gut, and continue to percuss towards the stethoscope until one recognizes the characteristic note.

Rectal examination.—Place the patient in a good light and in a semi-prone position—i.e. resting on the left breast with the right thigh and knee well drawn up, the inner aspect of the right knee resting on the couch. Draw aside the glutei and inspect the region of the anus, noting the presence of any eruption, of

external hæmorrhoids, etc. Smear the right forefinger with vaseline and fill the nail with soap. Pass the finger slowly and gently through the anus, directing it slightly forwards at first. Note the degree of resistance offered by the sphincter; this shows whether the latter is normal, spasmodic, or relaxed.

Once the anal canal is passed, direct the finger slightly backwards and upwards, asking the patient to bear down a little at the same time. The finger can then be swept round and the whole inner surface of the rectum explored.

Two folds of mucous membrane will be encountered (Houston's folds), one opposite the prostate in front, the other higher up, near the middle of the sacrum, and passing in from the left side. The prostate will be felt projecting into the rectum in the male, and above it is the trigone of the bladder flanked by the seminal vesicles; below it is the membranous urethra. In the female the cervix uteri will be felt projecting back in the form of a hard knob. The mucous membrane must be examined for polypi, ulcers, etc.

It must be remembered that hæmorrhoids are not palpable. The presence of scybala or foreign bodies can be determined, and the existence of any tumour, either in the bowel or pressing upon it, can be made out. If the lymphatic glands which lie in the hollow of the sacrum are enlarged, they can be felt.

IV. INVESTIGATION OF THE GASTRIC FUNCTIONS

The object of the investigations now to be described is to test the digestive and motor power of the stomach.

The **procedure** is as follows: The patient has a light supper on going to bed, and the tube is passed (p. 51)

the first thing next morning. The contents of the fasting stomach thus obtained furnish useful information. The presence of *food* indicates stagnation of the contents; if there be over 5 oz. of *clear gastric juice* without food, hypersecretion exists; if on washing out the stomach flakes of *mucus* are obtained which sink in the wash-water, one can diagnose gastritis.

Test Breakfast

The next step is to give a test breakfast consisting of two slices of toast and two cups of weak tea. It is withdrawn an hour afterwards.

Method of withdrawal.—Pass the stomach-tube as described at p. 51, but have it connected with a glass or vulcanite funnel. The patient should sit on a chair placed on a square of waterproof sheeting. He should be wrapped in a mackintosh and sit with his legs apart, so that anything spilt on the mackintosh may run into a pail placed between them.

When the tube has reached the stomach,* ask the patient to retch or cough, the end of the funnel being depressed at the same time. In this way a sample of the contents can usually be obtained and caught in a clean vessel. Should this fail, press over the epigastrium while the patient leans forward, and try to drive the stomach contents up into the tube. Should the contents still fail to come away, the following procedure, as recommended by Gillespie, may be tried: Fill the funnel with warm water, pinching the india-rubber tube a short distance below with the left hand, so that no air can get down; then, relaxing the pressure, let the water fill the tube to some way beyond the piece of glass inserted in the middle of it. When just enough water has been allowed to pass down to fill the

* The best flow is usually obtained when the tube has passed about 22 in. from the teeth.

tube, lower the funnel, let the water run into the pail, and the contents of the stomach are sucked up by the siphon-action thus induced, and may be caught, when they appear after the water, in a clean basin. If it is done carefully, little or no water enters the stomach, and hence the contents are not diluted.

If one still fails, one may be obliged to aspirate the contents by means of a rubber ball syringe (such as is used for giving an enema) or a partially exhausted aspirating bottle. As far as possible, however, any method of aspiration should be avoided, as none is free from some risk.

When the stomach contents have been obtained they should be allowed to settle in a tall jar, after which one can proceed to examine them. If necessary, they may be decolorized by shaking them up with animal charcoal.

The points that have to be investigated in the product are as follows :—

1. Physical characters.
2. Acidity.
3. Enzyme content.*
4. Microscopical characters.

1. **Physical characters.**—A very small result from the test meal, containing little fluid and imperfectly dissolved masses of food, indicates defective secretion or hypermotility. An abundant and very liquid yield points to excessive secretion or diminished motility, or both. When the contents consist of a large quantity of greenish fluid with a deposit of starchy material at the bottom, hypersecretion is present. A froth on the top of the fluid, with a yeasty odour, indicates fermentation from pyloric obstruction. A sour, acrid smell points to the presence of

* This need only be investigated if free HCl be absent and the total acidity less than 20.

organic acids. If the contents are viscid and filter slowly, mucus is present in excess (gastritis).

The product of the test meal should now be filtered through a folded filter-paper, and the investigation of the filtrate proceeded with.

2. **Acidity.** (*a*) **Are the contents acid?**—Test with litmus.

(*b*) **Is free HCl present?** — *Congo-red paper* (Appendix, 5) is turned blue. An approximate idea of the amount of acid present may be obtained from the depth of tint produced. Normal gastric juice turns the paper a sky-blue.

Günzburg's test for free HCl.—Place 10 drops of the stomach contents in a porcelain capsule, add an equal quantity of the phloroglucin and vanillin solution * (Appendix, 5). Heat gently, taking care to avoid charring. When almost dry, complete the evaporation by blowing on the surface of the fluid. If free hydrochloric acid be present a pink colour appears, usually at the periphery of the dried fluid.

The reaction is only given by *free* hydrochloric acid. The combined acid and organic acids do not yield it.

Boas's resorcin reagent (Appendix, 6) may be used similarly. It gives a purplish colour. This method is less sensitive but much more economical than that of Günzburg.

Dimethyl-amino-azo-benzol (methyl-orange) gives a red reaction with free hydrochloric acid, but is not such a delicate test as either of the above. It is best used in the form of test papers soaked in a 0·5 per cent. alcoholic solution of the dye.

(*c*) **Are organic acids present?**—These need only be tested for if free HCl is absent, or if there are indications of stagnation in the contents.

* The solution should be freshly prepared.

Tests for organic acids.—These must first be dissolved out by means of ether. Shake up 10 c.c. of the fluid with 50 c.c. of ether very thoroughly in a separation funnel or tall cylinder. Pour off the ether. Divide the ethereal extract into two equal portions, and place these in wide beakers. Set one in hot water; allow the other to evaporate slowly at the temperature of the room. In the former all the ether will soon have disappeared. Dissolve the residue in about 5 c.c. of water. Take some of Uffelmann's reagent (Appendix, 7) in a test tube, and add to it a few drops of the watery solution. If the blue solution changes to yellow, *lactic acid* is present.

The residue of the second beaker is used to test for acetic and butyric acids. These, being volatile, would be driven off unless the ether had been evaporated at a low temperature. Dissolve the residue in a little water. Neutralize part with a little carbonate of soda, and add to it some *very dilute* neutral perchloride of iron solution. A claret-red colour indicates the presence of *acetic acid*. To the other part of the solution of the residue add a small fragment of calcium chloride. If oily drops appear on the surface, it follows that *butyric acid* is present.

Acetic acid can also be recognized by the odour of vinegar; butyric acid by its characteristically rancid smell.

(*d*) **Total acidity.** Take 10 c.c. of the filtered gastric contents (in a beaker), add a few drops of dimethyl-amino-azo-benzol,* and cautiously run in decinormal caustic soda solution until the pink colour is discharged. Read the burette (first reading). Now add a few drops of an alcoholic solution of phenol-phthalein. Again run in the soda solution, this time until the least trace of persistent pink colour appears

* Instead of adding the indicator, the papers of this indicator or of Congo red may be conveniently used. In this case a drop of the beaker contents is removed on a glass rod from time to time during the filtration until the free acid is shown to be neutralized

in the beaker. This can best be appreciated by holding against a white surface. Read the burette (second reading). The first reading gives the amount of the "free acidity"; the second reading shows both the "total" and the "combined" acidity,* the "total" by the number of cubic centimetres run in from the beginning of the titration, the "combined" by the number added after the first reading was made.

Should there be no free acid, the "deficit" may be determined, i.e. $\frac{N}{10}$HCl is added to 10 c.c. of the filtered contents until free acid is present. Then, after adding phenolphthalein, the acidity is determined with $\frac{N}{10}$NaHO. In this case the "total" acidity is obviously the number of cubic centimetres of alkali used minus the number of cubic centimetres of $\frac{N}{10}$ acid previously added.

The results of these titrations may be stated in one of two ways: (1) Directly, from the number of cubic centimetres of $\frac{N}{10}$NaHO required to neutralize 100 c.c. of the stomach contents; e.g. if to neutralize the 10 c.c. of contents 5 c.c. of $\frac{N}{10}$NaHO were necessary, then for 100 c.c. of contents 50 c.c. of soda would be required, and the "total" acidity would be 50. If of this 5 c.c. of soda 3 c.c. were required to neutralize the free acid and 2 c.c. to neutralize the combined acid, the "free" and "combined" acidities would be respectively 30 and 20. Normally the total acidity varies from 40 to 60.

(2) Indirectly, in terms of HCl. Thus, one litre of decinormal soda is required to neutralize 3·65 grm. of HCl. If, therefore, 100 c.c. of stomach contents require 50 c.c. of soda to neutralize them, then the acidity of the 100 c.c. is equal to that of 0·18 grm. HCl—that is to say, the acidity is 0·18 per cent. The normal total acidity in terms of HCl is about 0·2 per cent.†

* The term "combined," as used here, includes acid combined with proteins, with enzymes, and with mineral bases in the form of acid salts. Acidity due to free organic acids will also be included in the second reading of the burette.

† The necessity for calculation may be avoided if one remembers that, provided 10 c.c. of contents have been taken and that decinormal soda is used for titration, the number of c.c. of soda required × 0·365 = HCl per 1,000 parts. In order to get the per-

3. **The enzyme content.***—The presence of albumoses proves the presence of pepsin in the gastric juice. In the absence of albumoses, **pepsin** can be tested for by its digestive action on egg albumin. Strips of hard-boiled white of egg are cut with a double-bladed knife, and discs punched out by a cork borer. These are preserved in glycerine till required. To each of four test tubes 5 c.c. of filtered gastric contents and a disc of egg-white are added. The first tube receives no further addition ; to the second add 2 drops of dilute hydrochloric acid (B.P.) ; to the third, 3 gr. of pure pepsin ; and to the fourth, both acid and pepsin. Keep all the tubes about body temperature. The rate at which the disc dissolves in the different tubes will show which ingredient, if any, is deficient in the gastric juice.

Many authorities, holding that the pepsin and rennin enzymes run parallel in their secretion, now estimate rennin instead of pepsin, and thus judge of the amount of peptic enzyme present. **Rennin** may easily be tested for by adding 2 c.c. of the gastric contents (neutralized and filtered) to 10 c.c. of raw milk and keeping the mixture about the body temperature for a quarter of an hour. If the milk sets into a solid mass the ferment is present. For purposes of **quantitative estimation** take six test tubes, each containing 5 c.c. of milk. Take 1 c.c. of contents and add 9 c.c. of distilled water. To tube 1 add 5 c.c. of this $\frac{1}{10}$ diluted contents and $2\frac{1}{2}$ c.c. of 1 per cent. $CaCl_2$. Now dilute the remaining 5 c.c. of contents to 10 c.c., and add 5 c.c. of this $\frac{1}{20}$ diluted contents with $2\frac{1}{2}$ c.c. of $CaCl_2$. Repeat the dilution, and add respectively

centage of HCl, one has merely to shift the decimal point one place to the left. For example, if 10 c.c. of decinormal soda solution were required to neutralize 10 c.c. of gastric contents, the amount of HCl present is 3·65 per 1,000 or 0·365 per cent.

* *See* foot-note, p. 83.

5 c.c. of 40, 80, 160 times diluted contents in each case with 2½ c.c. of $CaCl_2$. Place for one hour at 37° C., and from the amount of coagulation the amount of enzyme can be estimated. Normal juice gives a "cheesy" coagulation with $\frac{1}{80}$, a "flake" coagulation with $\frac{1}{160}$ dilution.

4. **Microscopic characters.**—Films are made in the usual way and stained with dilute gentian violet. Long bacilli (Oppler-Boas bacilli) are believed to be indicative of carcinoma; yeasts and sarcinæ (Fig. 8) are common in benign stenosis of the pylorus.

Product of test breakfast in health.—If a test breakfast be given to a healthy person and the contents removed one hour afterwards, it will be found that 20 c.c. to 40 c.c. of fluid are obtained. This is transparent, straw-coloured, of an acidity equal to that of 0·2 per cent. or so HCl (40–70, Ewald scale); it contains free hydrochloric but no organic acid.

In some functional disorders of the stomach the total acidity of the contents is increased, rising above that of 0·2 per cent. HCl. Sometimes no free HCl is found. In cases of malignant disease of the stomach the absence of HCl is of sufficient constancy to be of diagnostic value. The presence of organic acids is an indication of the existence of abnormal fermentative processes in the stomach.

Determination of gastric motility.—To estimate the motility of the stomach a **test dinner** should be given. This may consist of a plate of soup, 5 oz. of meat, a roll, a little mashed potato, some stewed fruit, and a glass of water. Seven hours after such a dinner the stomach should be found empty. If food remnants are present in any quantity (4 oz. or upwards), *defective motility* (atonia) is present. In order to test for complete *stagnation* of the contents

the fasting stomach should be washed out in the morning after a light supper the night before, as already mentioned (p. 82).

The Desmoid reaction (Sahli).—The gastric function may be investigated by this means without using the stomach-tube. A methylene blue pill (0·05 grm. mixed with ext. glycyrrhizæ) is twisted inside a bag of thin dental rubber (cofferdam) and tied with fine 0 0 *raw* catgut which has been rendered soft by soaking in water. A bag thus made should be airtight and sink immediately when placed in water. The principle of the reaction is that the gastric juice alone can digest catgut, it being quite indigestible in pancreatic juice. The contents of the bag will therefore only be liberated in the stomach. After ingestion, generally with the midday meal, the urine is examined, in five, seven, nine to twenty hours, for methylene blue. If this appears inside twenty hours a satisfactory gastric function is indicated.

A somewhat similar test may also be used to test pancreatic sufficiency. In this a capsule of gelatin hardened with formalin (glucoid) containing 0·1 to 0·15 grm. of iodoform is given. Such a capsule is not attacked by the gastric juice, but is readily digested by the pancreatic juice. Iodine is then tested for, better in the urine than in the saliva, after two, three, four to six hours from taking (preferably with a morning test meal). Normally it takes from four to five and a half hours. Salol may also be used and tested for in the urine by ferric chloride (*see* p. 370). In pancreatic insufficiency the reaction is negative or very much delayed. In diarrhœa with digestion unimpaired the reaction is earlier; in those with impaired digestion and absorption it is delayed.

V. EXAMINATION OF VOMIT

1. **Naked-eye characters.**—The general character of the vomit varies greatly, of course, with the nature of the food which has been taken. In *dilatation of the stomach* the vomit is apt to be very copious, sour-smelling, and after standing exhibits a froth on the surface. *Bilious vomit* is yellow or green in colour; *fæcal vomit* presents a very similar appearance, but is distinguished by its fæcal odour and by its neutral or alkaline reaction. The presence of much *mucus* gives to the

vomit a viscid consistence. The appearance of the vomit in hæmatemesis varies. If the bleeding be very copious, the vomit may present the appearance of pure blood and may contain clots. Such bleeding may proceed from a gastric ulcer or from varicose œsophageal veins. More commonly the blood is altered in colour by being retained for some time in contact with the gastric juice; thus it may be blackish in colour, or dark brown. The latter appearance is due to the conversion of hæmoglobin into hæmatin. The altered blood gives to the vomit an appearance often compared to that of *coffee grounds* or hare soup. It should be borne in mind that the taking of preparations of iron or red wines may produce a very similar appearance in the vomit. Vomit which contains dark-green bile may resemble very closely vomit which contains blood. On diluting with water, however, the green colour of the bile becomes more apparent, while blood remains dark.

2. **Chemical examination.**—The vomit should be filtered through fine muslin. The filtrate can then be examined, if desired, in the manner already described for the stomach contents.

Bile can be detected by Gmelin's test (p. 366). For the chemical detection of **blood** in the vomit the guaiac test is not satisfactory. It is better to take up some of the brown deposit with a pipette, place it in a porcelain capsule, and add a pinch of powdered chlorate of potash and a few drops of strong hydrochloric acid. Heat till dissolved. Cool and add a few drops of ferrocyanide of potash solution. A blue colour indicates that blood is present. The reaction is due to the iron contained in the blood pigment. If the patient has been taking iron the test is, of course, inapplicable. In such a case some of the deposit should be digested with caustic potash, filtered, and the solution exam-

ined for the spectrum of alkaline hæmatin, or the deposit may be subjected to Teichmann's test (Appendix, 22). To confirm the test, add to the alkaline hæmatin solution a few drops of sulphide of ammonium, which converts it into hæmochromogen. The spectrum of the latter is identified by its possessing two bands—one, narrow and dark, in the yellow between D and E; the other, broader and less dark, at

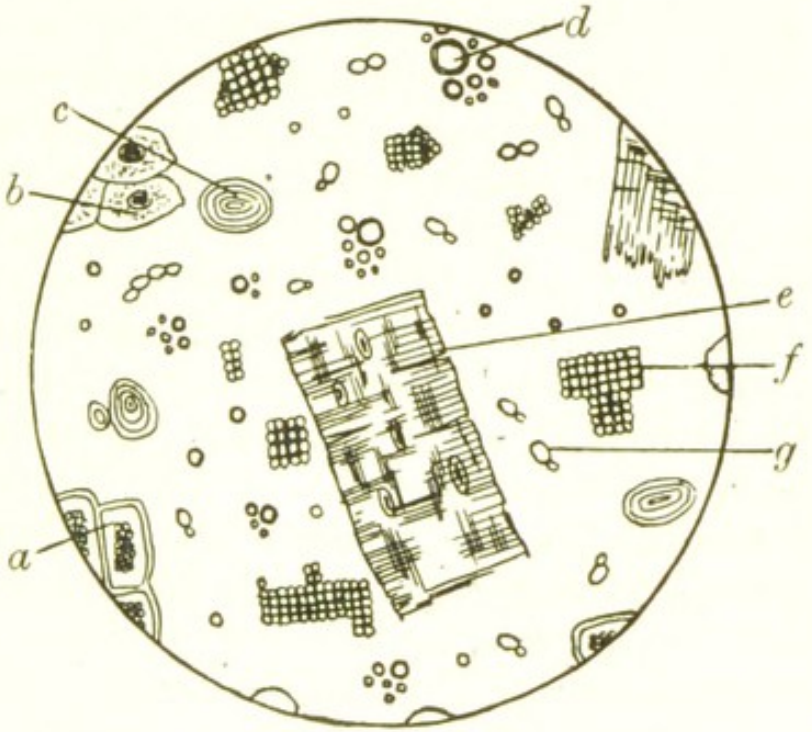


Fig. 8.—Microscopical view of vomited matter.

a, vegetable cell; *b*, epithelial cells; *c*, starch granule; *d*, oil globule; *e*, muscle fibre; *f*, sarcina ventriculi; *g*, torula.

the junction of the yellow and green between the lines E and *b* (Fig. 70, p. 240).

3. **Microscopical examination** (Fig. 8).—Take up some of the deposit which adheres to the muslin, spread it out on a slide, and examine either directly or in a drop of salt solution.

Various particles derived from the food may be recognized—*muscle fibres* by their transverse striæ; *starch granules* by their concentric lines and the fact that a drop of very dilute iodine solution turns them

blue; *elastic fibres* by their double contour and bold curves; *fatty particles* by their high refractility.

Various **vegetable parasites** may be present. The most important are the **sarcina ventriculi** (a large micrococcus) and the **yeast fungi.** The former can be recognized by their forming small cubical packets of cells resembling miniature bales of wool; the latter consist of round or oval cells in chains or clusters. They are usually about the size of white blood corpuscles.

The addition of a very little dilute iodine solution to the vomited matter may render the detection of sarcinæ more easy. The iodine stains them a deep mahogany brown.

Permanent preparations of these fungi may be made by spreading out some of the deposit in a thin layer on a cover-glass and drying over a flame. The best stain for sarcinæ is an extremely dilute—almost transparent—solution of gentian violet. Stain for a minute or two. Bismarck brown also gives good results; stain very briefly. For yeasts use a 2 per cent. solution of methylene blue, and stain for half a minute. Wash in water in both cases, dry between filter-papers, and mount in balsam.

VI. EXAMINATION OF FÆCES *

1. **Naked-eye inspection.**—The following points should be attended to:—

(*a*) Amount of the daily stools.
(*b*) Their colour.
(*c*) Their odour.
(*d*) Their consistence and form.
(*e*) The presence of any abnormal ingredients.

* It is often desirable to examine the fæces of a patient upon a certain diet. This may be satisfactorily accomplished by giving 0·2 grm. of powdered charcoal in a gelatin capsule, or a couple of charcoal biscuits, at the beginning and at the end of the diet, and

As regards **amount**, it is usually sufficient to state whether the stools are copious or scanty. The average daily amount of fæces in health is 120–180 grm. (about 4 oz.).

The **colour** of normal fæces is partly due to stercobilin, partly to chlorophyll and other pigments. A mixed diet yields stools varying in colour from light to dark brown—being darker with an exclusive meat diet, lighter with a milk diet. The presence of unaltered bile pigment is always abnormal, and may be due to increased rapidity of peristalsis. **Black** stools may be produced by the administration of iron, bismuth, or manganese. In hæmorrhage high up in the intestine the altered blood makes the stools dark, tarry-looking, and very offensive. The blackness due to blood may be distinguished from that produced by drugs by mixing part of the stool with twice its volume of water and allowing it to stand in a glass jar. If blood be present the water becomes reddish; under other conditions it remains dark or greenish.

Pallor of the stools may be due to an obstruction to the entrance of bile into the intestine, as in jaundice, or to extreme dilution of the stool, as in cholera.

The **odour** of the fæces is due to the presence of indol and skatol. The intensity of the odour depends to a large extent upon the amount of meat ingested. The absence of bile seems to favour putrefaction; hence the stools in jaundice are often very offensive. Cholera stools, on the other hand, contain very little

collecting the fæces between the two black stools thus produced. Various "test diets" for the investigation of the intestinal functions have been proposed, but it is sufficient to make the patient include the following substances in the dietary for about forty-eight hours before the stool is examined, viz. (1) milk, (2) eggs, (3) meat in some form, (4) farinaceous food, e.g. bread, potatoes, or rice, (5) green vegetables and stewed fruit, (6) fats, e.g. butter, bacon-fat, ham, etc. The choice and amount of the individual articles may be left to the patient's taste.

organic matter, and are almost free from odour. In fermentative processes in the intestine the stools may have a sour smell.

The **form** and **consistence** of the stools is of importance. In obstinate constipation the stools may be much drier and harder than normal, and even friable. In all forms of diarrhœa they are more fluid than normal, and may even be watery. Slimy stools are due to the presence of an excess of mucus.

It is important to note whether the stools are formed or fluid. If formed, any abnormality in the shape should be noted. The stools of constipation have often the form of round balls, frequently coated with mucus. In obstruction in the large intestine the stools may be ribbon-like. If ascites be present, the pressure of the fluid on the bowel often leads to flattening of the fæces. The presence of a rectal polypus may produce a groove or furrow along the fæcal mass.

In order to facilitate the detection of **abnormal ingredients,** the stool should be placed on a fine sieve, and a large quantity of water added. The whole is shaken and stirred up till the soluble parts are all washed away. The residue is then examined.

For the detection of finer particles it is better to take a portion of the fæces of about the size of a walnut and to rub it up in a mortar with some distilled water to the consistence of pea soup. It should then be poured into a shallow glass dish with a black background against which particles of mucus or of food residues are easily seen.

Gall-stones are easily recognized. It is important to note whether they are faceted or not, for if they are, then the stones are multiple. Particles of undigested food, fruit stones, foreign bodies, concretions—e.g. those produced by magnesia—and parasites should all be looked for.

The full consideration of the parasites which may be found in the stools is undertaken later. We would only mention here that one has often to search stools for the **head of a tapeworm.** The best method of procedure in such a case is to add to the stool a considerable quantity of water containing a little carbolic acid, and to shake the mixture gently for a few moments. It is then allowed to stand for about ten minutes. The parasite sinks to the bottom, the supernatant fluid is poured off, and more water added till the residue is nearly colourless. The parasite will then be readily found. The head is only about as large as that of a large pin, and the neck about as thick as a stout thread.

Special terms are applied in clinical medicine to some particular **varieties of stool.**

The **bilious** stool is well illustrated in the typical stool of typhoid fever. Its characters are described in the term of " pea-soup " stool, usually applied to it.

Watery stools are found in all cases of colliquative diarrhœa, and after the administration of hydragogue cathartics. To the stools of cholera the special name of *rice-water* stools is applied. Such a stool is colourless, almost devoid of odour, alkaline in reaction, and contains a number of small flocculi consisting of shreds of epithelium and particles of mucus. The name is applied to it from its resemblance to the water in which rice has been boiled. **Purulent** or pus-containing stools are found in severe dysentery or intestinal ulceration, or in cases where an abscess has found its way into the intestines. **Slimy** stools are due to the presence of an excess of mucus, and point to an affection of the large bowel. The mucus may envelop the fæcal masses, or may be intimately mixed with them. **Bloody** stools vary in appearance according to the site of the hæmorrhage. If the bleeding takes place high up, the

stools look like tar, as has been already mentioned. In an ordinary intussusception the stools may look like red-currant jelly. In those rare cases in which the intussusception occurs in the jejunum the appearance of the material passed per anum has been compared to that of a melted strawberry ice. If the hæmorrhage be from the large intestine, the blood is less intimately mixed with the fæcal matter, and may even be of a bright colour. In hæmorrhage from the rectum or anus it may merely streak the fæcal masses.

In membranous colitis the motions contain **casts of the bowel,** which are mainly composed of **mucin.** The individual casts vary considerably in size, being commonly from 1 in. to 6 in. long, but in exceptional cases attaining a length of 1 ft. or more. They vary in width from narrow strips to tubes more than 1 in. in diameter ; they are sometimes grey and transparent like ordinary mucus, at other times they are more opaque and resemble the casts formed in the air-passages in membranous croup. Sometimes they are stained of a brownish-yellow colour from adherent or incorporated fæcal matter ; rarely they are red from the presence of blood. They are often very abundant, and may become agglomerated into irregular masses which, when floated on water, can be unravelled into their component parts. As a rule the cast is tubular, but the wall varies much in thickness, and the lumen may be obliterated. If teased out in water one can sometimes separate the cast into membranous layers between which small particles of fæcal matter may be observed.

These casts, when small, may, on casual observation, be mistaken for segments of tapeworm. On the other hand, cases are recorded in which the remains of indigestible substances in the food or firm clots of curdled milk have been regarded as casts.

Chemically, they are mainly composed of a mucin body with which small amounts of globulin and other albuminoids are associated. The presence of fibrin in any quantity appears to be very uncommon, if, indeed, it ever occurs.*

The stools on rare occasions contain **intestinal sand,** which may be of either mineral or vegetable origin. The former, which is sometimes called "true sand," chiefly consists of phosphate of calcium, associated with smaller amounts of carbonate of calcium and of silica, around an organic nucleus of animal origin. When it is washed and dried it usually presents the appearance of ordinary fine sand, some of the particles being yellowish-brown and others almost colourless. On microscopic examination the grains are found to be very varied in shape ; some are oval and smooth, others irregular and rough, whilst in structure they are granular rather than crystalline.

When the sand is of vegetable origin it is known as "false sand," and consists of sclerenchymatous particles such as those that are present in pears, bananas, and some other fruits. Under the microscope it is easy to recognize the nature of the grains, as, after the removal of any inorganic incrustation by an acid, the thick transparent walls of the woody cells, traversed by channels passing between the surface and the narrow cell-cavities, are clearly visible.

2. **Chemical examination.** i. **Reaction.**— If fluid the stool should be well mixed, and if solid a small portion rubbed in a mortar with distilled water to a soup-like consistency. A drop should then be applied with a glass rod to a piece of blue or red litmus

* If present, fibrin may be demonstrated by fixing and hardening a small portion of the cast with sublimate and alcohol, and, after preparing a section for the microscope, staining it with Ehrlich's triple stain, when the mucin is coloured green, whilst albumins and fibrin appear red.

paper previously moistened. The reaction can easily be seen on the other side of the paper. A normal stool should be nearly neutral. Marked acidity indicates fermentation; marked alkalinity, putrefaction.

ii. **Test for bile pigment.**—Mix some of the stool with a concentrated solution of corrosive sublimate and allow it to stand twenty-four hours. A normal stool is turned red from the presence of urobilin; a green colour shows unaltered bile pigment. A complete absence of either green or red colouring shows the absence of bile altogether.

The tests for bile pigments and urobilin described under Urine (pp. 310, 366) may also be performed with a semi-fluid mass of fæces and water; indeed, they are preferable. Bile acids, if present, may be obtained by extracting the fæces with alcohol, filtering, evaporating off the alcohol, and dissolving the residue in dilute alkali. Test with a few drops of cane-sugar solution and H_2SO_4; a purple colour indicates the presence of cholalic acid (Pettenkofer's test). *See also* the tests for bile salts in urine (p. 366).

iii. **Test for occult hæmorrhage.**—Previously to carrying out this test the consumption of red meat in any form should be avoided for two or three days. Proceed as follows: A portion of the stool, the size of a hazel-nut, is broken up with about 2 c.c. of water in a mortar. This is placed in a test tube. Half its volume of glacial acetic acid is added, and the whole is shaken thoroughly. Then the test tube is nearly filled with ether, and is carefully reversed twenty or thirty times. In this way hæmatin acetate is extracted and an emulsion is avoided. To about 1 in. of this resulting translucent yellow ethereal solution are added (*a*) a few drops of glacial acetic acid, (*b*) 1 in. of saturated solution of benzidin in rectified spirits,* (*c*) 1 in. of liquor

* This solution should be prepared each time, as it does not keep.

hydrogenii peroxidi. The mixture is shaken and a few drops are poured on to a white porcelain slab. Blood present even in minute quantities causes a blue colour to appear.

The fæces may also be examined for blood by suitably diluting and examining spectroscopically (*see* p. 241).

3. **Microscopical examination.**—It is best to use a low-power objective, preferably about 1½ in.

Prepare a film as follows: Remove a portion of the fæces, about the size of a split pea, with the end of a match. Place it in the centre of a slide and lay another *slide* on the top, and press the two together. If the stool is very hard, one may soak the selected portion in water for a few minutes before preparing the film. If liquid, a drop of the sediment may be taken up with a pipette, placed on a slide, and covered with a *cover-slip*.

The following are the chief constituents of the fæces to be looked for: (1) Muscle fibres, (2) connective-tissue and elastic fibres, (3) starch granules, (4) vegetable detritus, (5) fat, fatty acid crystals and soaps, (6) triple phosphate, oxalate and cholesterin crystals, (7) mucus, (8) blood, (9) yeasts and other fungi.

Muscle fibres are easily recognized by their cross-striation. If present in large numbers they indicate defective intestinal digestion.

Connective tissue may somewhat resemble mucus, but is distinguished by its striation, which disappears on the addition of acetic acid. If masses of it be seen, defective gastric digestion may be inferred. Elastic fibres have no significance.

Starch granules are readily detected if a drop of iodine solution be added. Their presence in excess* is pathological, and such a stool is usually marked by acid and often shows signs of fermentation (gas bubbles) and the presence of yeasts.

* Experience alone can tell what constitutes an excess of starch granules in the fæces.

Detritus derived from vegetables and fruits is easily identified by its areolar tissue, spiral ducts, vascular bundles, and pigment cells.

Neutral fat occurs as colourless, highly refractile droplets, or as bile-stained irregular masses which stain with Sudan III. and are soluble in ether.

Fatty acids occur as sheaves of colourless acicular crystals which dissolve on warming or in ether.

Soaps occur as greasy-looking amorphous masses, or as needles which are shorter and thicker than those of fatty acids. They dissolve on warming, but not in ether. On heating the slide with a drop of acetic acid, crystals of fatty acid will be seen to separate out.

A simple way of distinguishing fats from mucus or vegetable material is to press down the cover-slip. If the material be fat the slip remains down ; if vegetable detritus or mucus, it springs back when the pressure is released, and air rushes in from all round.

Normally, fat in the fæces is almost entirely in the form of amorphous masses of soap ; less often as crystals. Neutral fat should be practically absent.

Triple phosphate crystals (Fig. 95) are always present in normal fæces and are never coloured by bile. Oxalate crystals are generally found as well, especially when much vegetable food is taken. Cholesterin crystals may also be found in normal fæces.

Mucus occurs as transparent blobs or shreds, sometimes bile-stained. It is always pathological, and, if it contains numerous leucocytes or epithelial cells, is conclusive evidence of intestinal catarrh.

VII. INTESTINAL PARASITES

The parasites which occur in the intestinal tract include worms and protozoa. The worms belong either to the nematoda or to the flat worms, the latter group containing the cestoda, which are fairly

common, and the flukes, which, in Europe at least, are by no means ordinarily found in man.

A. Nematoda

1. Perhaps the commonest of all internal parasites

Fig. 9. — Oxyuris vermicularis.
1, female; 2, male. Nat. size. (*After Payne.*)

is the small thread-worm, **Oxyuris vermicularis,** whose presence is associated with considerable itching about the anus. It inhabits the large intestines, cæcum, and vermiform appendix, and specimens can often be seen wriggling about in the recently passed motions of their host. To the naked eye they look like small white threads, ½–1 cm. in length. Under the microscope the female may be distinguished by the large uterus

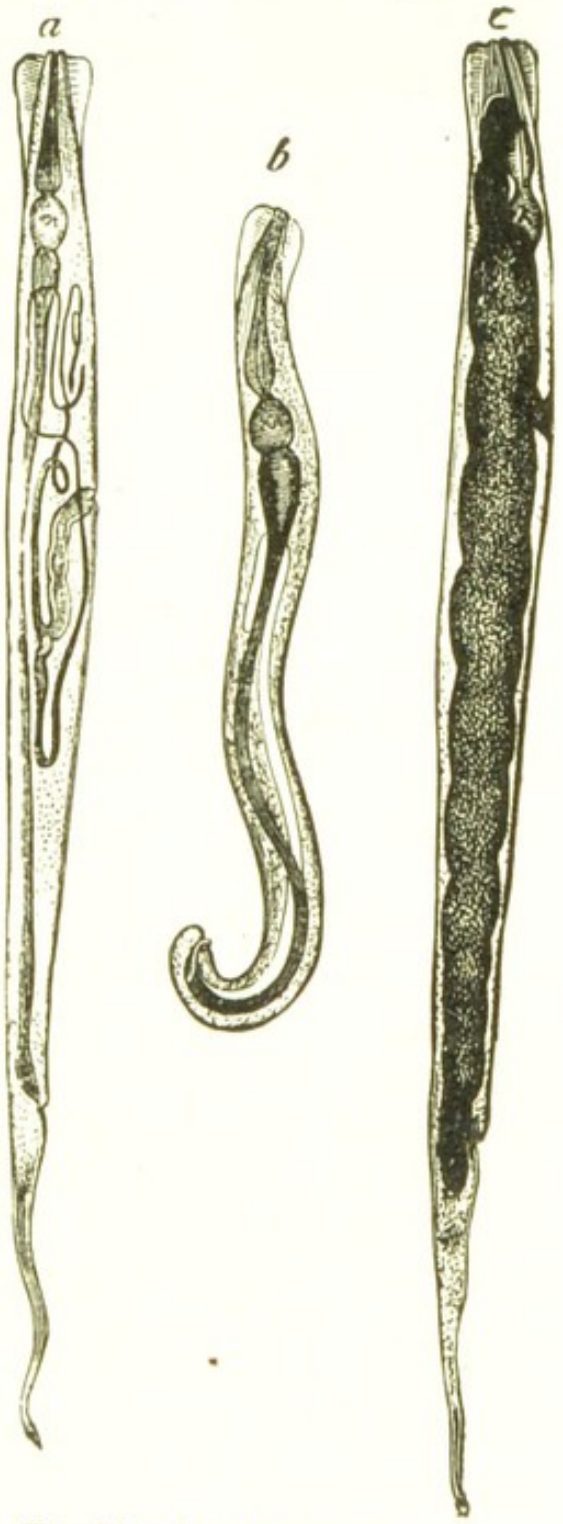


Fig. 10.—Oxyuris vermicularis.
a, young female; *b*, male; *c*, mature female. Magnified. (*After Payne.*)

Fig. 11.—Ankylostoma duodenale.
a, male; *b*, female. Nat. size. (*After Payne.*)

filled with ova, and the pointed posterior end, whence its name is derived (Figs. 9, 10, 15, A).

2. **Ascaris lumbricoides** has a general resemblance to an earthworm. It measures, as a rule, from 6 to 8 in., and sometimes considerably exceeds this length. Not infrequently its presence in children is associated with nervous disorders. The ova, which can occasionally be found in the dejecta, have brownish-yellow granular contents, and in many cases the shell is surrounded by an irregular albuminous sheath (Fig. 15, B).

3. **Ascaris mystax,** a closely allied worm, is sometimes parasitic in children; the infection is got from cats.

4. **Ankylostoma duodenale** is a parasite whose presence is fraught with much greater risk to the host than that of those already mentioned, as it causes profound anæmia by drawing blood from the wall of the bowel. It lives for the most part in the upper part of the jejunum, and its existence there is rendered probable when, in an infested district, severe anæmia, otherwise inexplicable, sets in. The diagnosis is clinched by the discovery of ova in the motions. They exhibit a

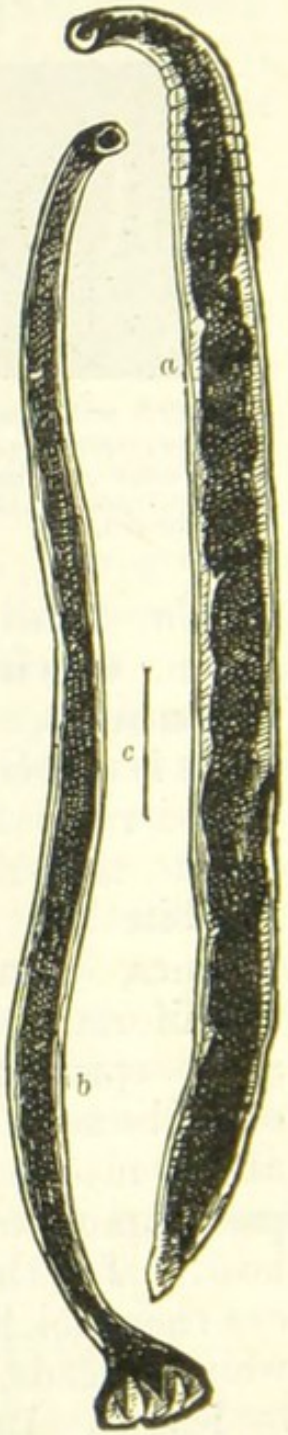


Fig. 12.—Ankylostoma duodenale.
a, female; *b*, male—magnified. *c*, nat. size. (*Bristowe.*)

segmented yolk enclosed in a thin shell, and are sufficiently numerous to be readily detected. The adult worm, which is but rarely seen before therapeutic agents have been employed, is about half an inch long, and the mouth is provided with four claw-like teeth. The male is distinguished by its lobed caudal bursa. (Figs. 11, 12.)

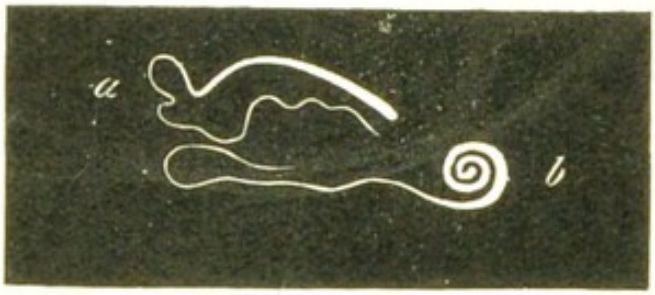


Fig. 13.—Trichocephalus dispar.
a, female; *b*, male. Nat. size. (*After Payne.*)

5. **Trichocephalus dispar** is, perhaps, a commoner parasite than might be suspected; its presence does not seem to cause any very serious inconvenience. The length of the worm is under 2 in., its colour is white; the anterior portion is much narrower than the posterior, and is buried in the mucosa of the cæcum. The ova are very characteristic, and when present are readily recognized. (Figs. 13, 15, c.)

6. **Trichina spiralis** occurs in the alimentary canal in the sexually mature state. The striped muscles are the habitat of the embryonic form, and when much affected they may become shortened. This is well seen in the biceps, where the contraction induces a very typical flexion of the forearm. The adult male measures 1·5 mm. in length; the female, which is viviparous, is about twice as long.

B. Cestoda

Many different kinds of tapeworm have been found as parasites in man, but those of most importance are *Tænia solium*, *T. mediocanellata*, and *T. echinococcus*. *Tænia cucumerina* is also occasionally found in children. Besides its occurrence in the fully developed

state, *T. solium* may be present in the tissues, in the form of a cysticercus; *T. mediocanellata* is almost never found in this condition in man; whilst *T.*

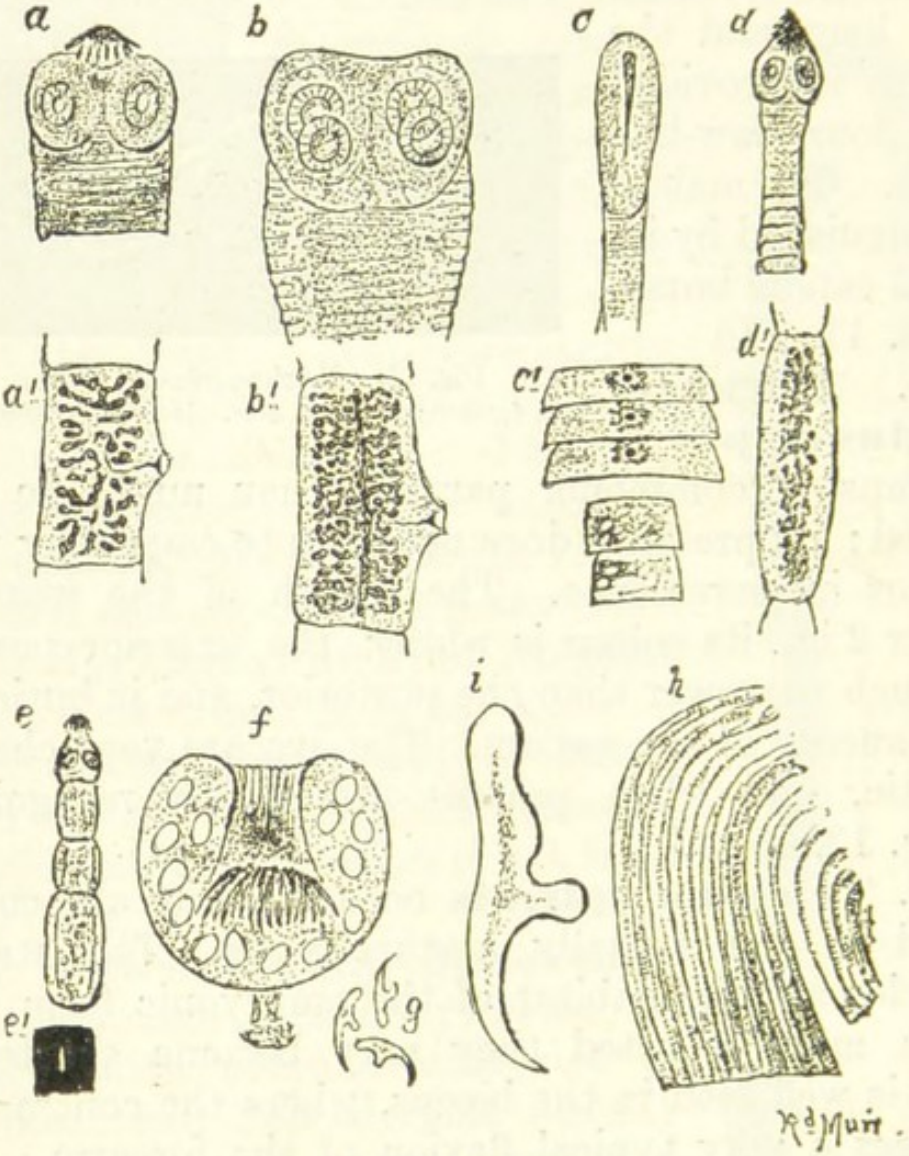


Fig. 14.—Cestoda.

a, head of Tænia solium, × 10; *a'* mature segment of do., nat. size.; *b*, head of T. saginata, × 10; *b'*, mature segment of do., nat. size; *c*, head of Bothriocephalus latus, × 10; *c'*, mature segment of do., nat. size; *d*, head of T. cucumerina, × 10; *d'*, mature segment of do., nat. size; *e*, T. echinococcus, × 10; *f*, hydatid scolex (invaginated), × 250; *g*, hydatid hooklets, × 250; *h*, hydatid membrane (ectocyst), × 220; *i*, booklet from cysticercus, × 25.

echinococcus always occurs in the cystic stage, and has never been found in the mature condition in the human intestinal tract. (Fig. 14.)

The presence of an adult tapeworm in the bowel is

generally revealed by the passage of ripe proglottides in the stools, and after the administration of anthelmintics the head may be detected by the methods previously described.

1. **Tænia solium** (Figs. 14, *a*, *a'*, 15, D). The mature worm measures 2 or 3 yards in length; a ripe proglottis is about 10 mm. long, and 6 mm. broad, with the sexual opening placed laterally; the uterus is coarsely branched. The head, whose size is about the same as the head of a large ordinary pin, has four suckers, often pigmented, and a small rostellum, with a ring of 20 to 30 hooklets. The ova, which are nearly spherical, are readily recognized by their thick shell, with radiating striations. Inside the ovum, when mature, the six hooklets of the embryo may be visible.

The cysticercus varies in size according to its situation, but never attains anything approaching the magnitude of an echinococcus cyst. The vesicle contains one head only, whence the adult worm is developed.

2. **T. mediocanellata (sagimata)** is larger than *T. solium*, and attains a length of 5–9 yards. The ripe proglottides measure 16 mm. in length, by 5 mm. in breadth, but immature segments are broader than they are long. Very often they exhibit movements after they have been detached from the strobilus and have passed from the bowel. The sexual opening is lateral, and the uterus is finely ramified, with frequent dichotomous divisions of the primary branches. The head is rather square in outline, and is larger than that of *T. solium*. It has four suckers, but is devoid of hooklets. The ova closely resemble those of *T. solium*, but are slightly longer in proportion to their breadth (Figs. 14, *b*, *b'*, 15, E).

3. **T. echinococcus.**—The adult worm, which consists of a head and three segments, and whose length is only 4 or 5 mm., need not be fully described,

since it is not found in man. The *cystic stage* is very important, as it gives rise to serious disease in many of the viscera, and especially in the liver.* The cysts of this tænia are not simple, but produce, from their inner surface, one or two generations of secondary vesicles, on which the brood-capsules, containing the cestode heads, are formed. During the period in which this process is going on, the primary vesicle dilates to accommodate its increasing contents, and may eventually reach the size of a cocoa-nut. The vesicles may rupture spontaneously and their contents may escape by the lungs, by the bowel, or by the urinary passages, or specimens may be obtained by aspiration, or after surgical interference. In a case of suspected hydatid disease one may require to found the diagnosis either on the chemical nature of the fluid withdrawn, or on the recognition of hooklets or scolices, or on the appearance of the ectocyst, portions of which are sometimes discharged, especially when the cyst has opened into the lungs and bronchi (Fig. 14, *e*, *e'*, *f*, *g*, *h*).

The *fluid* is clear, alkaline, devoid of albumin, and contains abundance of sodium chloride and traces of glucose. Its density is low, being generally under 1010. The appearance of echinococcus *hooklets* is shown in Fig. 14, *g*, *i*. The *scolex*, if it is obtained in a perfect condition, is about 1–1½ mm. in diameter, and a number of them often spring in a group from one brood-capsule. They have four suckers and a crown of hooklets. Portions of the *ectocyst* appear as whitish-yellow shreds, which can be recognized under the microscope (× 250 diam.) by their lamination, and by the pectinate markings on the laminæ (Fig. 14, *h*).

* The so-called "hydatid thrill" is described elsewhere (p. 62). It may occur where there are no daughter cysts, and it may be absent when daughter cysts occur.

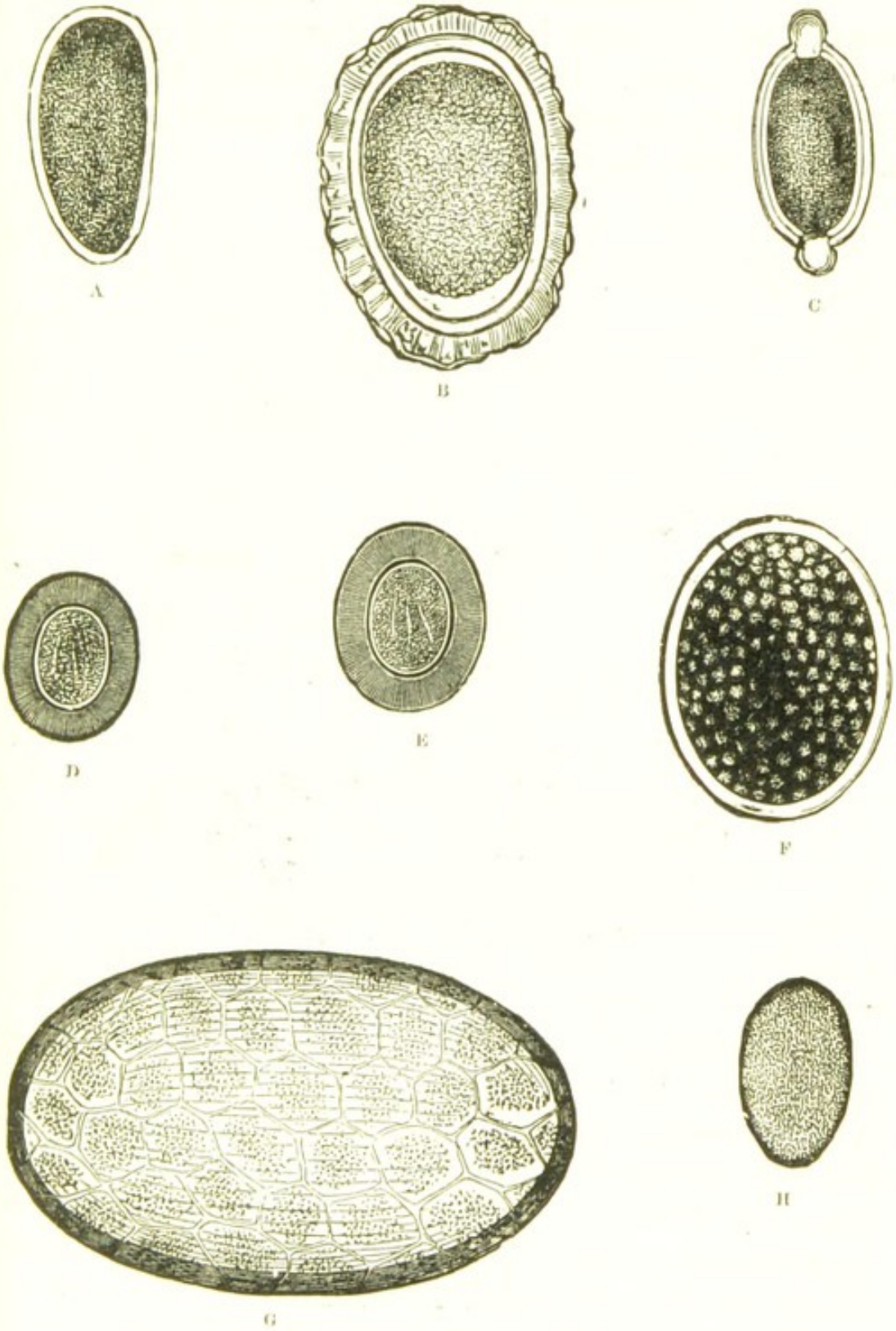


Fig. 15.—Ova of entozoa, × 350. *(After Hiller.)*

A. Oxyuris vermicularis; B. Ascaris lumbricoides; C, Trichocephalus dispar; D, Tænia solium; E, Tænia mediocanellata; F, Bothriocephalus latus; G. Distoma hepaticum; H, Distoma lanceolatum.

4. **T. cucumerina** and **Bothriocephalus latus,** though rare in Britain, are common in some parts of Europe. The appearance of the head and of a mature segment of each is shown in Fig. 14, *d*, *d'*, *c*, *c'*, and the ovum in Fig. 15, F.

C. Trematoda

1. **Distōma hepaticum** (Fig. 15, G) is rather

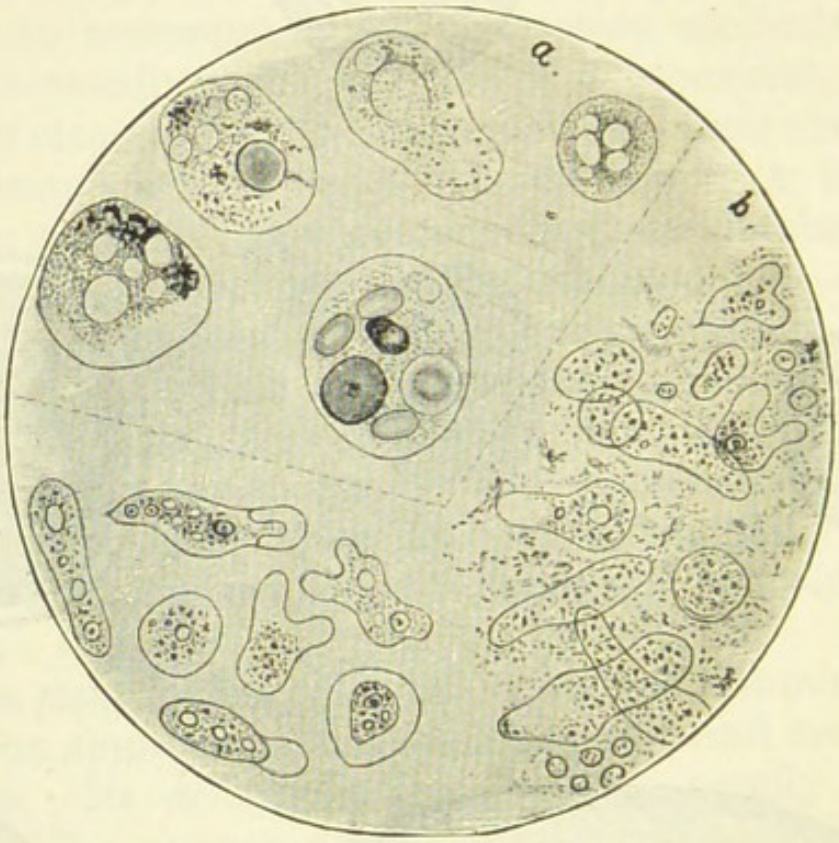


Fig. 16.—Amœba dysenteriæ.

a, A. dysenteriæ fixed and stained (*Councilman*); *b*, A. dysenteriæ in stools (*After Lösch*, "*Virchow's Archiv*," Bd. 65.)

rare as a human parasite. When it does occur, the ova may be found in the fæces, and are recognized by their brown colour, and by the presence of an operculum at one pole. The adult fluke is leaf-shaped, and measures about 25 mm. by 12 mm.

2. **Distōma lanceolatum** (Fig. 15, H) is considerably smaller, and is narrower in proportion to its length. The ova are similar to those of *D. hepaticum*, but smaller.

D. Protozoa

A number of protozoa, including members both of the Rhizopoda and of the Infusoria, have been found in the fæces. The only one of undoubted clinical importance is the **Amœba dysenteriæ,** which is present in a great proportion of cases of tropical dysentery, and also in tropical abscess of the liver. (Fig. 16.)

When in a state of activity amœbæ vary in form, and throw out pseudopodia, which are thick and few in number ; usually only one or two are present. The division of the two plasma layers is indistinct and the ectoplasm is small in amount. At rest they become spherical, measure on an average 12–26 μ in diameter, and generally exhibit a nucleus. The resting condition is that in which they are usually found in the fæces, when they are recognized by their high refractility and their greenish tint. For careful examination, a portion of the dejecta should be hardened in Müller's fluid, and subsequently cut and stained like sections of tissues ; or cover-glasses, smeared with a thin film of the fæcal material, can be prepared and fixed with corrosive sublimate. The specimen should be stained with methylene blue. The amœba is most easily detected in the little masses of mucus which occur in the stool, and attention should always be carefully directed to these. When the motion is pultaceous the addition of warm solution of sodium chloride will be found to faciiitate the preparation of a suitable film, in which the organisms may be seen moving about if care be taken to maintain the slide at the temperature of the body. If the motion is a formed one the mucus adhering to its surface should be exclusively examined. In addition to amœbæ, dysenteric stools invariably contain bacteria, amongst which streptococci and the *Bacillus coli communis* seem to preponderate.

CHAPTER IV

THE CIRCULATORY SYSTEM

I. ANATOMY

THIS system is composed of two main elements, the heart and the blood-vessels, and these are for the most part dealt with separately, although, when the chest is exposed for the examination of the heart, the vessels in the thorax and at the root of the neck are more conveniently examined along with it. (*See* Plate 1.)

The heart lies obliquely in the thorax, being inclined from above downwards, forwards, and to the left. Two-thirds of it lie to the left of the middle line. The part which reaches highest in the thorax is the **left auricular appendix,** which in the cadaver extends as far up as the 2nd left costal cartilage. During life it is usually opposite the 2nd interspace or lower border of the 2nd cartilage, as the diaphragm then occupies a lower level. The greater portion, however, of the **left auricle** lies posteriorly, and constitutes the hindmost cavity of the heart.

The **right auricle** is the chamber that lies most to the right. It extends somewhat beyond the right margin of the sternum, and its border may be traced by a curved line joining the 3rd and 7th right chondro-sternal articulations, and reaching about 1 in. to the right of the sternum.

The **right ventricle** occupies the great portion of the front of the heart. Its inferior margin extends from the 7th right chondro-sternal articulation to the apex, and constitutes the lower border of the heart.

The **left ventricle** only appears in front as a narrow strip, scarcely ½ in. broad, and its outline completes that of the heart on the left, where its border forms a curved line, ascending from the apex to the lower margin of the 2nd left interspace at a point just internal to the parasternal line. The topographical anatomy of the valves of the heart and of the great vessels will be discussed in connection with auscultation, as it is in this department that a knowledge of their situation is most necessary (pp. 153–56).

The most important organs which come into relation with the heart are the lungs on either side, the liver below, and the great vessels above. A small portion of the anterior surface is only separated from the thoracic wall by the anterior mediastinum, whilst behind, the heart is in relation with the structures that occupy the posterior mediastinum.

That portion of the anterior aspect of the chest which overlies the heart is known as the **præcordial region.**

It is often necessary to define the exact situation of a point on the front of the thorax, and certain landmarks, some natural and some artificial, are commonly made use of for this purpose.

The **ribs** and **interspaces** on either side form convenient horizontal landmarks. In order to count them, one must feel for the ridge which marks the junction of the manubrium with the body of the sternum, known as the angle of Louis.* When this has been found, by running the finger outwards it reaches the 2nd costal cartilage, which articulates with the sternum at this level. It is then easy to reckon upwards or downwards to the other ribs. The determination of the 1st rib directly is neither so easy nor so certain, since it is overlapped by the clavicle.

* Angulus Ludovici.

In order to define the distance of any given point from the mesial sagittal plane of the body, a series of vertical lines is imagined to be drawn on the chest. These are the **midsternal** and **lateral sternal lines,** drawn down the middle and either border of the sternum; the **mammary line,** best defined, since the situation of the nipple is inconstant,* as the vertical line dropped from the centre of the clavicle, or, what amounts to the same thing, the line midway between the middle of the suprasternal notch and the tip of the acromion; the **parasternal line,** midway between the lateral sternal and mammary lines; the **anterior, mid,** and **posterior axillary lines,** descending from the anterior border, the centre, and the posterior border, respectively, of the axilla; and the **scapular line,** which is defined as the vertical line drawn through the angle of the scapula.

The methods commonly employed in the examination of the heart are inspection, palpation, percussion, and auscultation. These will be taken up consecutively, although in practice inspection and palpation are often advantageously combined.

II. INSPECTION

Inspection determines—

(A) **Form**—

1. Of præcordia { Bulging. / Flattening.
2. Of surrounding parts (especially bulging).

(B) **Movements**—

1. In præcordial region { Apex beat. / Diffuse pulsation. / Pulsation at base of heart. / Local indrawing.

* The ordinary situation of the nipple in an adult male of average development may be placed in the 4th intercostal space, 4 in. from the midsternal line.

2. Outside præcordial region { Pulsations at root of neck, ,, in thorax, ,, in epigastrium.

(C) **Dilated veins and venules.**

For inspection of the chest, the patient should be stripped to the waist, and set in a good light, either sitting up or lying on his back. The observer should directly face him, but must be careful not to obstruct the light. In some cases the observer may with advantage take up a position at the top of the bed, and lower his head until he looks along the chest tangentially. By this manœuvre he will be able to study various pulsations with great facility.

The following points must then be systematically noted :—

1. The shape of the præcordia.
2. Pulsations in the præcordial region.
3. Bulging or pulsation outside the præcordia, either at the root of the neck, or the front of the chest, or the epigastrium (scrobiculus cordis).
4. The presence or absence of distended veins on the chest wall or in the neck.

1. **The shape of the præcordia.**—In health the chest is bilaterally symmetrical, and there is no greater prominence on the left side than on the corresponding area of the right. When the right pectoral muscles are exceptionally well developed, the præcordia may be less prominent than the right side of the chest.

In cases where the **præcordial area is prominent,** it must be remembered that other conditions than disease of the heart may have caused the projection, whilst it is equally to be observed that serious disease of the heart is comparatively seldom accompanied by bulging of the præcordia unless it had already

manifested itself when the patient was young and the bones were incompletely ossified.

Should prominence be observed, note whether the ribs are involved, or whether the intercostal spaces alone bulge. The latter condition occurs in pericarditis with effusion. Prominence of the præcordia may be due to disease in the framework of the thorax, such as scoliosis, parietal tumour, or abscess, or to a diseased condition of the thoracic contents, such as cancer of the lung or effusion into the pleural cavity, mediastinal tumour, fluid in the pericardium, enlargement of the heart, especially if it occur in early life, and aneurysm behind or above the heart.

Flattening of the præcordia may be congenital; it may mark the former occurrence of pericarditis; it may be due to retraction of the lung; and in some instances, particularly in certain trades, it may be the result of pressure.

2. **Pulsations in the præcordial region.**—Besides the movement of respiration, which affects the præcordia with the rest of the chest, an impulse which occurs three or four times to each respiration is generally seen in the lowest and leftmost part of that region.

This pulsation is called the **apex beat** of the heart, and in health usually exhibits the following characters:—

(1) It is found in the 5th left intercostal space.

(2) It is limited to an area less than an inch in breadth, and is only visible in one interspace.

(3) It is situated outside the left parasternal line, and inside the left mammary line.

(4) It is due to the impact on the chest wall of the apical segment of the heart; and for clinical purposes the actual apex of the heart may be assumed to be situated at the lowest and leftmost part of the

above area of pulsation, although it may really be slightly lower down and farther out, under cover of a rib.

The apex beat may be abnormal in **force**, in **position**, or in **extent**. Even in perfect health, if the chest is well clothed, and the apex lies behind a rib, it may be quite invisible. *Disappearance*, therefore, of the apex beat is not to be regarded as necessarily indicative of disease, though it must not be forgotten that it is in cases of weak action of the heart that it is most frequently absent or diminished in force. When abolished, its place may be taken by a more *diffuse impulse* over the lower part of the præcordial area, in cases where the apex is pushed away from the chest wall by a dilated right ventricle, or when pericardial effusion separates the heart from the front of the thorax. On the other hand, the apex beat may appear to be *more forcible* than usual in cases where the heart's action is excited, where the chest wall is thin, or where the left ventricle is hypertrophied. Such changes are more accurately observed by palpation, and will be discussed under that head.

The **position of the apex beat** may be altered in three classes of cases. The cause may be (*a*) *congenital*, where the heart is reversed so that the apex lies to the right (*dextrocardia*), or where other developmental anomalies are present. The displacement of the apex beat may be due to (*b*) *extrinsic causes*, where the heart is displaced by diseased conditions of surrounding viscera which push or pull it from its usual site. Instances of this are found in pleurisy with effusion, in abdominal tumours, and in retraction of a lung.

Where the heart is pushed over to the right by a left pneumothorax, or pleuritic effusion, the pulsation which is conspicuous to the right-hand side of the sternum is not that of the apex, which is usually lying

somewhere behind the bone, but is due to pulsation of the right ventricle and auricle.

Third, the displacement may result from (*c*) *disease of the heart or pericardium.* The apex beat is displaced *mostly outwards* when the heart is dilated; *downwards and outwards* when the left ventricle is hypertrophied; and often *upwards* when fluid is present in the pericardial sac.

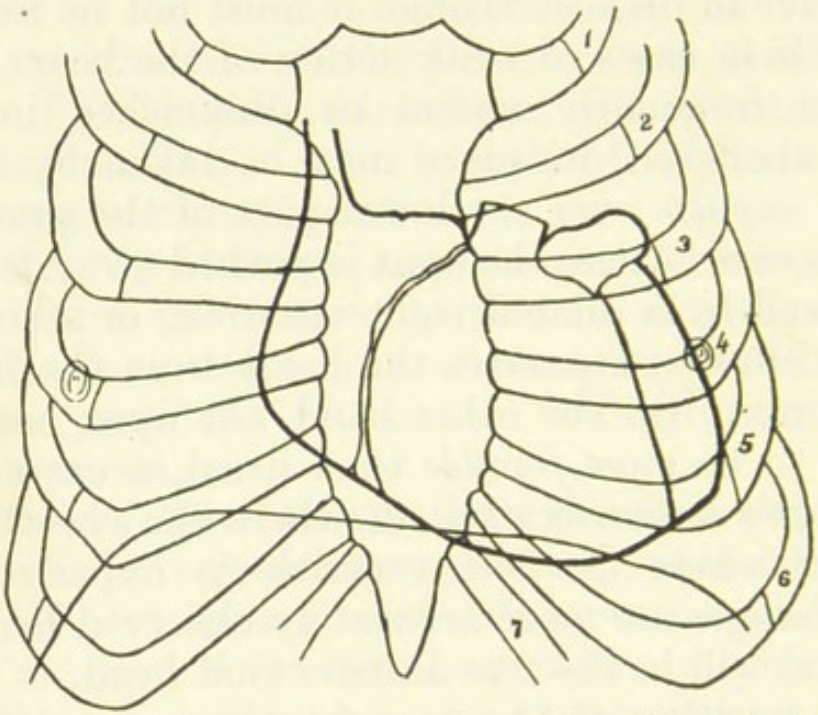


Fig. 17.—The heart in an infant. (*After Symington.*)

In addition to these causes, it should be remembered that the position of the apex beat varies considerably with the patient's age; *in children* it is usually as high as the 4th interspace * (Fig. 17); *in the aged* it descends as low as the 6th.

In certain cases the apex beat is replaced by an *indrawing* of the same area during cardiac systole. This, when it is marked in degree, and when it appears over an extensive area of the lower segment of the

* Even in young children, although the apex beat is seen in the 4th interspace, the actual apex lies lower down—usually behind the 5th rib.

præcordia, suggests pericardial adhesion. When only slightly visible and limited to the apex, it is of no great consequence.

The **extent of the apex beat** is increased in cases where the heart is hypertrophied. This condition must not be confused with diffuse pulsation over the præcordia. *Doubling of the apex beat* sometimes occurs, and may be due to various causes, of which the most important are alternate systole of the right and left heart, and hemisystole.

The consideration of **other pulsations** which may be visible in the præcordial region must next be undertaken. Allusion has already been made to the diffuse pulsation which occurs when the right ventricle is dilated and hypertrophied, and which in these circumstances is visible over the lower part of the area in several of the intercostal spaces, and nearer the middle line than the normal apex beat.

Pulsation is also seen at times in the 2nd left intercostal space. It may arise either in the pulmonary artery, which lies half under cover of the left side of the sternum, and half under the inner end of this interspace, or in the left auricular appendix. In the former case it follows the apex beat and marks the closure of the pulmonary valves; in the latter case, which is not common, it immediately precedes the apex beat.

In chests which have thin parietes, and especially when, in addition, the left lung is retracted from phthisis or other disease, pulsation of a diffuse nature can be observed over most of the interspaces of the præcordial region, as well as at the apex. In these cases the apex beat still causes a limited area of the chest wall between the left parasternal and mammary lines to bulge forward with each beat of the heart, whilst the diffuse pulsation which is caused by the

systole of the right ventricle is associated with indrawing of some portion of the intercostal spaces. This retraction, accompanied by a normal apex beat, and not confined to the lower præcordial segment, must not be confused with the systolic indrawing already described as characteristic of pericardial adhesion.

3. **Pulsations outside the præcordia.**—In addition to the pulsations already described, movements should be looked for at the root of the neck, the front of the chest, and the epigastrium.

At the root of the neck pulsation may occur either in the episternal notch or externally to the sterno-mastoid.

In the episternal notch the pulsation is usually systolic in time, and when well marked is generally an indication of dilatation or aneurysm of the transverse portion of the arch of the aorta. Less commonly it is due to a thyroidea ima artery of considerable size, or to an abnormal origin of the right subclavian from a point to the left of the middle line. Palpation generally enables these conditions to be discriminated. Pulsation here and in the carotids is not uncommonly seen in cases of chlorosis and in other forms of anæmia.

Outside the sterno-mastoid various pulsations may be observed. These may be either arterial or venous. The carotids pulsate visibly on exertion; from mental excitement; in diseases which cause excitement of the circulatory system, such as exophthalmic goitre; in cases of hypertrophy of the left ventricle, especially when associated with aortic incompetence; and in aneurysm of the artery.

The jugular veins may exhibit undulation or pulsation. This is usually caused either by overfilling of the veins when the right auricle is distended with blood, or by actual regurgitation through an incom-

petent tricuspid valve. It will be discussed under the head of Venous Pulse (p. 202).

In the thorax, besides the pulsations referred to as occurring in the præcordial region, a *diastolic pulsation* may occasionally be observed in the 2nd right intercostal space, and results from the closure of the aortic valves. An important source of pulsation in unusual parts of the thorax is *aneurysm of the aorta.* Such aneurysmal pulsations always manifest themselves at first above the level of the 4th rib, though at a later period they may affect a very considerable portion of the chest wall. The position of the impulse varies according to the part of the aorta which is diseased. If the *ascending aorta* be affected, the pulsation is chiefly to the right of the sternum, whilst the *transverse aorta* gives rise to pulsation under the manubrium sterni, and the *descending aorta* still more to the left. Aneurysm of the *innominate* may project far into the neck. The time of this pulsation is systolic, following immediately on the apex beat, and it may be observed to be expansile in character. The pulsation will, of course, be much earlier manifest when the vessel lies behind soft parts than when it is covered by bone.

Pulsating empyema may be present; it generally occupies the præcordial area from which the heart is more or less displaced, and malignant tumours with a large blood supply may also give rise to pulsation in the part of the chest wall that overlies them.

Sir J. Broadbent has pointed out that marked *systolic retraction* of some of the lower ribs on the lateral and posterior aspects of the thorax may occur as a result of extensive pericarditic adhesion, involving not only the central tendon, but also the muscular part of the diaphragm on the one hand, and the interior wall of the thorax on the other. It usually occurs on the left side.

In the epigastrium there may be several kinds of pulsation. The first thing to be determined is whether it is *strictly systolic*, coinciding exactly with the apex beat, or whether the pulsation is *slightly delayed*, so as to appear just after the apex beat has occurred.

In the former case the pulsation is caused by a dilated and hypertrophied right ventricle, which either conveys its impact directly to the parietes, or does so indirectly by exercising a thrust upon the liver, or else it is due to the apex beat of a heart displaced to the right by some diseased condition, of which the most important are left-sided pleurisy and pneumothorax.

In the case of delayed pulsation, the cause may be arterial. The existence of an aneurysm of the abdominal aorta would produce such an effect. More commonly, however, the condition is simply neurotic; whilst in other instances the pulsation of a normal abdominal aorta is conveyed to the surface either by the liver or by an abdominal tumour, such as pyloric cancer, which lies in front of it. (*See also* p. 56.)

In cases of regurgitation from the right heart, pulsation also occurs just after the apex beat, and is due to a distensile pulsation of the liver itself from the back flow of blood into the hepatic veins. It should be observed, however, that distensile pulsation of the liver is by no means a common condition, and does not occur in all cases of tricuspid regurgitation.

It is comparatively rare to observe a *systolic depression of the epigastrium*. If well marked, it would probably indicate adhesions resulting from an old pericarditis.

In order to observe with greater facility the characters and time relations of these various pulsations, one can employ small flags, made as light as possible and attached to the various areas of the chest wall. To determine whether a pulsation is expansile, place

one of these flags on either side of the tumour. If it be expansile their free extremities will recede from each other as the tumour fills. If it is desired to time the occurrence of two pulsations, after fixing a flag on the point where each occurs, one may take up a position in which they are as nearly in line as possible. It is then quite easy to determine which of them begins to move first.

Flags can readily be improvised by taking a piece of straw or a bristle about 3 in. long, fixing a fragment of gummed paper to one end, and surrounding the other with a pellet of modeller's wax or stiff ointment which will adhere with sufficient tenacity to the skin. Other and more primitive methods may also be used, such as passing a pin through a piece of adhesive plaster, with the head to the sticky side, and fixing it on the chest, or affixing little cones of cotton-wool to the points in question by means of vaseline.

4. **Conspicuous veins.**—The veins of the thoracic wall may be unduly conspicuous. This occurs (*a*) when the patient's skin is unusually transparent; (*b*) when the patient has been undergoing considerable exertion, especially when the effort is of such a kind (e.g. playing a wind instrument) as to throw a strain on the respiratory system; (*c*) when intrathoracic tumours impede the return of blood to the heart; (*d*) when the action of the right side of the heart is laboured; (*e*) when, in consequence of portal obstruction or of blockage of the inferior caval system, the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels.

In a number of instances, where the right side of the heart is slightly overworked, a belt of dilated capillaries appears along the line of attachment of the diaphragm.

III. PALPATION *

Palpation determines—

(A) **Form of præcordia, etc.** [Confirms or modifies results of inspection.]

(B) **Movements**—

- (*a*) Apex beat { Position. / Character.
- (*b*) Other præcordial pulsations.
- (*c*) Pulsations outside præcordia { Heaving. / Expansile.

(C) **Vibrations**—

- (*a*) Originating within the heart and blood-vessels. (Thrills.)
- (*b*) Originating exocardially. (Friction.)

By palpation the observer not only confirms the facts determined by inspection and adds to their precision, but is also able to detect movements and vibrations which are too slight to be noted by the eye alone. For palpation the patient should be placed in an attitude which he finds easy to maintain, since the exertion which a constrained position demands is certain to increase the observer's difficulties. If the patient is lying down, care must be taken to keep him on his back. By turning to his left side he will produce a very material alteration in the position of the apex beat, which is thereby displaced outwards towards the axilla ; whilst if he lie on his right side the apex of the heart may recede from the chest wall, and an impulse, which in the more favourable dorsal attitude would be easily felt, may entirely disappear.

The position of the observer is almost as important as that of the patient. For the examination of the præcordia he should stand or sit at the top of the bed, on the right-hand side. He should then place his right hand, which must be thoroughly warm, on the patient's chest, so that the palm lies over the base of

* The study of the pulse, which also belongs to this department, is for convenience placed by itself in another section.

the heart, whilst the fingers are directed towards its apex. To begin with, the whole palm of the hand should be in contact with the chest wall, and care must be taken not to dig the finger-tips into the intercostal spaces, as this causes discomfort, and may thereby interfere with the subsequent observations.

When pulsation is detected over any part of the region under examination, its exact localization is best determined by the pulp of the fingers.

The first pulsation to attract attention is that due to the **apex beat.** Not infrequently the fingers will determine that this is really farther from the middle line than inspection would have led one to suppose. In such a case that point is to be taken as the cardiac apex which is the leftmost and lowest where the finger is distinctly forced up with each beat of the heart. The sensation of a thrust from below raising the finger is important, because in not a few cases where the heart is acting forcibly it communicates some vibration to portions of the chest wall considerably beyond those which actually lie above it.

The observer, having thus determined the site of the apex beat, must study its **extent** and **character.** As has been previously stated (p. 114), it lies in health well outside the left parasternal line, but never beyond the left mammary line, is as a rule confined to one interspace, and seldom can be seen over an area of more than 1 in. in diameter. These points will now be carefully examined by palpation, and any deviation from them noted. In addition, however, an estimate must be made of the *energy with which the heart is acting*, and the apex beat may be found to differ from the normal—and this can only be recognized by continued practice at the bedside—in possessing a "*heaving*" *character* in cases where the left ventricle is hypertrophied, a *sharp slapping impact* where there is irrita-

bility of the heart, or, on the other hand, a *feeble or almost imperceptible tap* when the heart is fatty, or exhausted towards the end of an acute fever.

When the pulsation of the apex of the heart is so feeble as to be imperceptible when the patient is lying down, it often becomes quite distinct if he sits up, and still more so if he leans forward. If, however, these postures are uncomfortable for a patient who is seriously ill, it is better to forgo such advantages as they afford than to fatigue one whose strength is already taxed to the utmost. The chief causes of **impalpable apex beat** are (*a*) a thick chest wall, (*b*) a feeble heart, and (*c*) emphysema of the lungs.

When analysed, the varying characters of the beat will be found, after due allowance has been made for the thickness of the chest wall and intervening lung, to depend upon the force with which the palpating finger is driven upwards, and upon the celerity and amplitude of the movement of the cardiac apex as it approaches the front of the thorax at each ventricular systole. *A shock* or "*jog*" is sometimes felt at the apex in consequence of sharp closure of the pulmonary and aortic cusps.

In addition to pulsation, vibrations may sometimes be observed at or near the cardiac apex. Such vibrations are termed **thrills.**

The time of their occurrence in relation to the apex beat must be determined. When they commence with the apex beat and continue during the period of ventricular contraction, they are termed **systolic**; if they are felt whilst the ventricles are relaxed, they are termed **diastolic**; if they occur near the close of diastole, when, though the ventricles are still relaxed, the auricles have entered upon systole, and run up to the apex beat, they are termed **presystolic.**

These thrills may be due either to valvular disease,

to pericardial friction, or to friction resulting from pleurisy over that part of the left lung which lies in front of the heart. The thrills due to valvular disease will exhibit a more definite relation to the apex beat, both in point of time and in situation of maximum intensity, than those whose origin is exocardial. A systolic thrill, best felt at the apex, may indicate mitral regurgitation, though in some cases the thrill of aortic obstruction is very distinctly felt in this area. A diastolic, and still more a presystolic thrill, when it is best felt at or just internally to the apex beat, is so characteristic of mitral obstruction that, if it is clearly present, one may with a high degree of probability assume that lesion to exist, even in the absence of all other signs and symptoms.

Pericardial or **pleural thrills** will be readily recognized as such when the patient is auscultated (*see* p. 175). They are generally to and fro in character, and are always audible as well as palpable. Pulsation and thrill may be detected over the right side of the heart when its chambers are dilated and hypertrophied, or when its valves are diseased.

Over the second left interspace **pulsation of the pulmonary artery,** sometimes systolic, sometimes diastolic (*see* p. 117), and of the left auricle, always presystolic, must be sought for; and a thrill may not rarely be detected in the pulmonary artery in certain diseases, especially in exophthalmic goitre (*see further* under Auscultation, p. 169).

Over the aorta, where it approaches the front of the thorax near the sternum in the 2nd right interspace and behind the 2nd right costal cartilage, pulsations or thrills may also be detected, while in cases of aneurysm of the root of the vessel or of the ascending part of its arch a characteristic expansile pulsation can sometimes, though by no means always, be

observed. In many cases a diastolic shock of considerable strength can be felt over the aneurysmal sac. The time of occurrence of all these phenomena must be given with reference to the apex beat.

At the root of the neck palpation will frequently enable one to identify a pulsating vessel, and so clear up a doubtful diagnosis. When pulsation occurs in the episternal notch one should, if possible, try to push the finger below the pulsating vessel. By so doing one may be saved the inconvenience of diagnosing an aortic aneurysm when the patient has merely an abnormal origin of his right subclavian.

By pressing the finger firmly down from the episternal notch behind the upper part of the sternum—due care being exercised not to cause the patient too much discomfort—a **commencing dilatation** of the transverse arch of the aorta may be identified in time to allow of effectual treatment being carried out, since in health the aorta lies at so low a level that its pulsation can scarcely be detected by this manœuvre.

Another method of discovering an early dilatation of the aorta, when the under side of the arch is the part involved, has been described by Surgeon-Major Oliver and others under the name of **tracheal tugging.** The explanation of this phenomenon depends upon the fact that in passing from the trachea to the lung the left bronchus lies just below the arch of the aorta, and consequently each time that the aorta is distended the aneurysmal dilatation pushes the bronchus downwards before it, and the latter drags in turn upon the trachea, causing it to descend at each beat of the heart. By standing behind the patient and pressing the cricoid lightly upwards with the finger-tips of both hands, whilst the patient keeps his mouth closed and elevates his chin, the downward tug can in many cases be detected with great facility. The phenomenon can

also be observed when the finger-tips are applied to the cornua of the hyoid; and the patient is put to less discomfort when this method is adopted.

Pulsations and thrills may be observed in the carotids and must be fully investigated by palpation. Occasionally a thrill may be felt in the supraclavicular fossa, where the subclavian artery crosses the apex of the lung. It may indicate a constriction of the vessel resulting from pleuritic adhesion, or disease of the lung itself.

In the epigastrium the fingers should be pressed gently but firmly upwards under the left costal margin when pulsation of the right ventricle is suspected. By this means it may be readily differentiated from pulsation of the liver.

When the **liver exhibits expansile pulsation,** owing to backward pressure in the veins due to tricuspid incompetence, the whole organ will be found to be affected, and in most cases the expansile character of the movement can be distinctly made out. The pulsation is most readily recognized by placing one hand on the 5th and 6th costal cartilages, and the other on the lateral region of the liver in the midaxillary line. When the right ventricle, by pressing on it during systole, causes epigastric pulsation, the movement can rarely be detected, except in a part of the liver. In cases of doubt as to the nature of epigastric pulsation, a change in the patient's posture, particularly if he is made to assume a knee-elbow position, frequently clears up the difficulty.

IV. PERCUSSION

Percussion determines—

(A) **The boundaries of the heart and surrounding viscera.**

1. Deep dullness.
2. Superficial dullness.

(B) **The presence of certain abnormal conditions of the cardio-vascular system,** e.g.—

(*a*) Pericardial effusion.

(*b*) Aneurysmal dilatations.

Theory.—When a sharp tap is given over any part of the body, the underlying structures either resound to the blow or merely respond with a dull thud like that which a lump of putty would yield under similar conditions. The former bodies are described as resonant, the latter as dull, on percussion. The resonant structures in the body are the air-containing organs and the bones. The latter when struck emit a sound that is totally distinct from that yielded by the viscera which contain air. It is known as the osteal percussion sound, and is typically heard when the skull is lightly tapped. The cause of resonance is that such bodies as possess it are able to vibrate with more or less regularity. In the case of the bones this power of regular vibration is due to the elasticity of the osseous substance; in the case of a hollow viscus, such as the stomach, colon, or small intestine, it depends on the periodic oscillations of the contained air columns, and on the tension of the limiting stomach or bowel wall; in the lung the factors become more complex, for we have to deal with very greatly subdivided air columns, and the septa are under a considerable degree of tension. Since the exact quality of the resonance which is produced is influenced by the amount, disposition, pressure, and subdivision of the included air, and also by the tension of the walls and septa of the viscus, it is evident that each organ will, on percussion, give out a sound which is fairly characteristic. With a comparatively simple air-space such as the stomach presents, the resonance resembles that of a drum, and is therefore described as *tympanitic*; in the lung, on the other hand, the innumerable septa so break up the air-space that the resonance acquires a peculiar and quite distinctive character. There is no other sound which bears any close resemblance to it; hence it is simply known as *normal lung resonance*, and the student must learn to recognize it from frequent practice.

The depth of tissue which is thrown into vibration by the percussion stroke depends, when other things are equal, on the force of the blow. When the chest wall over two regions of the lung is percussed with equal force, and when in one case a considerable depth of lung tissue underlies the point, whilst in the other only a thin layer of lung intervenes between the surface and a subjacent solid organ, a characteristic difference is observed in the sound produced, just as, when a big drum

and a little one are struck, they emit perfectly distinct sounds. The difference of sound depends on several factors, the important points to note being that the thick layer of lung and the big drum emit a sound which lasts longer and conveys an impression of greater resonance and lower pitch. The thin layer of lung and the little drum, on the other hand, yield a sound of shorter duration, of less resonance, and of higher pitch. The exact pitch, however, is always somewhat indeterminate, as it is compounded of a mixture of several independent, more or less tone-like, sounds and their overtones. For practical purposes we may describe the resonance of a thick layer of lung as "*full*," and that of a thinner layer as "*emptier*," the words being employed, in a general but sufficiently intelligible sense, to describe the complex impression which the observer readily recognizes, but which, under the conditions of clinical work, baffles exact analysis.

When a very light blow is delivered the resonance of only a thin layer of the subjacent lung is elicited, and the sound produced is comparatively empty, even although a thick part of the lung lies below the point of percussion. Hence it follows that the presence of a solid organ underneath the lung will only render the resonance "emptier" when it approaches sufficiently near the surface to encroach upon the layer which is being set in vibration. A firmer stroke would throw a thicker layer into vibration, and in this case a solid body advancing from below would sooner reach the vibrating area and render the resonance emptier. In other words, when the object aimed at is to detect the presence of an organ which lies underneath a thick layer of lung, then the percussion stroke should be firm. If, on the contrary, the object of inquiry is to ascertain the spot where a resonant viscus terminates, more especially if it grows thin, wedgewise, as its border is approached, it is evident that the end in view will be best achieved by very light percussion, since by this procedure the resonance, though comparatively empty, remains of uniform quality until the edge of the organ is reached, when it is replaced by an absolutely dull thud.

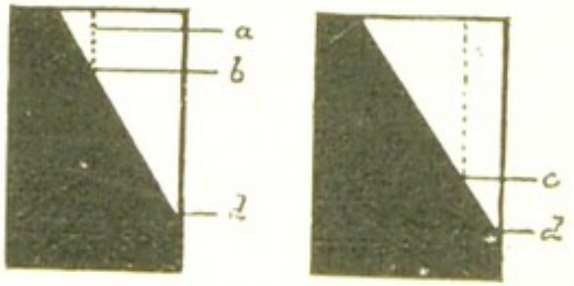


Fig. 18.—Percussion diagram (*see* text).

This is diagrammatically represented in the accompanying figure (Fig. 18), where it is easily seen that with firm

percussion the resonance begins to grow emptier at *b*, and gradually alters thereafter at every point till the emptiness is completed at *d;* whilst with lighter percussion the resonance remains uniform, although comparatively empty, until *c* is reached, when it rapidly gives place to absolute dullness, and thus the final extinction of resonance is much more readily appreciated.

If the tight membrane which is stretched over a drum be covered with a soft solid, the sound which it yields is muffled; and, in like manner, if a solid organ overlies an air-containing one, the resonance of the latter will be thereby *muffled*, and the more softly the percussion stroke is delivered the more marked will the muffling become. A soft stroke is particularly necessary when the solid organ is thin, or when its border requires to be accurately defined.

The following points also demand consideration in the practical application of percussion. First, the percussion stroke being delivered perpendicularly to the surface, the percussion outline may be greater than the actual width of the organ under examination when the surface is curved, as may be seen in Fig. 19, where the dotted lines represent the true projection, and the continuous lines the percussion outline of a solid organ. Second, since the percussion stroke not only affects the structures directly below its point of application, but elicits resonance from a region extending to a certain distance around that point, a certain degree of dulling may occur before the solid organ is reached. This error becomes greater as the percussion stroke is increased in force.

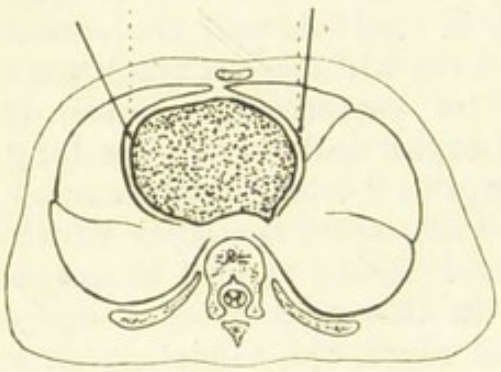


Fig. 19.—*See* text.

Methods of percussion.—When percussion was first introduced, the tap was delivered directly on the patient's skin without the interposition of any substance over the point struck. This method, known as *direct percussion*, is now seldom used except on the clavicles, which in examination of the lungs are lightly tapped by the observer's finger-tip. In order to obtain better resonance, as well as with a view to

the patient's comfort, various materials were subsequently interposed between his skin and the percussing finger. A flat plate of bone or ivory, of such a size and shape as to be readily applied and closely adapted to the surface of the chest, is frequently employed, and is called a *plexímeter*. In some instances, when the curvature of the surface renders the application of an instrument difficult, a short rod of bone or vulcanite, set pillarwise on the region to be percussed, is employed. A very good pillar pleximeter may be improvised by utilizing a short piece of good cork, such as is used for closing specimen tubes. It conveys the impact well, and itself emits but little sound when struck. Most physicians, however, prefer to make use of the middle or forefinger of their left hand as a plexímeter, and the preference is due not only to the fact that it can be readily adapted to almost any surface, but also that it often conveys information additional to that obtained by the percussion sound, as it takes cognizance of the different degrees of resistance which the tissues offer to the percussion stroke.

Sometimes a small rubber-tipped hammer, known as a *plessor*, takes the place of the percussing finger, and is occasionally of service ; but as a rule the finger should be preferred.

The ordinary method, then, of percussion is conducted in the following manner : The middle finger of the left hand is placed firmly on the part which is to be percussed, and is adapted to any inequalities of surface, so that no air-space is interposed between it and the skin. The back of its middle phalanx is then struck with the tip of the middle finger of the right hand. The stroke should be delivered from the wrist and finger-joints, not from the elbow, and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the

metacarpal bones, and strikes the pleximeter perpendicularly. Whenever the blow has been given, the striking finger must be raised, lest it should impair the vibrations it has excited, just as the hammers of a piano fall back from the wires as soon as they have been struck. In cases where the percussion requires to be firmer, several fingers may be used; but it is better, whenever possible, to employ only one percussing finger. In some cases a modification, known as flicking percussion, is useful, and this is particularly valuable in the examination of the abdomen, where the method is more fully described (p. 61).

There are **three cardinal rules** which should always be remembered when percussion is being carried out. The **first** is that in defining the boundaries between contiguous organs the percussion should invariably be performed from the resonant towards the less resonant. The **second** is that the longer axis of the pleximeter should be parallel to the edge of the organ whose delimitation is being attempted, and the line of percussion should be at right angles to that edge. The **third** is that the pleximeter finger must be kept in firm contact with the chest wall.

It is seldom necessary to deliver more than two or three strokes at any one situation; repeated blows cause much discomfort to a sensitive patient.* The points to be noted on percussion are the *volume* and *pitch* of the resonance elicited, and the sense of *resistance* experienced by the finger.

Percussion of the Heart

The objects which are aimed at in percussion of the heart are twofold: first, to ascertain the size and

* Students who lack expertness in percussion are advised to gain the necessary skill on a table or some other inanimate object of varying resonance rather than on hospital patients.

position of the organ as a whole; and, second, to determine how much of it is uncovered by lung and lies against the chest wall.

Since for the most part the heart is surrounded by resonant lung, but does not lie so deeply as to be out of reach of a firm percussion stroke, one can delimit its extreme boundaries with a considerable degree of accuracy by observing, as one percusses towards the cardiac region, the points at which the lung resonance begins to grow emptier. In two areas this cannot be achieved. At the base of the heart the roots of the great vessels produce a dulling or emptying of the lung resonance which cannot be discriminated from that caused by the heart,* while the lower border of the viscus is in relation to non-resonant liver, which yields on percussion the same dull thud that the heart itself does.

It is also important to determine how much of the heart is quite uncovered by lung. This is done by continuing to percuss in the same direction as for the determination of the extreme limits of the organ, but with a lighter stroke, and observing when the slight resonance of the lung is replaced by absolute dullness.

The region of the heart covered by lung is called the *area of deep or relative cardiac dullness*. Its outline corresponds approximately to the anatomical outline of the organ. The region of the heart which is uncovered by lung and lies directly against the chest wall is called the *area of superficial or absolute cardiac dullness*.

The accompanying diagram (Fig. 20), which represents an antero-posterior section of the chest in the left parasternal line, will explain the sequence of

* The vagueness of the results obtained in this region is still further increased by the "sounding-board" property of the sternum.

phenomena which are observed on percussion. At *a* the resonance is full, at *b* it begins to grow emptier, and continues to do so till *d* is reached, when the sound becomes absolutely dull, and continues so over the uncovered surfaces of the heart and liver until *e*

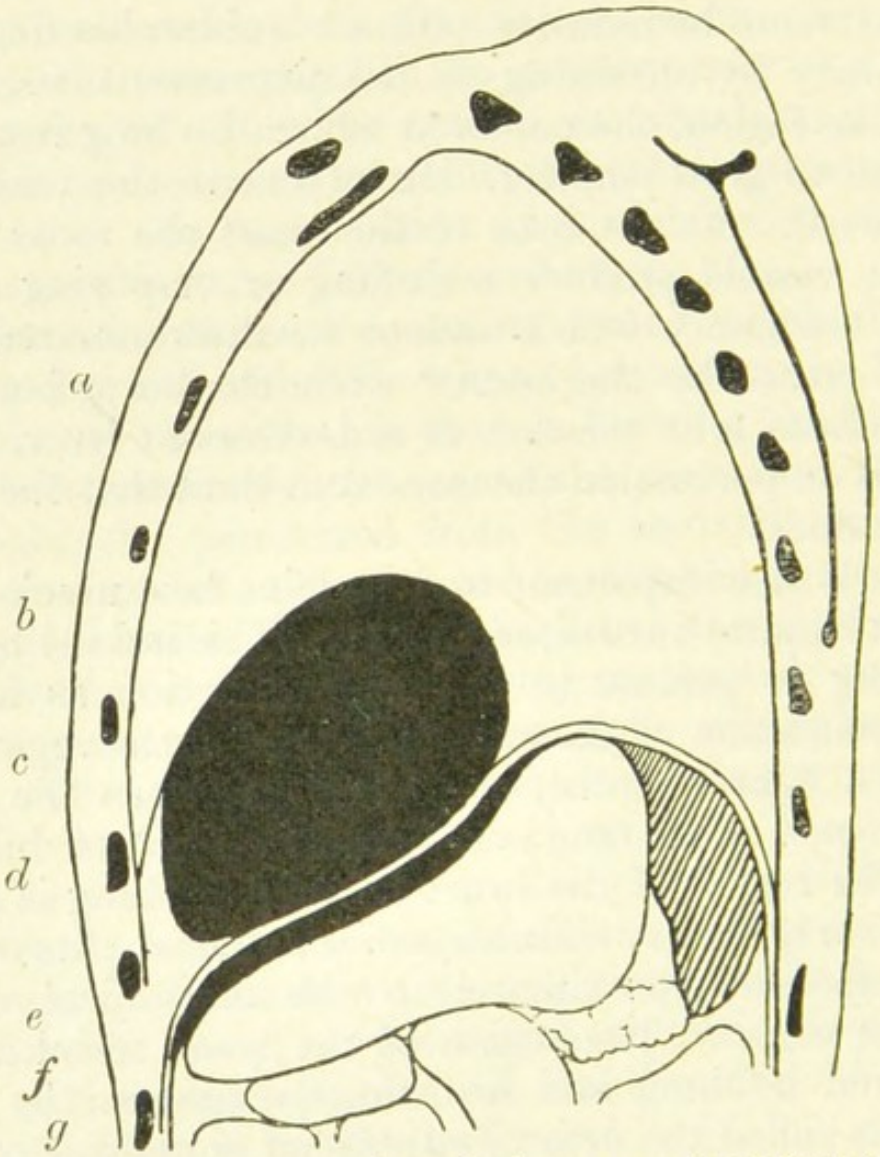


Fig. 20.—Antero-posterior section of thorax near the left parasternal line. (*See* **text.**)

(*Slightly modified from Luschka.*)

is reached, when the resonance of the stomach, though muffled, may be distinctly detected, and at *f* the muffled resonance gives place to the full tympanitic stomach note. The percussion stroke must be firm

in passing from *a* to *b*, light from *c* to *d*, and light also as one percusses upwards from *g* to *e*.

1. **Deep dullness.**—By firm percussion, then, the right, the left, and that part of the upper border of the heart which lies to the left of the roots of the great vessels can be defined. To do this, percussion is performed in two directions: first, down a vertical line far enough from the middle plane to be quite to the left of the great vessels, but not so far out as to miss the upper border of the heart altogether—the left parasternal line, or one a shade internal to it, fulfils these requirements; second, percuss from right to left along a line as far down the chest as possible, but yet clear of any trace of hepatic dullness; and also along the continuation of this line to the left of the heart, but this time percussing from left to right. In the vertical line one should begin at the 1st interspace, comparing its resonance with that of the 2nd, and this with the 3rd, and so continuing downwards until the first trace of impaired resonance is observed. One then knows that the boundary of the heart has been reached. But the dullness may also be present at the level of the rib above the interspace in which it was detected, and therefore the percussion sound of this rib must be compared with that of the one next above it. If the resonance of the lower of the two be less full than that of the upper, one knows that the outline of the heart lies behind it. The reason why rib is compared with rib, and interspace with interspace, is that otherwise the alternate presence and absence of the osteal resonance, according as rib or interspace is being percussed, introduces an element of unnecessary confusion. Before percussing the right border of the heart, the upper limit of deep hepatic dullness between the right parasternal and the right mammary lines must be determined, just as the upper

border of the heart was. When this has been done, the right border must be defined by percussing from the right mammary line towards the sternum along the rib or interspace above the level at which the first traces of hepatic dullness were observed.

Although the lower border of the heart cannot be percussed out, a sufficiently close approximation to it

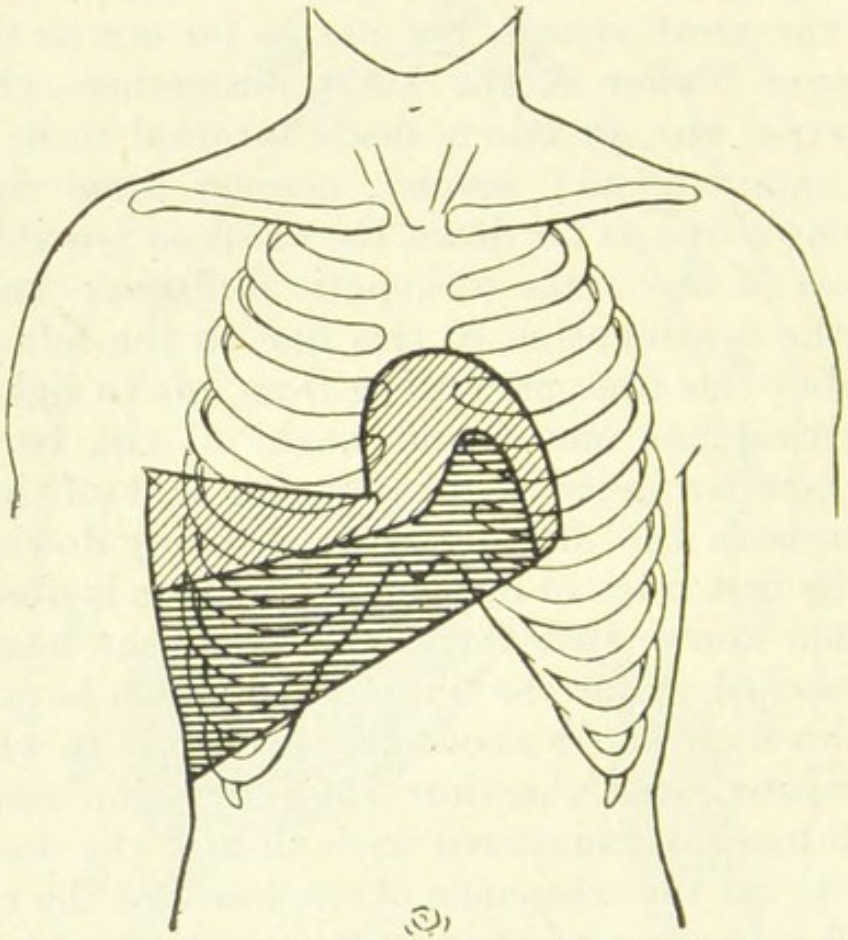


Fig. 21.—Superficial and deep dullness of normal heart and liver.

is attained by drawing a line from the upper limit of deep hepatic dullness, which has already been determined, and which is usually found at the level of the 4th interspace or 5th rib, to the apex, whose position has been fixed by palpation.

By percussing in the 4th interspace from the left lung towards the heart, one is able to define the left border with considerable precision, and in cases where

it is desirable to obtain further points one can percuss in various other lines perpendicular to the border from the lungs towards the heart. The student must not confuse the slight muffling of the lung resonance which occurs when he arrives over the pectoral muscles, with the deep dullness of the heart. It will be found that in an average healthy chest the percussion limits of the heart are as follows (Fig. 21) :—

Upper border (in left parasternal line), at the 3rd rib, or upper border of the 3rd interspace.

Right border (at level of 4th rib) is just to the right of the right lateral sternal line. If, however, the lungs are somewhat voluminous, it may be impossible to detect the impairment of resonance at this point, and behind the sternum accurate observations are not very easy to make, as the sternum acts as a sounding-board, and collects vibrations from all the structures behind it, whether they lie directly beneath the point of percussion or are at some distance from it.

Left border (at level of 4th interspace), a shade internal to the mammary line. If it is percussed at a higher level it will be found to curve round so as to merge insensibly with the upper border.

Dr. Graham Steell has proposed the following convenient method of recording the position of the various boundaries of the heart : A vertical line is drawn, with a horizontal one lying across its upper end like the letter T. Above the horizontal line is recorded in Roman numerals the number of the rib to which the upper border of the heart extends in parasternal line. Below it, and to the writer's left of the vertical line, the distance to which the right border of the heart passes to the right of the mesial plane is noted in Arabic numerals which represent inches, whilst to the right of it the position of the left border of the heart is similarly indicated. The right border of the

heart is determined along a line just clear of the liver dullness, the left immediately above the situation of the apex beat. Thus the record of an average healthy adult heart would be

$$\frac{\text{III}}{1 \mid 3\frac{1}{2}}.$$

2. The **superficial dullness of the heart,** which depends on the position of the borders of the lungs, must be determined by light percussion. To ascertain the upper border, one should percuss downwards between the left lateral sternal and left parasternal lines. The left border is found by percussing from the left mammary towards the middle line along the 4th intercostal space, or 5th rib; the right, by light percussion at the same level, beginning to the right of the sternum. In health it will be found that the upper limit is at the level of the 4th rib. The left at its upper end is rather more than $\frac{1}{2}$ in. within the left border of the heart, as already determined; at its lower end it is decidedly nearer that border, and may extend outwards almost to the apex beat. The right limit does not correspond with the edge of the right lung, which, placed as it is behind the sternum, cannot be exactly defined, but lies in the left lateral sternal line, where it extends from the 4th to the 6th costal cartilage. The left limit curves gradually round to be continued into the upper, the latter joins the right limit at an angle. The space is therefore of triangular outline, but the left side of the triangle is not straight, but convex outwards. The lower side of the triangle cannot be defined by percussion, but corresponds with the inferior border of the heart; it is marked out in the manner already described.

Lying as they largely do behind the sternum, the dullness due to the great vessels can seldom be made out with precision. If, however, there is aneurysmal

dilatation of the ascending aorta, a dull area can be mapped out. It is continuous below with that of the heart, above it bulges outwards to the right of the sternum at the level of the 2nd interspace and adjacent ribs ; whilst the sound produced by percussion of the manubrium sterni is also rendered much less resonant, or even, in cases where the aneurysm is large, absolutely dull (Fig. 22).

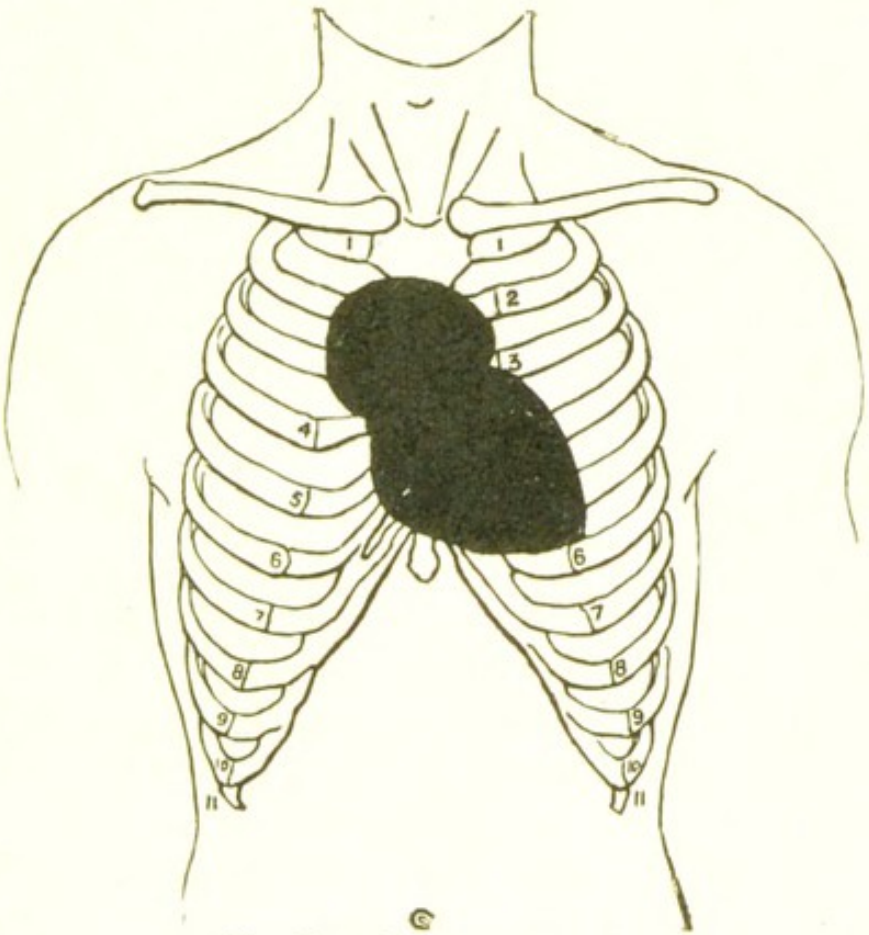


Fig. 22.—Aortic aneurysm.

ALTERATIONS IN CARDIAC AREAS IN DISEASE

In diseased conditions both the relative and the absolute cardiac dullness may be altered in size or in position. Except in cases of pericardial effusion the determination of the area of superficial dullness seldom yields any information of great importance regarding the heart ; but it is valuable in revealing certain diseased conditions of the lungs and pleura.

When the relative or deep dullness is enlarged the condition may be due either to disease of the heart and pericardium, or to morbid conditions in the surrounding viscera. If the dullness in the left

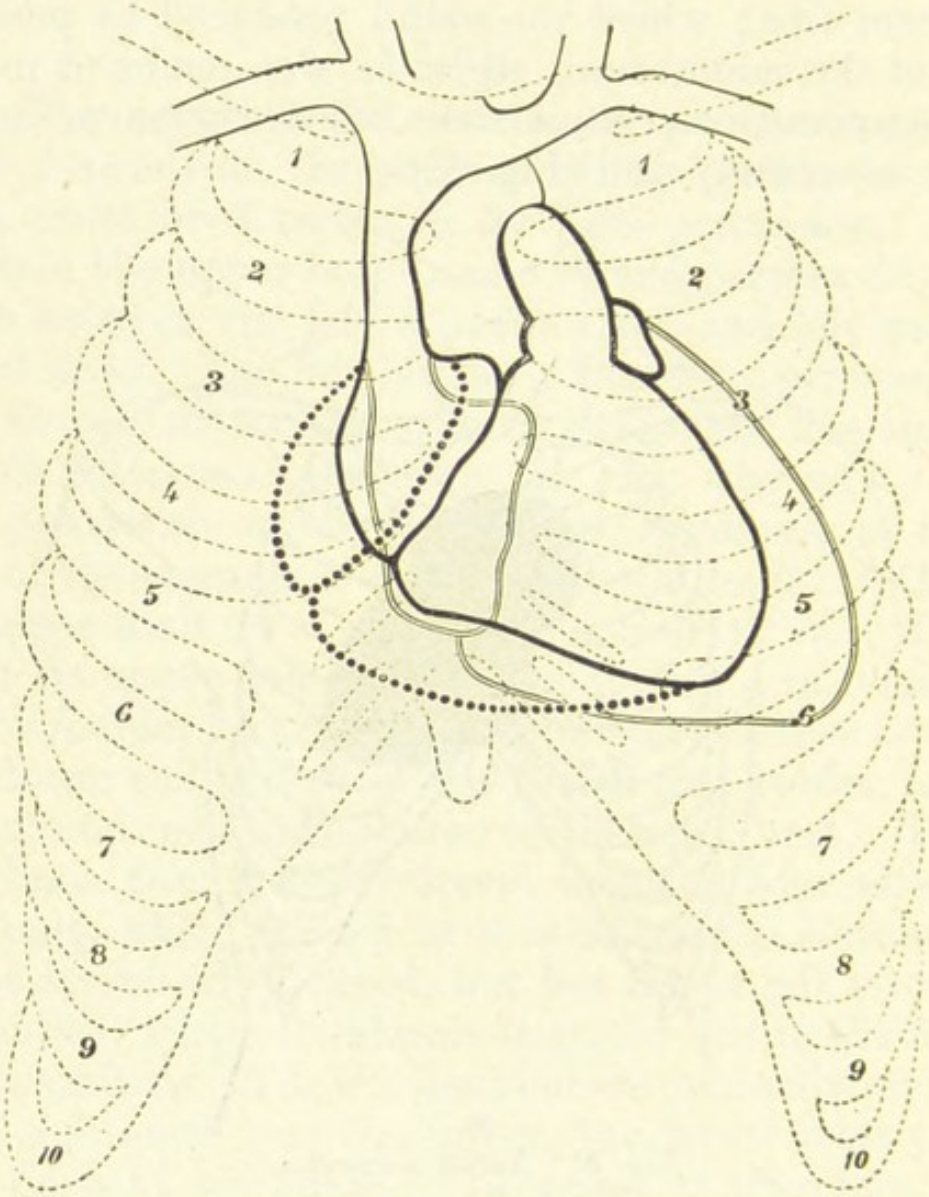


Fig. 23.—Diagram to illustrate the effect of dilatation of the right and left sides of the heart respectively. (*Gee, after v. Dusch.*)

Continuous heavy outline, normal heart; dotted line, dilatation of right side; thin double outline, dilatation of left side.

parasternal line be found to extend upwards into the 2nd interspace or higher, without any corresponding upward displacement of the lower boundary, such as would be present were the heart dislocated **upwards** as a whole, and in the absence of disease of the lung,

the condition is usually due to *pericardial effusion.* Aneurysm of the descending arch of the aorta has been known to cause dullness in this region, but this effect is only produced in the rare instances where the aneurysm passes far forwards. Most aortic aneurysms are found farther to the right.

If the relative dullness extends to the **left of the**

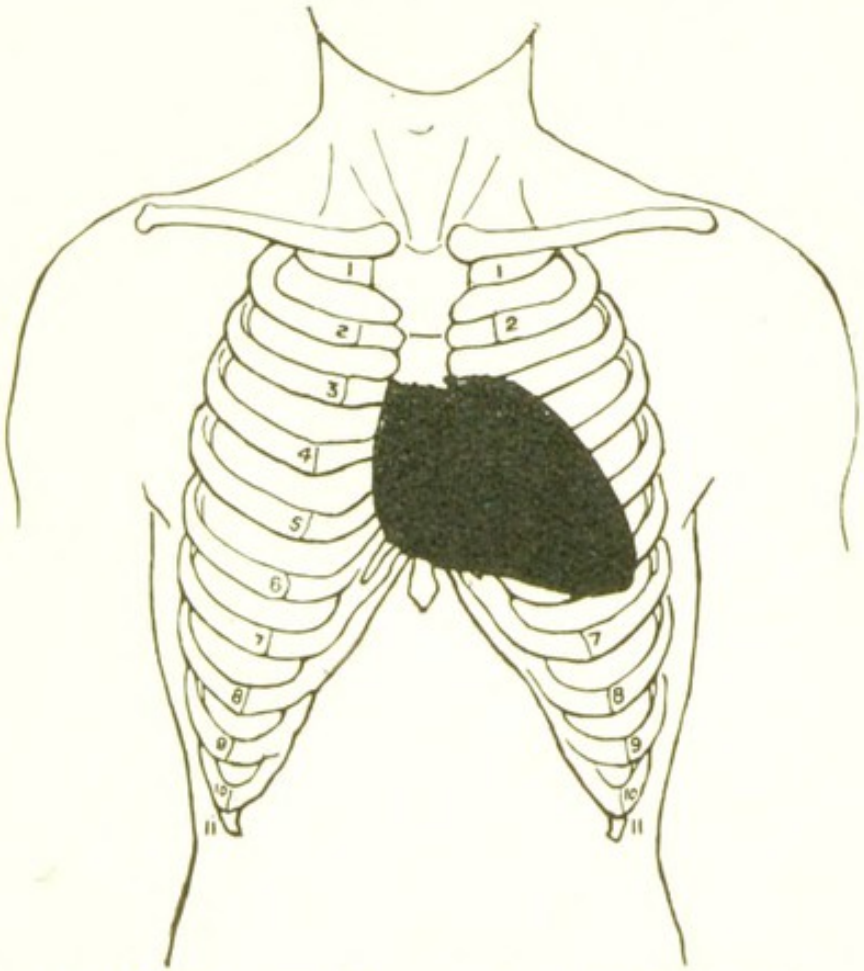


Fig. 24.—Dilatation and hypertrophy of left side of heart.

apex beat, provided the lung and pleura be healthy, we have to do with pericardial effusions; and in this case the right border will be found at a considerable distance to the right of the sternum—it may be as far as the right parasternal line. If the cardiac dullness extends to the left of the mammary line, but does not reach beyond the situation of the apex beat, the condition is probably due to dilatation and hypertrophy

of the left ventricle, unless the heart is bodily dislocated to the left by some such cause as massive pleural effusion on the right side. If the dullness extends, in the absence of lung disease, pleurisy, or pericardial effusion, more than about a finger's breadth to **the right of the sternum,** one is justified in concluding that the right heart has become dilated (Fig. 23).*

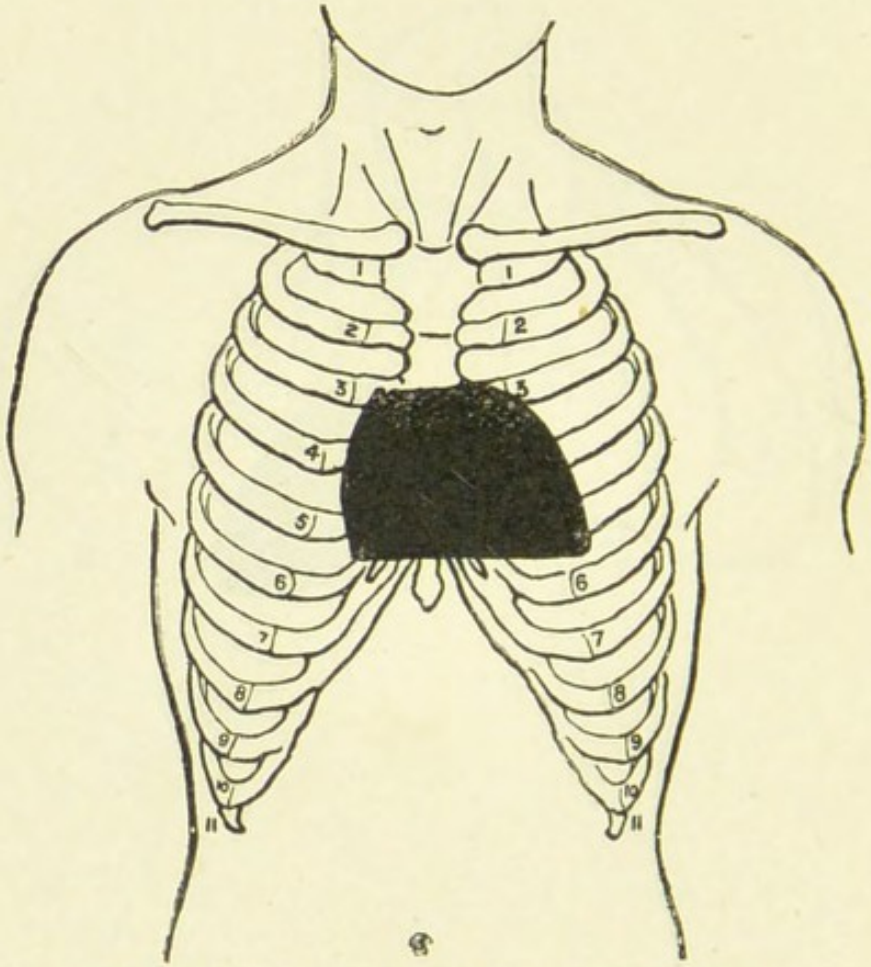


Fig. 25.—Dilatation of right side of heart.

Since dilatation and hypertrophy of the left ventricle not only displace the left border outwards but cause depression of the apex, the percussion outline of such a heart will become conical (Fig. 24), whilst dilatation of the right side, by causing the cardiac dullness to extend too far to the right without greatly

* The auricle whose boundary is thus determined is dilated the ventricle is usually both dilated and hypertrophied.

affecting the level of the apex, renders the dull area more square than usual (Fig. 25).

The dullness which one finds in *pericarditis with effusion*, or in *hydropericardium*, varies with the amount of fluid which is present, but in well-marked cases is pear-shaped, with the broader end downwards and the upper end higher than the ordinary upper limit of dullness (Fig. 26).

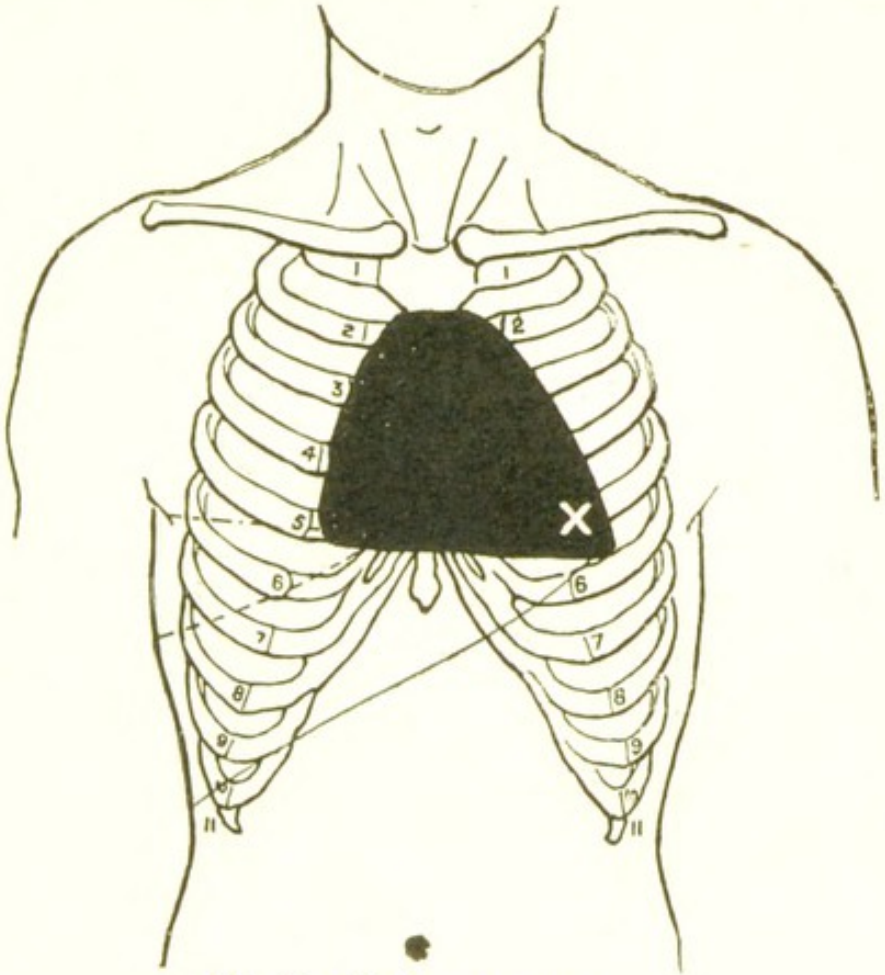


Fig. 26.—Pericardial effusion.

The chief causes outside the heart and pericardial sac that produce an increase in the area of cardiac dullness are due to diseases of the lungs and pleura. Here one may find consolidation or tumour of the lung, or pleural effusion ; or a cirrhotic contraction of the lung, or the binding of it back by pleuritic adhesions, may leave more than usual of the front of the heart exposed,

or at least near the surface. In comparatively rare instances the heart is pushed forwards by a tumour or aneurysm in the posterior mediastinum.

The relative dullness of the heart may be diminished in cases where the heart is unusually small, or the lungs so emphysematous as to interpose a layer of pulmonary tissue that is thick enough to

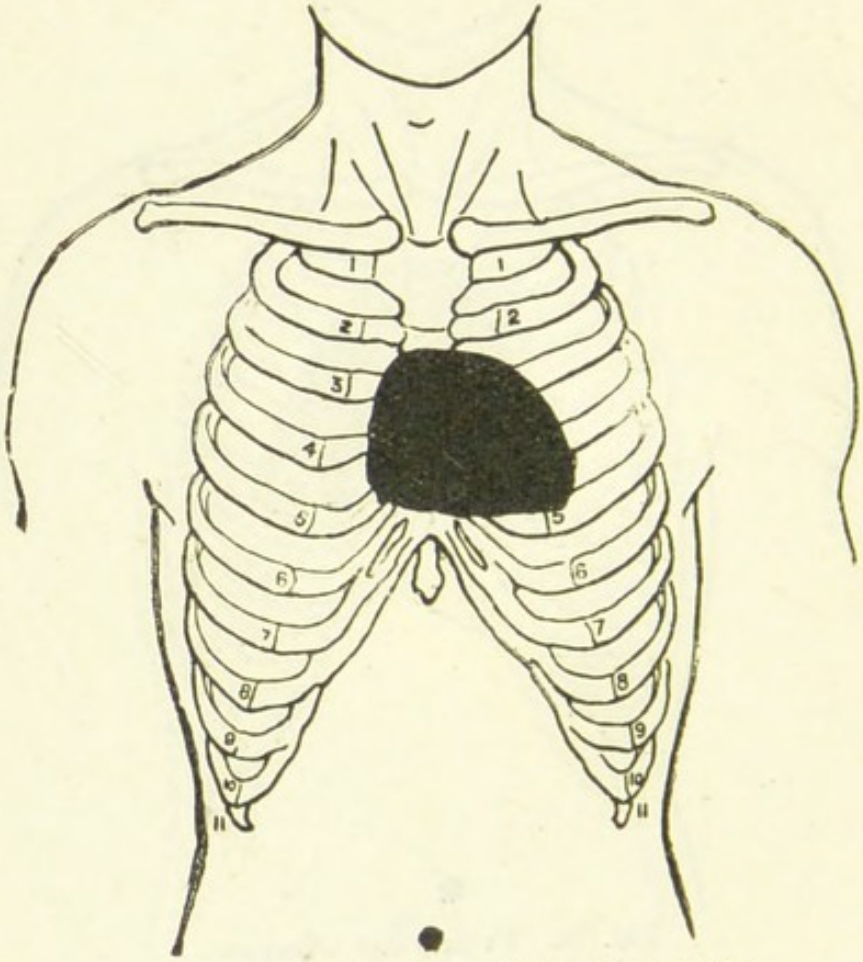


Fig. 27.—Displacement of heart upwards.

carry the margins of the heart beyond the sphere of action of the percussion stroke. As a corollary to this, it should be observed that if, when the lungs are emphysematous, the heart's dullness reaches fully up to the normal limits, one is justified in assuming that, as a matter of fact, it oversteps these limits. Air in the pleural cavity will also diminish the area of dullness; in pneumopericardium it is often quite abolished.

It is well to preserve a note of the breadth, in inches, of the cardiac dullness at the level of the 4th rib or interspace.

The area of absolute or superficial dullness is of less importance, being much affected by the state of the lungs. It is thus increased when they are retracted, decreased or almost abolished when they

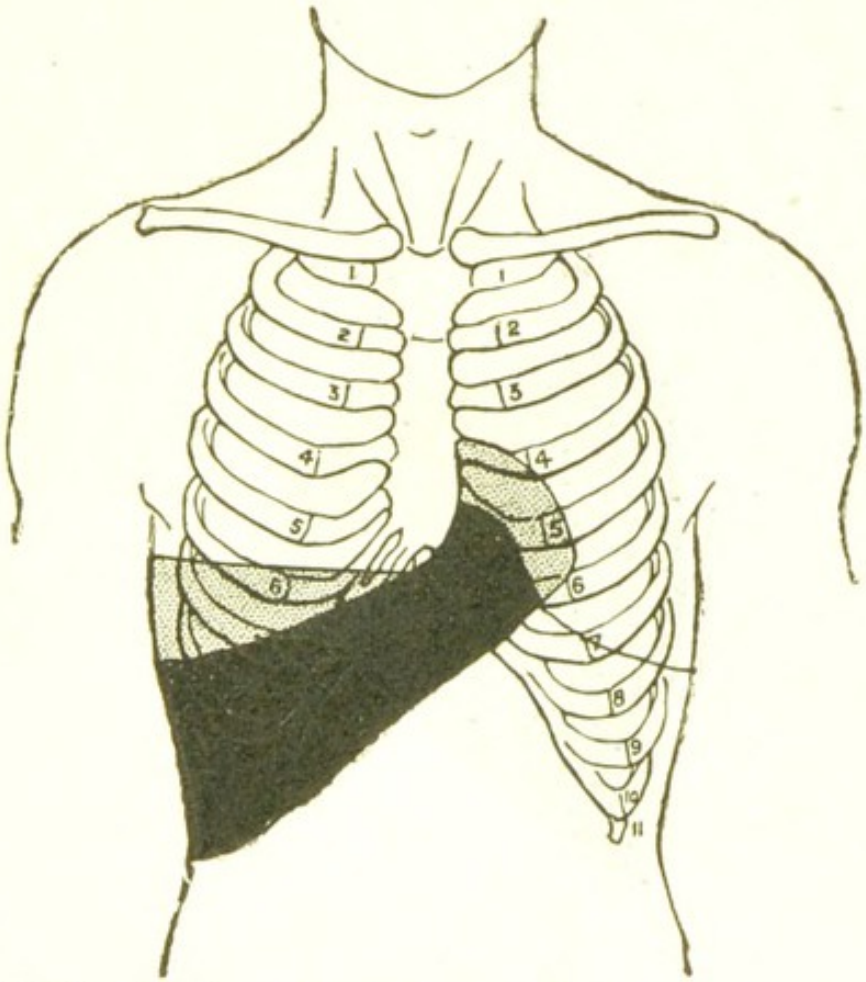


Fig. 28.—Displacement of heart and liver in emphysema.

are distended, as in advanced emphysema ; otherwise, it is altered by the same conditions and in somewhat the same manner as the area of relative dullness.

The situation of the area of cardiac dullness is naturally altered by **changes in the position of the heart.** These result from its displacement by the pressure or traction of other organs, or from developmental anomalies. Thus in dexiocardia the heart is

placed with its apex to the right, and the area of dullness is then the mirror image of what is usually found.

In cases of ascites or of massive abdominal tumour the heart is pushed upwards under the lungs. Hence its area of dullness is placed higher than usual, and, owing to the thickness of overlying lung, may be unusually difficult to map out (Fig. 27). Tumours of

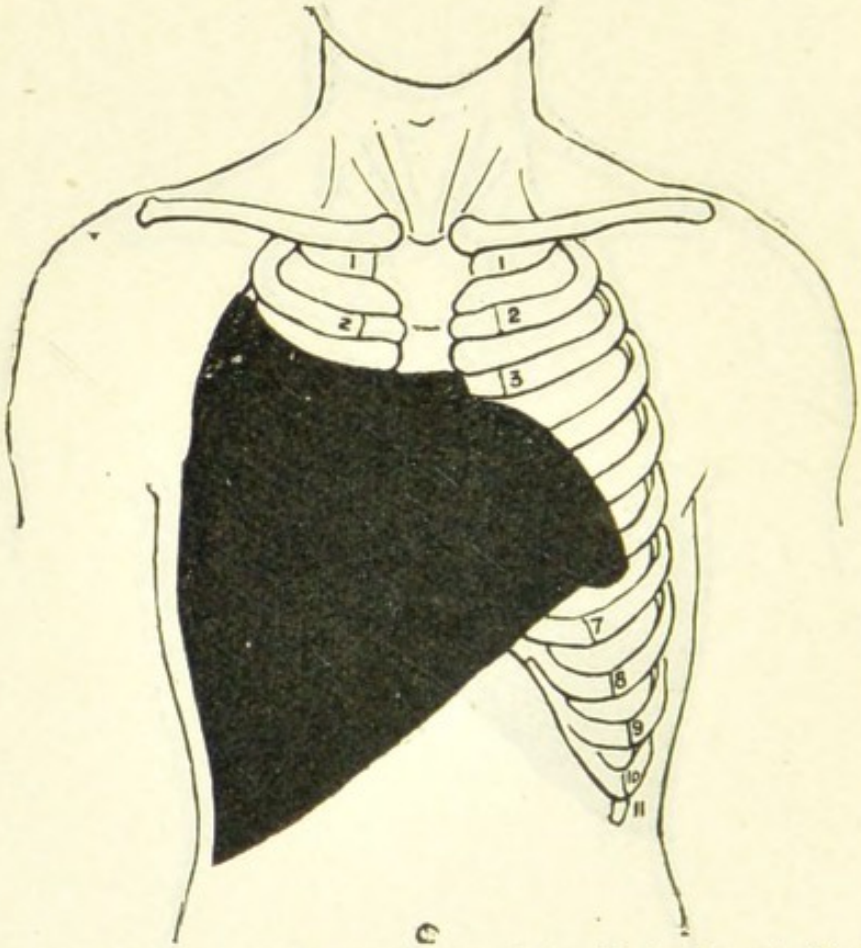


Fig. 29.—Displacement of heart in right-sided pleural effusion.

the liver displace the heart upwards and to the left. Pulmonary emphysema thrusts the heart downwards (Fig. 28), pleural effusion drives it towards the sound side of the chest (Figs. 29, 30), whilst cicatricial contraction of the left lung often draws it upwards and to the left.

In certain cases of pericardial effusion it is stated that a small dull area can be found posteriorly near

the angle of the left scapula. The phenomenon is of doubtful value, and has not been very satisfactorily accounted for. Those who wish to study it must refer to larger works or to special papers on the subject.

Whilst the student is percussing the heart, he should attend not only to the resonance which is elicited, but also to the **sense of resistance** of the

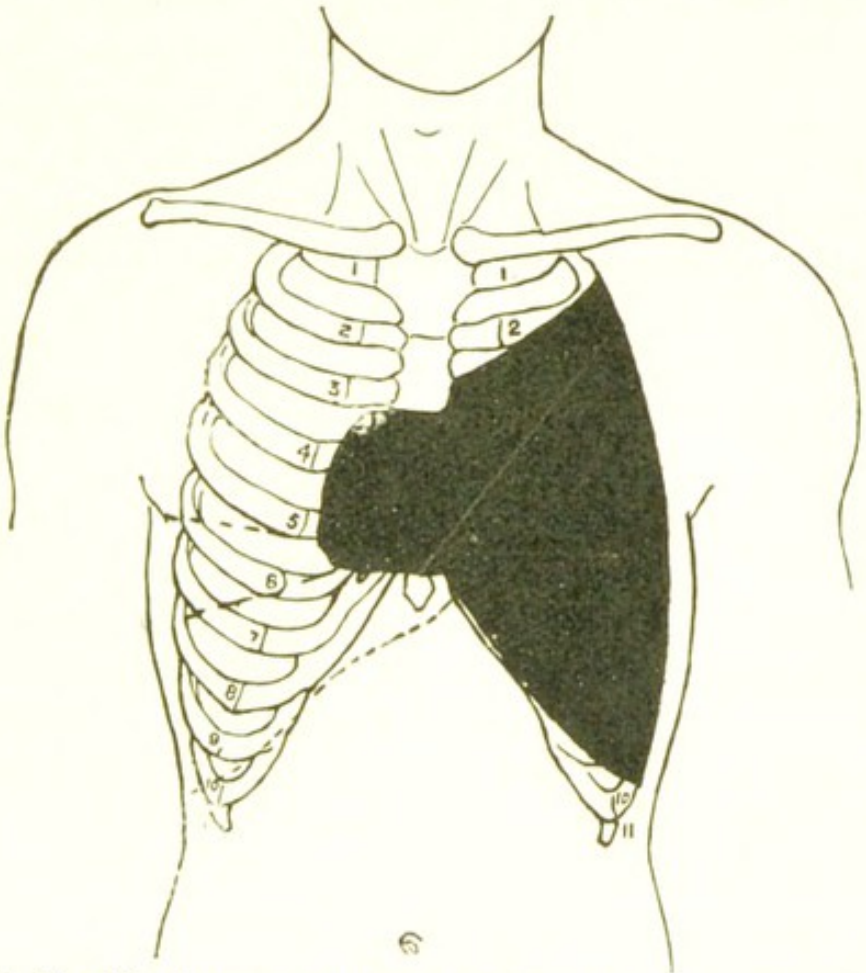


Fig. 30.—Displacement of heart in left-sided pleural effusion.

underlying tissues which the pleximeter finger experiences. By this means he may often form a shrewd guess of the nature of the subjacent structures. For example, a well-trained finger will detect decidedly greater resistance over a dull area when the dullness is caused by fluid, as in pleurisy or hydropericardium, than when it is produced by a solid organ like the heart or a pneumonic lung.

V. AUSCULTATION OF THE HEART AND VESSELS

Auscultation determines—

(A) **Character of the heart sounds** with respect to—

1. Intensity.
2. Rhythm.
3. Quality.

(B) **Abnormal sounds associated with the heart sounds**—

(*a*) Over præcordia { Endocardial murmurs. / Pericardial friction.

(*b*) Over vessels { Clear sounds. / Murmurs or bruits.

The stethoscope.—Auscultation, though sometimes performed by the direct application of the ear to the chest wall, is generally conducted by means of a stethoscope, and the student cannot take too great pains to choose a good one.

Stethoscopes are of two types, single and binaural. Each of these has its special advantages and disadvantages. Binaural instruments are particularly serviceable in the examination of children and of patients too ill to be much disturbed. Single ones, although they conduct less loudly, are relatively rather more sensitive to high-pitched sounds, and they also convey to the ear of the observer, when he listens over the apex of the heart, a distinct shock or jog at the moment of ventricular systole, thereby facilitating the timing of other phenomena.

In the choice of a binaural, one should avoid instruments with unnecessary joints and loose parts, or with woven tubes. The chest-piece should not be very large, nor made of metal; vulcanite is not so chilly, and is easily cleaned. Unless the ear fittings are suitably shaped, much discomfort will be produced.

In choosing a single stethoscope much depends on the grain of the wood; some woods, such as oak, will

be found to conduct better than others. The chest-piece should be of moderate size, so that it may be in complete apposition with the chest wall even when the patient's ribs are prominent. The ear-piece should be selected so as to fit the ear with comfort. The instrument should have no loose parts. A good rough-and-ready test of the efficiency of a stethoscope is to listen with it to the ticking of a watch, and to select the instrument which conducts the sound best. In practice the student must be very careful not to press heavily on the patient when using a single stethoscope.

The *phonendoscope* has not secured any very general recognition. This may be due in part to the fact that, though it conveys low-pitched sounds with great clearness, it does not conduct high tones so well as the ordinary stethoscope ; and, since high tones are peculiarly characteristic of many morbid conditions in the lungs, it is obvious that its use for clinical purposes is seriously limited by this defect.*

The cardiac cycle and surface anatomy of the valves and vessels.—In order to understand the various sounds which can be heard by listening to the heart through the chest wall, a clear conception of the events which occur during a cardiac cycle is essential.

After the completion of a beat the auricles and ventricles are both relaxed. Thereafter the auricles contract, forcing their contents through the cuspid valves into the ventricles, and filling them. The ventricles then contract in turn, expelling the blood into the

* A special stethoscope provided with an adjustable slit has been devised for the purpose of comparing the intensity of the different heart sounds at varying points. By adjusting the opening until the sound under examination becomes inaudible, one can obtain a definite measure of its intensity at any given point. The best form of this instrument is Bock-Oertel's stethophonometer, which is a modification of the pattern originally suggested by Gärtner.

vessels, whilst the auricles commence to relax and become refilled with blood; finally, the ventricles relax also, and so the cycle is completed. We have thus in rapid succession auricular systole, ventricular systole, and ventricular diastole; the auricular diastole commencing during ventricular systole, and ventricular diastole continuing through auricular systole.

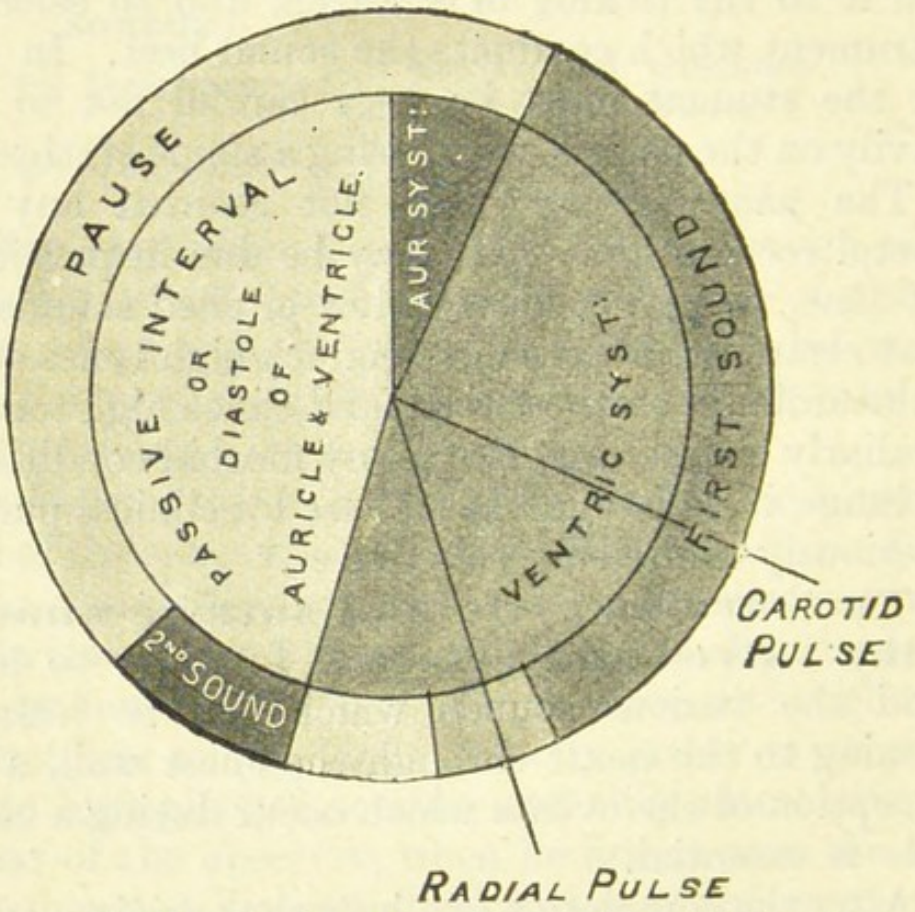


Fig. 31.—Cardiac cycle. (*Modified from Gairdner.*)

The beginning of ventricular systole is marked by the closure of the mitral and tricuspid valves, which had remained open during the systole of the auricles, and by the occurrence of the apex beat; the beginning of ventricular diastole is marked by the closure of the aortic and pulmonary valves, which remain closed until the beginning of the following ventricular systole. The pulse in the carotid occurs a short time after the commencement of ventricular systole; in the radial

artery it is decidedly later in its appearance, and therefore the radial pulse must never be taken as an index to the commencement of ventricular systole. The carotid pulse is less fallacious than the radial, but, as an index to the commencement of systole, preference should be given to the apex beat whenever it is available.

Various authors have constructed diagrams to represent the sequence of events in a cardiac cycle. The accompanying one (Fig. 31) may be taken as representing these in an ordinary case, though the relative duration of the successive events will be found in practice to vary within fairly wide limits. The most important variation is, that when the heart acts with unusual rapidity the duration of diastole is curtailed to a greater degree than that of systole, and hence a shorter interval elapses between the time of closure of the semilunar valves and the commencement of ventricular systole than one would infer from an examination of the diagram. For some purposes it is found more convenient to unroll the above diagram, so that the sequence is represented along a straight line instead of round a circle. Fig. 32 shows the details of the cardiac cycle, and especially their exact time relations, with greater accuracy than the simpler diagram of Professor Gairdner. It will be found of special value when the student arrives at the consideration of the venous pulse.*

* It must be recollected that in clinical language the words systolic and diastolic are used with reference to the state of the ventricles, events which take place during the auricular contraction being described as diastolic (or presystolic). On the other hand, physiologists generally regard the period of auricular contraction as included in the systolic period of the cardiac cycle. Clinically, then, the systolic phase of the cycle begins with the apex beat and commencement of the first sound. It terminates immediately before the second sound, whose commencement marks the beginning of the diastolic period.

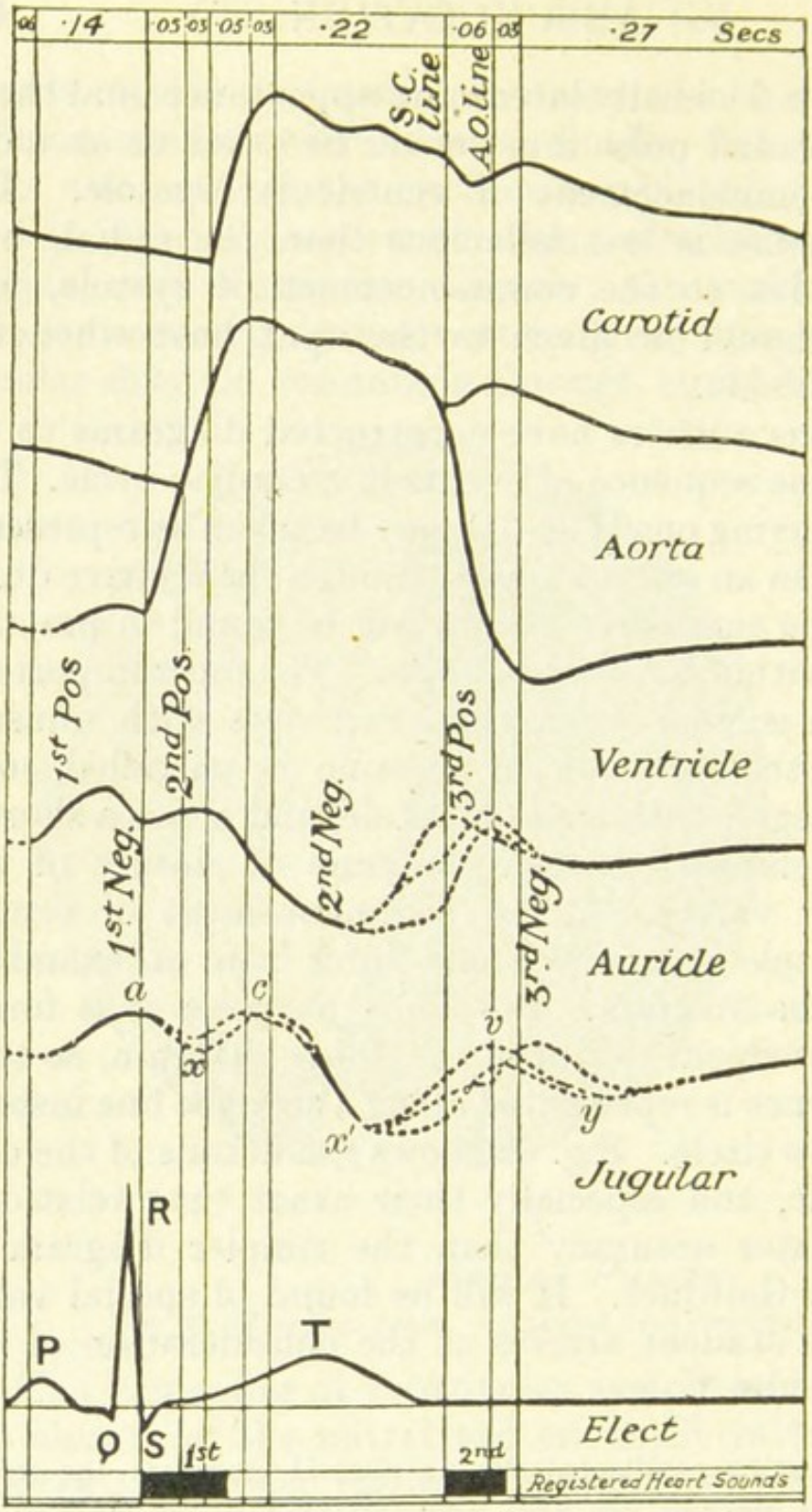


Fig. 32.—The cardiac cycle in relation to the pressures in the arteries, veins, and chambers of the heart, and to the electrocardiographic curve.

(From Lewis's "Mechanism of the Heart Beat.")

Dotted lines indicate parts of the curves which are liable to variation under normal conditions.

Time scale :—6 cm. per second.

The points where the aortic curve joins and leaves the ventricular curve indicate the times of opening and closing of the semilunar valves; the latter is marked S.C. The line marked A.O. corresponds to the opening of the auriculo-ventricular valves.

In the jugular curve, *a*, *c*, and *v* represent the auricular, carotid, and ventricular waves; *x*, *x'*, and *y* the troughs which follow them.

In the electrocardiographic curve P represents the "base-negative" summit caused by auricular systole, R and T are the expression of similar ventricular activity; Q and S are "base-positive" variations originating in the auricle and ventricle respectively.

In addition to a knowledge of the cardiac cycle, auscultation presupposes acquaintance with the situation of the valves of the heart and of the course of the principal arteries, as well as of the areas where sounds produced at the valves are best heard. For full particulars the student must consult works on regional

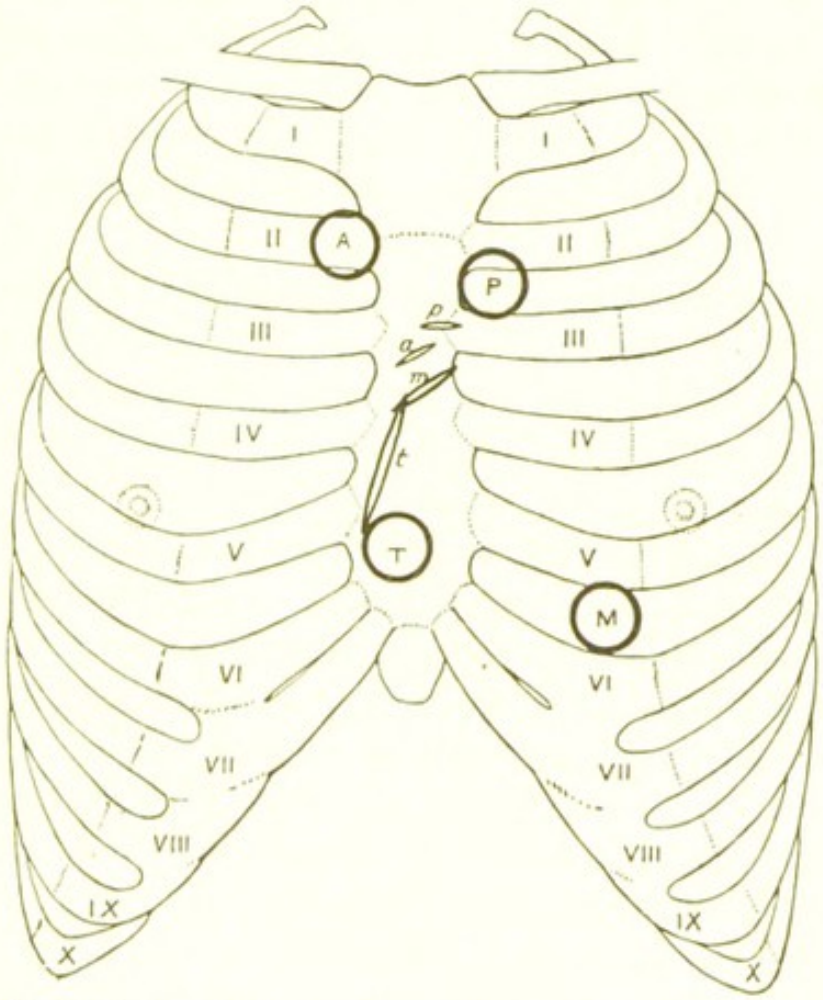


Fig. 33.—Position of the cardiac valves and auscultatory areas.

anatomy. The following summary merely recapitulates the most important facts :—

The **pulmonary valve** lies horizontally at the level of the upper border of the 3rd left costal cartilage ; the right half of the valve lies under cover of the sternum, the remainder passes outwards behind the costal cartilage (Fig. 33).

The **aortic valve** lies farther from the surface, and

at a slightly lower level. Its situation may be indicated on the front of the chest by a line drawn obliquely across the left half of the sternum on the level of the lower border of the 3rd costal cartilage.

The **mitral valve** lies slightly obliquely behind the inner end of the 4th left costal cartilage and adjoining part of the sternum. The **tricuspid valve** is placed much more obliquely; its upper end is opposite the 4th cartilage or interspace, and its lower near the lower border of the 5th right costo-sternal articulation. It marks the line of junction between the right auricle and right ventricle.

The **pulmonary artery** is situated at the inner edge of the 2nd left interspace, and behind the adjacent part of the sternum. At the lower border of the 2nd cartilage it divides into its branches to the right and left lungs.

The **ductus arteriosus** passes upwards from the left branch to join the aorta.

The **aorta** arises behind and slightly lower down than the pulmonary artery, and, passing upwards and to the right, approaches the surface of the chest most closely at the inner end of the 2nd right costal cartilage, arching backwards and to the left from that point. The **innominate artery** passes in a direction represented by a line drawn from the middle of the manubrium to the right sterno-clavicular junction.

A stethoscope placed over the valves of the heart would fail to distinguish at which of them a given sound takes origin, because they lie so near each other that the sounds from all of them would reach its chest-piece. Besides, in the case of the valves that lie more deeply the sounds would have to pass through the chambers of the heart which are situated between them and the surface, and thereby their clearness would be impaired. To avoid these inconveniences,

the sounds produced by each valve are listened for over that part of the chest where the cavity in which the valve lies most closely approaches the surface, and is most remote from the other cavities of the heart.

Hence one listens to the mitral valve at the cardiac apex, to the tricuspid at the lower end of the sternum, to the aortic over the aorta at the 2nd right costal cartilage, and to the pulmonary over the artery in the 2nd left intercostal space. It is practically found that in these regions the sounds of the respective valves are heard with a maximum of loudness and distinctness. They are therefore called the **mitral, tricuspid, aortic,** and **pulmonary areas,** although they do not lie immediately over the valves from which they derive their names. Auscultation should be performed systematically over these areas. In ordinary cases the student may begin with the mitral area, making certain of the time at which the sounds that he hears occur in the cardiac cycle, by feeling the apex beat whilst he listens. He may then pass to the tricuspid area, thereafter to the aortic, and lastly to the pulmonary. When necessary, auscultation may also be performed along a diagonal line joining the mitral and aortic areas. This is often of service, as, for instance, when a mitral systolic murmur is associated with an aortic one.

In health two sounds are often heard over each of these areas, the first corresponding with the beginning of ventricular systole, the second with the commencement of ventricular diastole. The first sound depends, from the clinical standpoint, chiefly on the closure of the mitral and tricuspid valves, and to a lesser degree on the muscle tone of ventricular contraction, on which, however, its duration depends, and on other subsidiary causes. The second sound is due to the closure of the aortic and pulmonary valves, and also, but very subordinately, to tension of the

vessel walls. This sound is sharper and shorter than the first, which continues through an appreciable period of systole, but not until its termination. At and to the left of the apex, only the aortic element of the second sound is audible. The observer must remember that it is always important to note the character of both the first and second sounds in each of the areas.

Deviations from the Normal in Disease

In disease the following deviations from the normal may occur :—

1. The sounds may have a different intensity, both absolutely and relatively to each other, from that which they possess in health. In estimating this, allowance must be made for the thickness of the chest wall and the volume of the lungs.

2. The sounds may be doubled, or their rhythm altered.

3. Adventitious sounds may be heard, either replacing or occurring along with the heart sounds.

1. alterations in intensity

(*a*) **The first sound may be weaker than usual.** Decided shortening or weakness of the first sound, still more its disappearance, indicates cardiac failure. In acute febrile disease this change may occur rapidly, and should always be looked for ; the left side of the heart generally yields first.

(*b*) **The first sound may be louder than usual.** It is then said to be **accentuated.** In *simple dilatation* the sound is often slightly accentuated and very clear, sharp, and short, as a large volume of blood is projected against the valve curtains, and the sound easily penetrates the thin ventricular wall to the observer's ear. In *hypertrophy* the sound is accen-

tuated, but dull, prolonged, and thudding, as the vibrations produced by the unusually forcible closure of the valve have to pass through the thickened heart walls, and the muscular element of the sound is specially prominent.

(*c*) **If the second sound is more distinct** in the mitral or tricuspid areas than the first, we have either to do with a weakened first sound or an accentuated second; whilst if the first sound is louder than the second in the aortic and pulmonary areas, the first sound is accentuated.

(*d*) **The relative loudness** of the second sound in the aortic and pulmonary areas varies somewhat, and is a good deal influenced by the patient's age. The pulmonary sound is rather more accentuated than the aortic in youth ; in old age the reverse is the case when the subject is in good health.

Accentuation of the second sound means that the valve where the accentuated sound is produced is closed with unusual force. The force of closure depends on the momentum of the column of blood that effects it, and the momentum depends equally on the mass of moving blood and on the velocity of its recoil against the valve. In the aorta the mass of blood is increased when the vessel is dilated near its origin ; the velocity of recoil when, in consequence of contracted arterioles or other obstruction to the outflow of blood, the arterial blood-pressure is increased. When the aortic accentuation is due to the former cause, the sound often assumes a peculiar resonance suggestive of the echo produced when a cork is drawn from an empty bottle. Over the pulmonary artery an accentuation of the second sound generally indicates increased blood-pressure in the pulmonary circulation, due to disease either of the lungs or of the left side of the heart. *In pneumopericardium* the

sounds are singularly clear and resonant, *in pericardial effusion* they are faint and muffled, *in pneumothorax* they are in many instances accentuated and clear, whilst when there are *cavities in the lung* near one of the valve areas the sound arising in that area may be reinforced by the cavity acting as a resonator.

2. REDUPLICATIONS

Under certain conditions the first or the second sounds may be **doubled.** The simplest explanation is that which assumes that when such a doubling occurs, the valves, either cuspid or semilunar as the case may be, close sooner on one side of the heart than on the other; the reason being, in the case of the mitral and tricuspid, asynchronism of systole of the respective ventricles; and in the case of the aortic and pulmonary valves, some variation of the normal relations of blood-pressure in the aorta and pulmonary arteries, since an increase in pressure will accelerate the closure of the valve which is subjected to it. This view is probably correct in some, but not in all cases. Clinically, **reduplication of the first sound** occurs under very various conditions, the most important variety being known as the "**bruit de galop,**" where the rhythm at the apex shows the accent on the second element of the triplet ⏑ – ⏑. In some cases the appearance of this bruit de galop is of very unfavourable import. Occasionally it is difficult to discriminate between a short presystolic murmur and a reduplication of the first sound. This difficulty is most apt to occur when the heart is acting feebly, and it is often found to clear up after a few days of rest and treatment. An experienced observer will seldom be at a loss, however, in determining to which class such a case should be referred.

Reduplication of the second sound indi-

cates, in a large proportion of the instances in which it is heard, an increase of pressure in the pulmonary circulation. It occurs, therefore, in certain lung diseases and in diseases of the left side of the heart, being very characteristic of mitral stenosis, in which disease it is heard in a large proportion of the cases. It should, however, be mentioned that many observers do not consider that the reduplication in mitral stenosis is due to early closure of the pulmonary valves.* It is also found where the right and left ventricles fail to contract simultaneously, whether the failure be due to an increase of work thrown upon one of them, or to the presence of structural changes in the heart muscle, or to derangement of the nervous mechanism which regulates their action. It also occurs physiologically at the end of a full inspiration and the beginning of the following expiration, being in this respect the converse of the first sound, which is more often reduplicated at the end of expiration and the commencement of inspiration. When reduplication of the second sound is observed, it is most important to determine its presence or absence, and its character, in each of the four valve areas.

Alterations in rhythm.—Alterations in the rhythm of the sounds deserve attention. The usual rhythm is that of triple time in music, with the accent on the first beat in the mitral and tricuspid areas, and on the second in the aortic and pulmonary, whilst the third beat is silent. This is slightly modified by the fact that a quickly acting heart gains time chiefly in the period of ventricular diastole, but the relation of the sounds is less modified by this than are the phases of the cycle. When, however, the vitality of the heart has been seriously impaired by long-continued high blood-tension, such as is seen in chronic nephritis,

* *Vide infra*, p. 164.

and especially if fever or some such cause assists in weakening the myocardium, the sounds become almost equidistant, the period of ventricular systole being unduly lengthened.

This *deliberate pendulum-like sequence* of the sounds should always be regarded with considerable anxiety, as it points—unless, indeed, the patient is being overdosed with digitalis—to serious involvement of the cardiac muscle.

Exactly the opposite effect is produced on the rhythm when systole is rapidly accomplished, or when from great weakness the ventricle fails to effect a complete emptying of itself, and the systole becomes abortive.

3. ADVENTITIOUS SOUNDS

Adventitious sounds may be of three kinds : endocardial, vascular, or exocardial. Abnormal endocardial sounds are called **murmurs,** or bruits. They are due to disease either of, or close to, the valve where they occur, when they are often known as organic ; or to some alteration in the state of the blood, which, by affecting its viscosity on the one hand and the nutrition of the tissues of the heart and vessels on the other, produces the conditions necessary for the development of a murmur.

The physical explanation of murmurs is by no means simple. The following are some of the factors concerned in their production :—

i. The viscosity of the blood.

ii. The velocity of the blood-stream.

iii. The passage of the stream from a narrower into a wider channel.

The third condition is equally well produced when a narrowed orifice leads to a normal cavity beyond it, or when a normal orifice opens into a dilated cavity.

Endocardial murmurs always have a definite relation to the events occurring in the course of the cardiac cycle,

their time and import varying with their point of origin.

In the so-called "organic" cases, where the valves or their surroundings are implicated, a murmur may either result from obstruction to the onward flow of the blood, or from leakage backwards through a closed but incompetent valve. The former are known as obstructive murmurs, the latter as regurgitant. In examining a murmur the following points must be noted :—

(*a*) Its time of occurrence.
(*b*) Its point of maximum intensity.
(*c*) Its direction of selective propagation beyond the præcordial area.
(*d*) Its character.

The **time of its occurrence** is noted with reference to the sounds of the heart, and these by comparison with the time of occurrence of the apex beat.

The **maximum loudness of a murmur** which has been produced at a given valve usually occurs at the point where the valve sound would be best heard in health. To this rule, however, there are some exceptions.

Experience shows that valvular murmurs are not equally well heard at all points of the chest wall which are equidistant from the point of their greatest intensity, but that each is much more distinctly audible at a distance in some directions than in others; this fact is expressed by saying that such murmurs have **directions of selective propagation.***

* The complete explanation of the selective propagation of many murmurs is still unknown. In most cases, however, the following factors greatly influence the direction in which the murmur is conducted: (1) The varying conductivity of the different structures in the chest wall, and between the chest wall and the heart; (2) the direction of the vibrating blood-stream; (3) the position of the chamber of the heart or of the vessel in which the valve lies.

The **character** of the murmur also helps to decide a doubtful case. Obstructive murmurs are apt to be rough ; regurgitant, to be soft and blowing.

The pitch and general quality of murmurs vary greatly ; some have quite a distinct musical character, others are harsh and sawing. The loudness of a murmur has no relation to the amount of damage which causes it. A very loud murmur is often far less serious than one so soft as to be nearly inaudible.

Murmurs due to disease of postnatal origin are

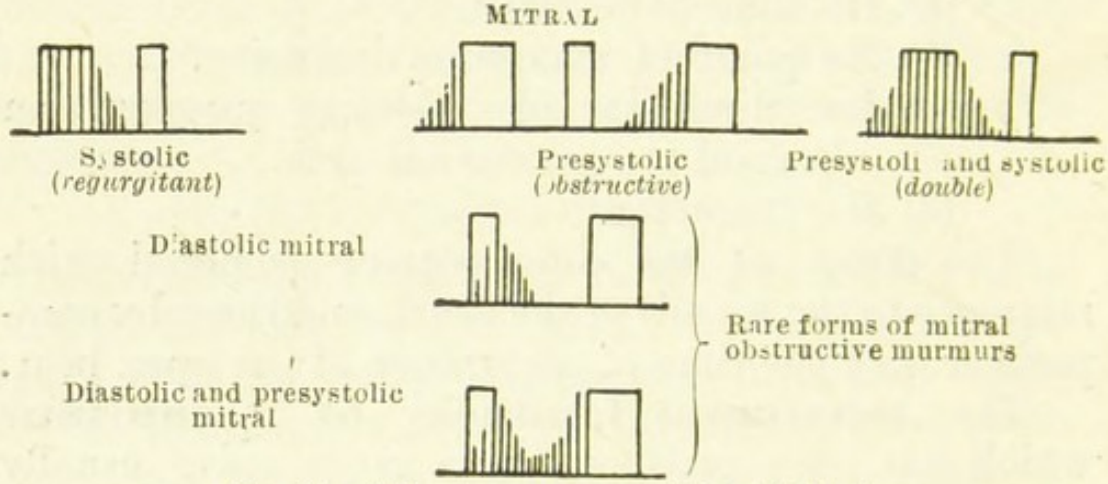


Fig. 34.—Mitral murmurs. (*After Wyllie.*)

very much oftener found to proceed from the valves of the left side of the heart than from those of the right, and in adult life murmurs at the tricuspid and pulmonary areas, due to morbid processes arising in these valves, are rare. The following is a short epitome of the chief murmurs which may be heard at the various valves, and of exocardial sounds : the diagrams illustrate the position of the more common murmurs in the cardiac cycle.

(1) *Mitral Murmurs*

Mitral murmurs may be either (*a*) obstructive or (*b*) regurgitant. (Fig. 34.)

(*a*) **Obstructive murmurs** occur during ventricular diastole, and are invariably of organic origin.

They sometimes follow immediately on the second sound, when they are known simply as *diastolic*. At other times the murmur is separated from the second sound by a brief interval, but terminates before the occurrence of the first sound; it is then called *mid-diastolic;* in yet other instances the murmur only begins with the advent of auricular contraction, when it is designated an *auriculo-systolic*, or, more often, a *presystolic* murmur. In each case the murmur is due to the onward rush of the blood through the deformed or narrowed mitral valve into the wider cavity of the left ventricle. During the earlier part of diastole this is effected by the aspiration of the relaxing ventricle, which is ordinarily strongest near the beginning of diastole—at the end of the period the contracting auricle is the main agent in producing the flow. Sometimes an early diastolic murmur is followed by a moment of silence, which is then succeeded by a presystolic murmur.* Best heard at the apex, or sometimes rather nearer the sternum, they have no direction of selective propagation; they are harsh and rough in character, more particularly when of the presystolic variety, and very often are associated with a distinct thrill. In the majority of cases the second sound is reduplicated, so that the murmur and accompanying sounds may be phonetically represented by "*rr*úp ti-ti"; or, where the heart's action is rapid and the murmur occupies a considerable portion of the diastolic period, by "ti-ti *rr*úp." Occasionally the presystolic murmur is accompanied by a mitral obstructive murmur occurring at the beginning of diastole, when the phonetic representation would become "*rr*úp ti tiff, *rr*úp ti tiff," or, if the murmur

* The term "postdiastolic" as applied to certain of these murmurs is very misleading. They are all diastolic in time, though, as has been stated, some are separated from the second sound by a pause.

occur a shade later in diastole, by "*rr*úp titi iff, *rr*úp titi iff."

The exact significance of the reduplication of the second sound in mitral obstruction is a matter of dubiety. At present many observers deny that the second element of the reduplication is produced at the

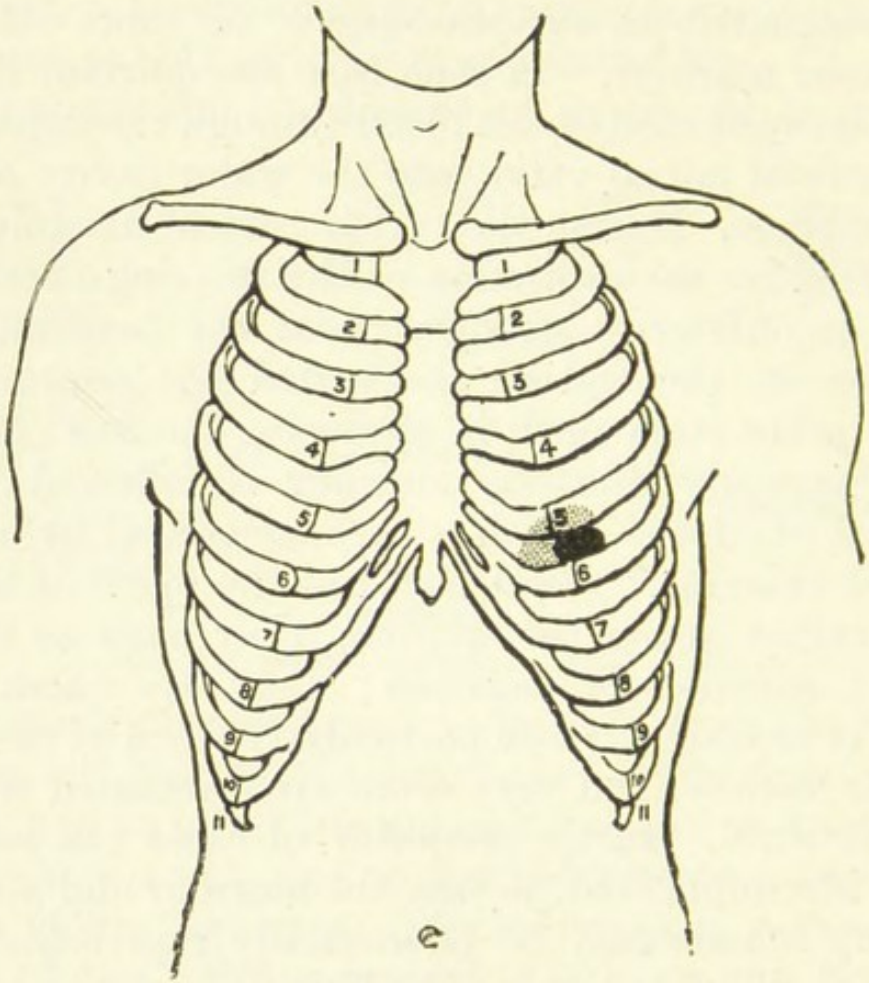


Fig. 35.—Presystolic mitral murmur.

semilunar valves, and the fact that it is better heard at the apex than at the base supports their contention. It is not improbable that one element of the double sound under discussion may originate in the mitral valve, whose segments, partially adherent along their adjacent margins, are no longer free to fall backwards before the blood stream as it again passes from the auricle to the ventricle at the commencement of

diastole, but are suddenly arrested in their progress, and, bulging into the ventricular cavity, become tense, and emit a sharp sound like that produced by a sail suddenly bellied by a gust of wind. (Fig. 35.)

(*b*) **Regurgitant murmurs** occur during ventricular systole, and may be either organic or simply due to dilatation. They begin with the apex beat and

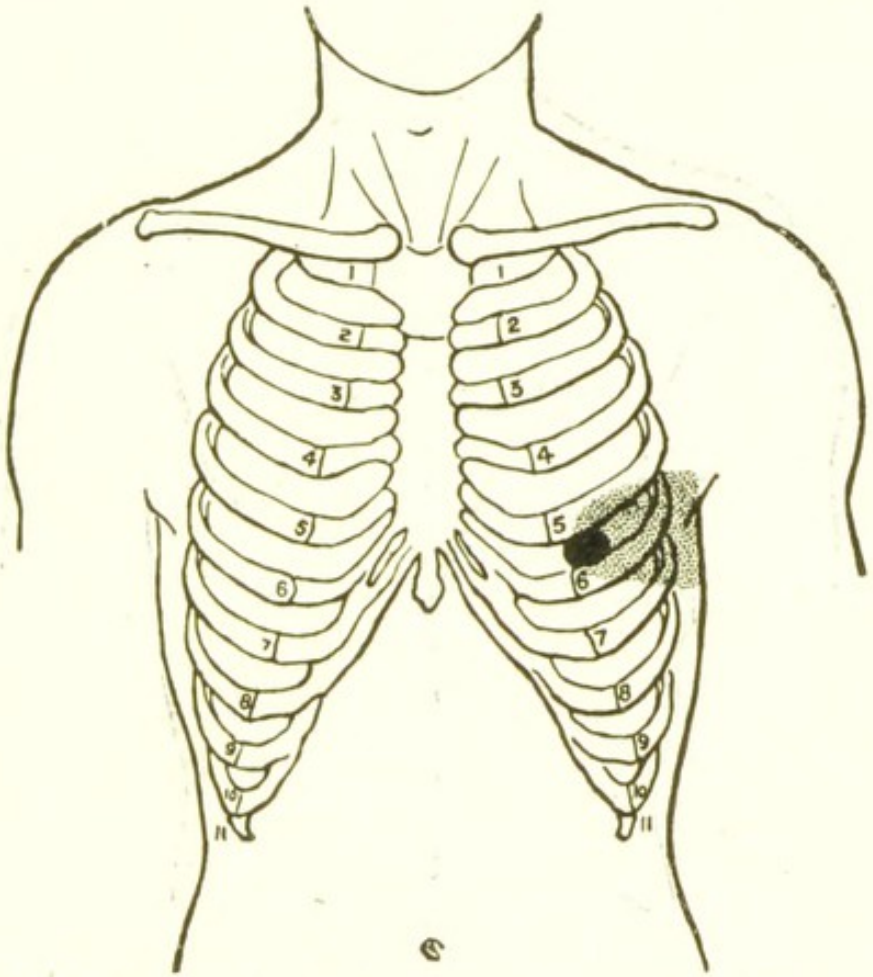


Fig. 36.--Mitral systolic murmur—propagation in front.

replace more or less completely the first sound in the mitral area. Their point of maximum intensity is at the apex, their direction of selective propagation is outward towards the axilla and angle of the left scapula, and they are generally soft and blowing in character. Slight mitral systolic murmurs, especially those due to dilatation of the ventricle and mitral orifice, and not to disease of the valve curtains,

frequently lack any selective propagation backwards. It is possible that some basal hæmic murmurs may be due to mitral regurgitation when the left ventricle is dilated (*see* p. 173). (Figs. 36, 37.)

A soft systolic murmur of exocardiac origin is occasionally audible near the apex of the heart in cases of pulmonary

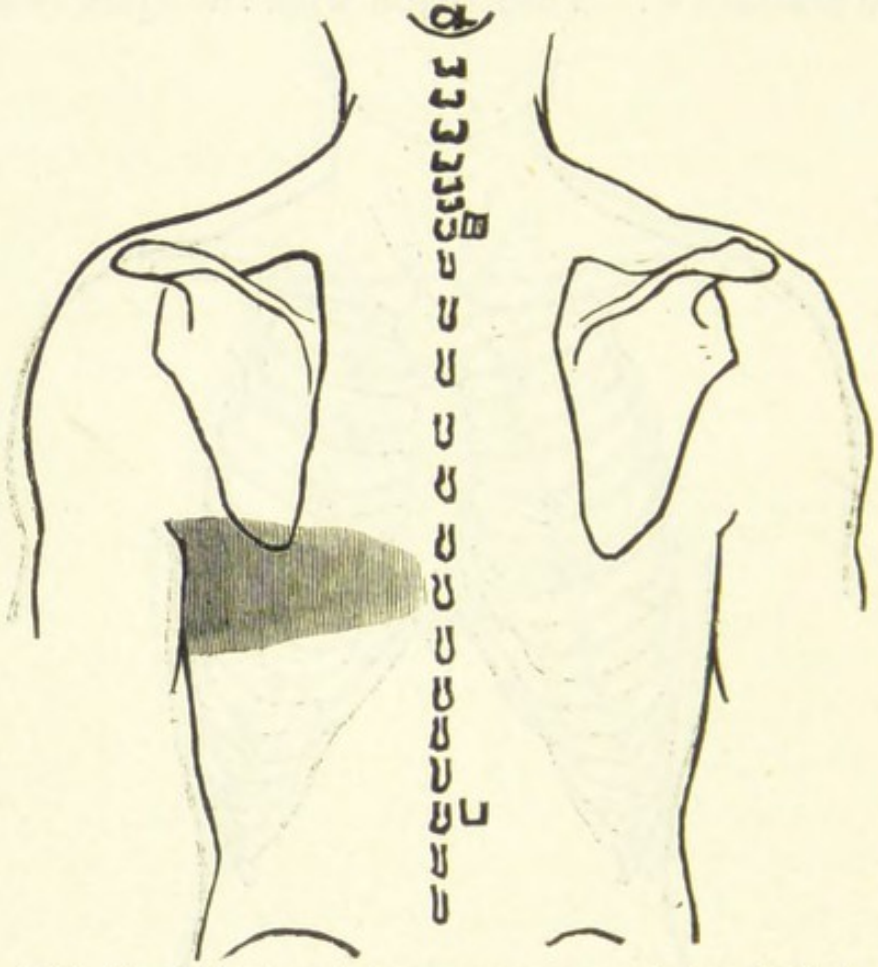


Fig. 37.—Mitral systolic murmur—propagation behind.

phthisis ; it is often propagated towards the axilla just as a mitral systolic murmur would be, but may be distinguished by its ceasing to be heard over the heart itself where uncovered by lung, and by disappearing or altering markedly in character when the patient holds his breath. Such cardio-respiratory murmurs must not be mistaken for true endocardial ones.

(2) *Aortic Murmurs* (Fig. 38)

(*a*) **Obstructive murmurs** occur during ventricular systole ; they are due either to obstruction of

the ostium aortæ from valve disease or to aortic dilatation beyond a normally sized ostium. They are rough in character; have their area of greatest loudness at the second right costal cartilage near the sternum; are propagated with the blood stream into the arteries; and may, in most instances, be readily heard over the carotids—sometimes at a much greater distance. (Fig. 39.)

(*b*) **Regurgitant murmurs** occur during ventricular diastole; they begin with the closure of the semilunar valves, and replace in part or completely the normal second sound in the affected region. They may be best heard in the aortic area; not infrequently,

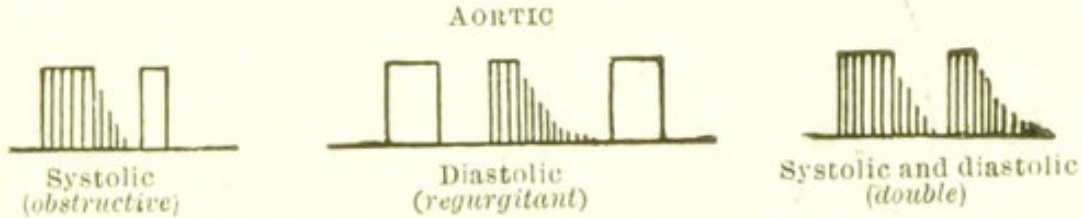


Fig. 38.—Aortic murmurs. (*After Wyllie.*)

however, they are as distinctly audible over the left half of the sternum, at the level of the 3rd rib and interspace. Their direction of selective propagation is towards the lower end of the sternum, though occasionally they are almost equally well heard near the apex; their character is less harsh than that of systolic aortic murmurs. Their intensity is greatest at first, and gradually diminishes during the diastolic period. Marked regurgitation leads to auscultatory phenomena in the arteries (*see* p. 174). (Fig. 40.)

In many instances one finds that a **double murmur** is present at the aortic orifice, the systolic element of which is not caused by real stenosis of the ostium, but by roughening and deformation of the valve segments, the diastolic murmur being due to the backward leakage through the misshapen cusps.

This double murmur often possesses a very distinctive "sawing" character.

Austin Flint has directed attention to the presence of a presystolic murmur at or near the apex of the heart in certain cases of aortic disease where, at the post-mortem examination, no change was found to be present in the mitral valve. Most probably this murmur indicates a dilatation of the left ventricle so

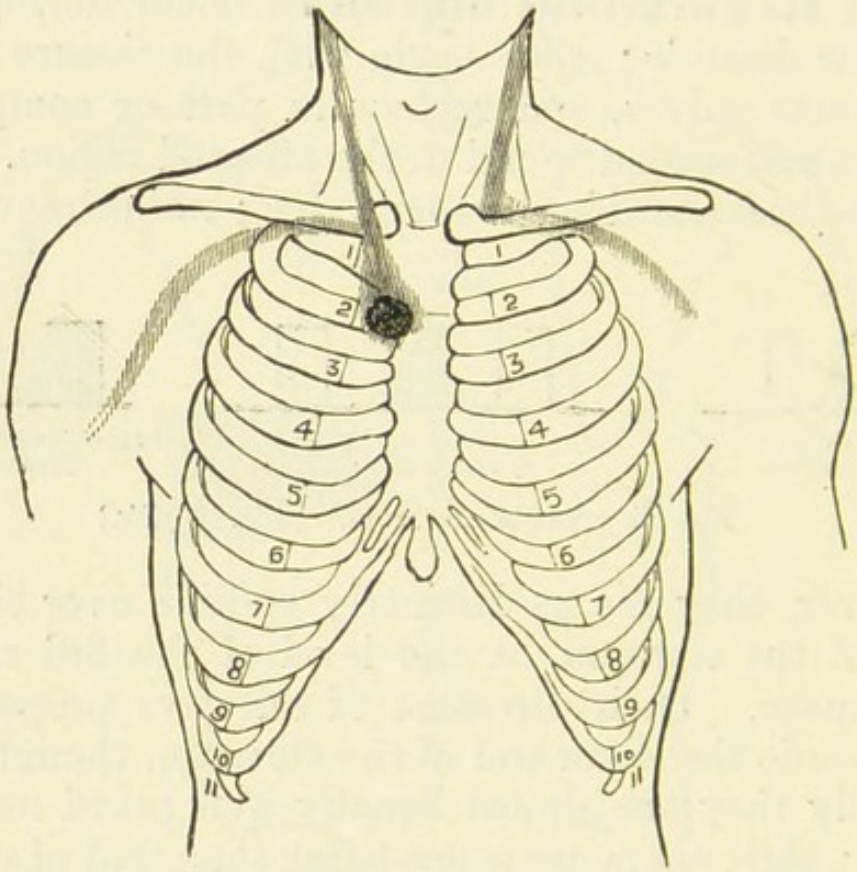


Fig. 39.—Aortic systolic murmur.

considerable as to render the mitral orifice relatively too narrow for the cavity beyond. The condition is a rare one.

(3) *Tricuspid Murmurs*

These are comparatively rare.

(*a*) **Obstructive murmurs** resemble those of the mitral valve, but have their maximum intensity at the lower end of the sternum. They have no selective propagation.

(*b*) **Regurgitant murmurs** have a similar character to mitral regurgitant murmurs, are best heard in the tricuspid area, and are associated with the venous pulse (*see* p. 202). They are usually a sequel to disease of the left side of the heart, after compensation has failed.

(4) *Pulmonary Murmurs* (Fig. 41)

These are best heard in the pulmonary area, have

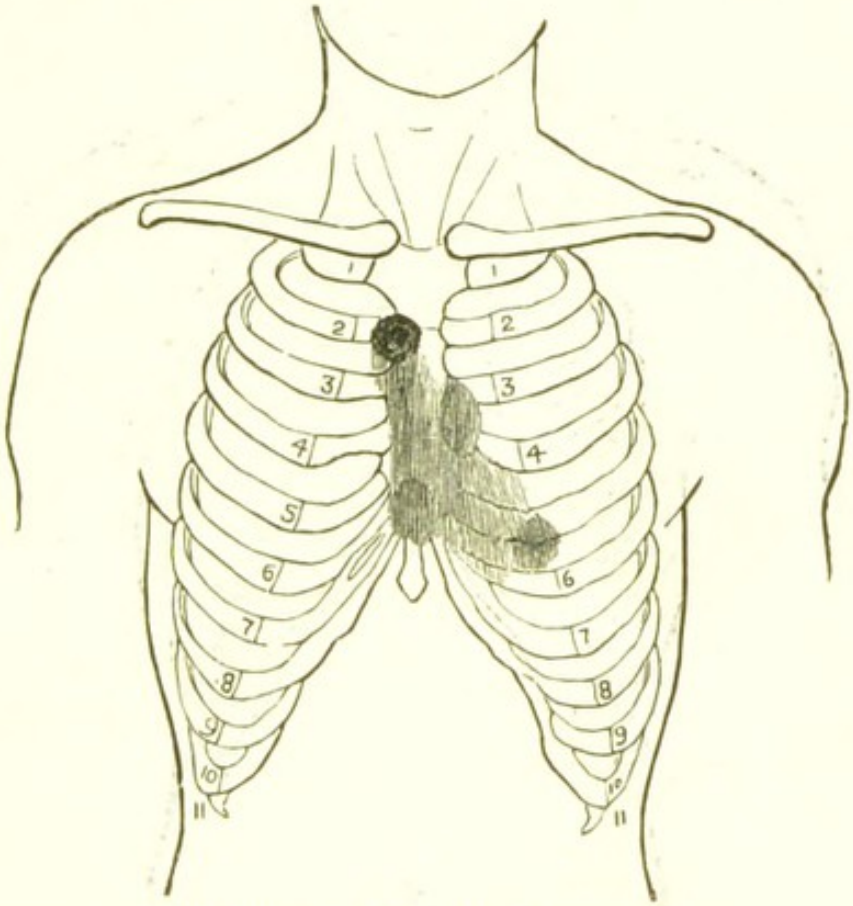


Fig. 40.—Aortic diastolic murmur.

no direction of selective propagation, though occasionally they are well heard as high as the 1st rib, are usually systolic, and are rarely due in postnatal life to disease of the valve, but most often to dilatation of the artery beyond the valve ring. They are very well heard in many cases of exophthalmic goitre.* A diastolic pulmonary murmur is excessively rare.

* In these cases there is often an aortic systolic murmur also.

(5) *Multiple Murmurs*

In a large number of cases **more than one murmur** is present during the cardiac cycle. When they occur at different epochs, it is easy to study each separately ; when two or more occur together, each will be found to possess its own position of maximum loudness, its characteristic direction of selective pro-

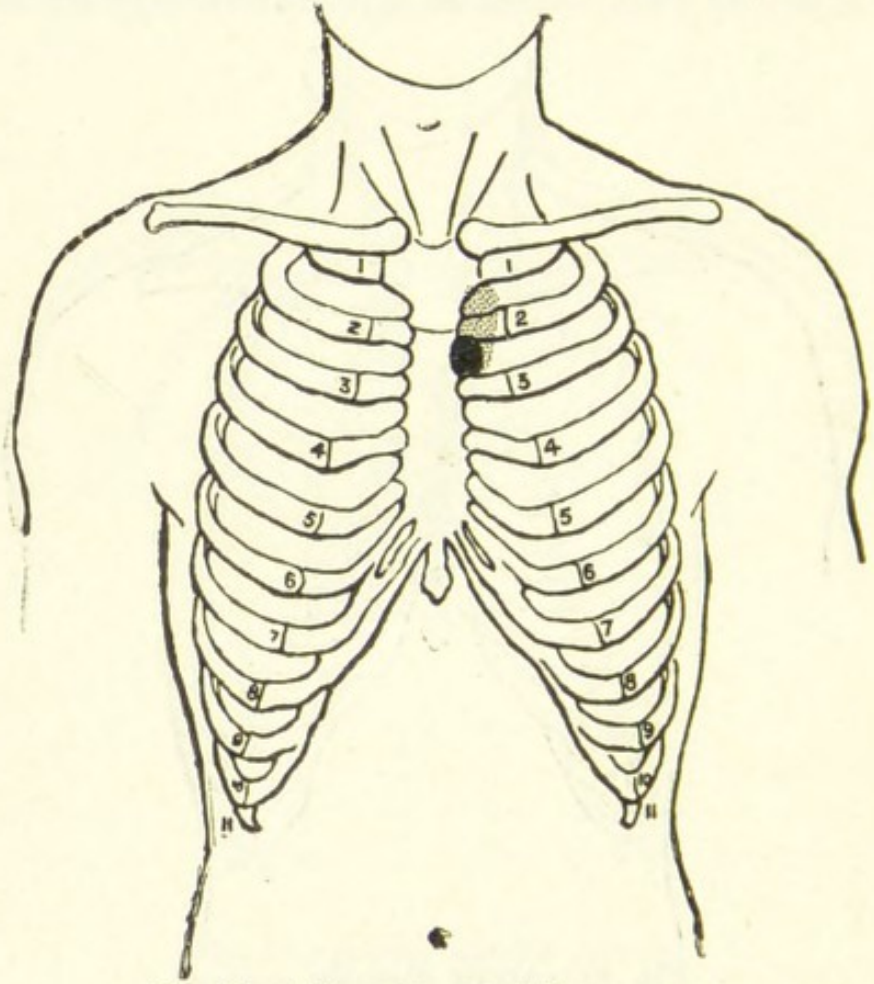


Fig. 41.—Pulmonary systolic murmur.

pagation, and its peculiar quality of sound. (Fig. 42.) Each lesion, moreover, will produce more or less definite effects on the general circulation, and by observing these by the other methods at our disposal a diagnosis can usually be arrived at. It must be recollected that during the last few days of life, when the diseased heart is acting feebly, serious lesions are often unaccompanied by any murmur, the force of the blood-

stream being too weak to produce any vibration. Some murmurs vary very much with the attitude of the patient ; it is therefore important to examine the heart in both the erect and supine positions.

(6) *Congenital Murmurs*

A **patent foramen ovale** may give rise to a murmur at the base of the heart, dependent on differ-

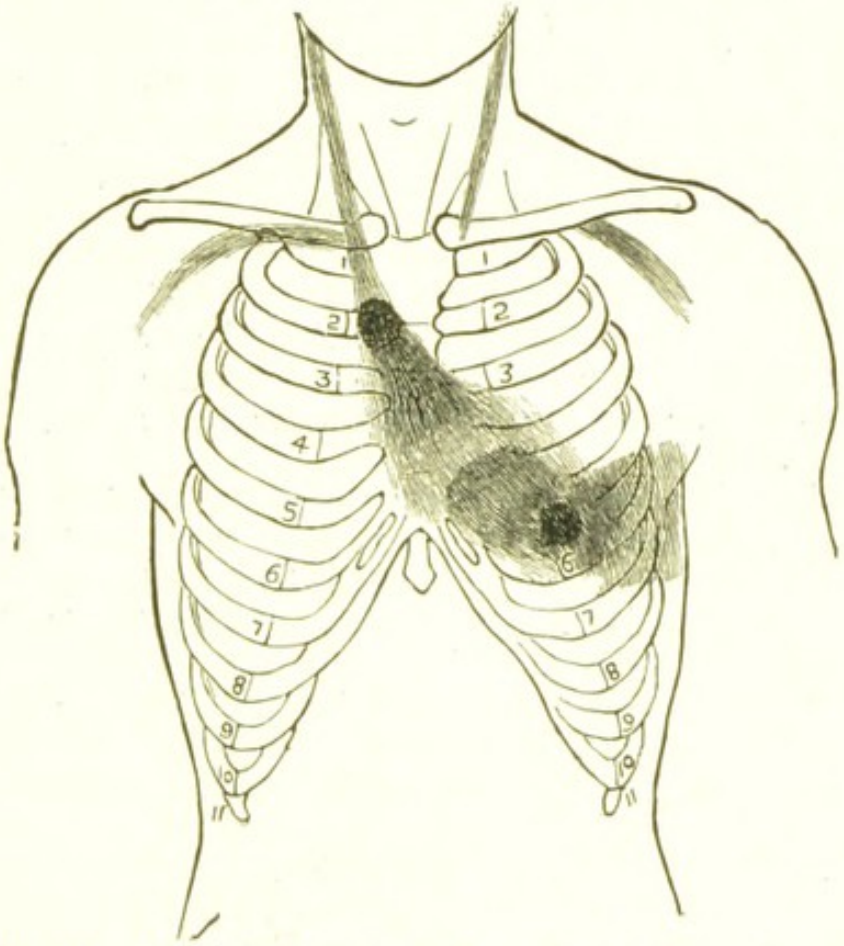


Fig. 42.—Combined aortic and mitral systolic murmurs.

ence of pressure in the right and left auricle. A murmur usually rather harsh in character, beginning after the commencement of systole and continuing into the earlier part of diastole, best heard in the 3rd left intercostal space near the sternum, and often associated with a distinct thrill, is caused by the **ductus arteriosus** remaining unclosed. The diagnosis of congenital cardiac murmurs is often fraught with consider-

able difficulty. The following points may be noted regarding them. They are mostly systolic; the situation of maximum intensity and the direction of propagation do not correspond with those of ordinary murmurs; whilst the fact that they are often associated with humming bruits at the base of the heart, and with an abnormal cardiac contour, may assist in their recognition.

(7) *Cardio-Pulmonary Murmurs*

Murmurs may be closely simulated by sounds due to diseased conditions in the neighbourhood of the heart, for in such circumstances the heart may be displaced, or it may be pressed upon, or its movements may through adhesions be communicated in an abnormal way to the lung, and induce abrupt movements of the air contained in its tissue and in the bronchi. It is also possible that a slight degree of pleural friction may become audible during cardiac systole if the roughened surfaces are situated near the apical region of the heart.

Cardio-pulmonary murmurs occur at the time of cardiac systole. They generally begin about the middle or near the end of that period. They are short in duration and are best heard during inspiration, but a very full inspiration may render them faint or inaudible.

Their commonest situation is just outside the apex beat. In a smaller number of cases they are only heard at the base of the heart, at or near the 2nd left intercostal space. Changes in the posture of the patient often cause them to disappear entirely. In arriving at a diagnosis the observer must take into account the condition of the lungs, pleura, and abdominal viscera, the phenomena observed on auscultation over the trachea, where true mitral murmurs are practically never heard, and the character of the pulse, which is dealt with in a subsequent section.*

(8) *Hæmic and Vascular Murmurs*

In **anæmia** several murmurs are frequently heard over the heart and vessels. One, which is of specially

* For further details consult Fowler and Godlee, "Diseases of the Lungs," p. 67; Coombs, *Quarterly Journ. of Med.*, v. 274; and Thayer, *Med. Mag.*, xix. 747, xx. 117.

common occurrence, is audible in the 2nd left intercostal space over or just external to the pulmonary area. Various theories have been advanced to account for it. The chief of these are—

i. **Balfour's theory,** in which, accepting the view first propounded by Naunyn to explain the existence of a basal systolic murmur in certain cases of mitral disease, it is held that the sound is due to regurgitation through the mitral valve into the left auricle, and that it reaches the ear by way of the auricular appendix, which for various reasons—including the fact that the anterior border of the left lung is often rather retracted in anæmia—approaches the chest wall more closely than under other conditions. The supporters of this theory have pointed out that the murmur attains its maximum intensity not over the pulmonary artery, as is found in undoubted cases of pulmonary systolic murmurs, but farther to the left.

ii. Many writers regard the murmur as due to **slight dilatation of the pulmonary artery** beyond the valve ring, and to a less viscous condition of the blood, which would accentuate the vibrations set up by such a dilatation.

iii. **Sansom** * holds that the vibrations which cause the murmur arise in the conus arteriosus, and perhaps also in the semilunar valves themselves, and are due to a fibrillary tremor of the overstrained muscular fibres which are found in these regions.

The second of the above theories has at present the greater number of supporters.

Hæmic murmurs are also heard at times in the mitral, and much less frequently in the tricuspid and aortic areas, the last being particularly uncommon. *In all cases such murmurs are systolic in time.*

A continuous humming sound is often audible over the veins at the root of the neck in **chlorosis.** It is known as the **"bruit de diable,"** and is caused, in part at least, by an alteration in the calibre of the internal jugular vein as it passes through the cervical fascia. In order to hear the bruit de diable clearly, the stethoscope must be held very lightly, so as to exert

* "Diagnosis of Diseases of the Heart," p. 285.

no pressure, over the clavicular head of the sterno-mastoid muscle. Not seldom one may also hear the sound perfectly distinctly when the stethoscope is placed on the sterno-clavicular articulation, by which manœuvre all possibility of creating a factitious bruit by pressure is avoided.

Hæmic murmurs may arise **in the larger arteries,** and are present independently of the pressure of the stethoscope, and it seems probable that an important, if not the chief, factor in their production is a disturbance of the vasomotor mechanism of the vessels.

There are, moreover, other sounds which may become audible in the arteries, and which are the result of changes in the pressure of the blood stream. The most notable instance of this is found where relaxed arteries are so rapidly distended by a large blood-wave that their walls are thrown into vibration by the sudden strain, and a sound is produced which corresponds with the advent of the pulse-wave. In cases of aortic regurgitation, where these conditions are most fully developed, we have also a second sound which occurs at the instant when the pressure once more falls off. This double sound, when heard in the femoral, is very characteristic of aortic regurgitation. Pressure produced by an ill-applied stethoscope often converts these sounds into murmurs.

A murmur in the subclavian artery as it crosses the apex of the lung may be caused by pulmonary disease.

When there is an **aneurysmal dilatation** of the aorta, murmurs may or may not be present, or the aortic second sound may be accentuated over the sac ; but no definite rule holds for such cases. When an aneurysm opens into another large vessel—e.g. the superior vena cava—the murmurs produced may be very loud, and are heard in unusual situations.

(9) *Exocardial Sounds*

Exocardial sounds may be due either to pericardial friction or to a localized pleurisy near the heart.

When **pericardial friction** occurs over an area uncovered by lung, it has a singularly superficial character, and thus can often enough be readily recognized.

Unlike the murmurs already described, pericardial friction does not correspond definitely with the events of the cardiac cycle. It is generally more distinct in systole than in diastole, but tends to exhibit a to-and-fro character, the first element occurring during systole and the second during diastole, but not necessarily

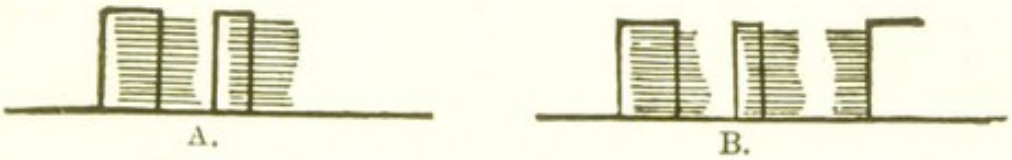


Fig. 43.—Pericardial friction. (*After Wyllie.*)

A. Ordinary type, one rub in systole and one in diastole.
B. Type with triple rhythm, one rub in systole and two in diastole.

commencing at the beginning of either phase. Sometimes the sound occupies the latter part of systole and the early part of diastole without exhibiting any pause between its first and second elements; sometimes it remains audible during the whole of the cardiac cycle. Further, its position of greatest intensity does not correspond with any of the areas in which valvular murmurs are best heard, and it is not propagated to a distance, but remains confined within narrow limits. Its position may be observed to vary from day to day. As a rule, it appears first near the base of the heart on the left side, but when the condition has become general it is best heard near the left nipple, and is sometimes associated with a distant thrill. The intensity is often considerably modified by the attitude of the patient. When the inflammatory process

involves the auricle as well as the ventricle, the to-and-fro rub may be replaced by a triple friction sound (Fig. 43).

In pericarditis the heart's action is apt to become tumultuous. *When fluid is poured out, the cardiac sounds become faint and distant.*

When air and fluid are both present in the pericardial sac—an event of very rare occurrence—a churning or "water-wheel" sound can be heard on auscultation.

To distinguish between the rub of pericarditis and that of pleurisy over a neighbouring portion of lung the patient should be instructed to hold his breath. Pericardial friction is unchanged by this, but if it be of pleural origin it will either be much reduced in intensity or will wholly cease. On the other hand, deep respiration will increase the pleural sound, but will not influence the pericardial.

The possible coexistence of both pleuritic and pericardial friction must not be overlooked.

VI. THE PULSE

The examination of the pulse gives us direct information regarding two things, namely, the condition of the vessel walls and the amount and variations of pressure of the contained blood. By intelligent observation of these facts we can obtain very valuable information regarding the state of the heart and circulation, as well as the general state of the patient.

When any observation is to be made on the pulse, the patient should be lying on his back, or at least sitting; and, except for special purposes, should not have been making any effort for some little time previous to the examination. The pulse is most readily felt when the patient's forearm is pronated. In cases of aortic regurgitation the peculiar character of the

pulse (*see* p. 196) is more distinctly brought out when the patient's arm is elevated.

To feel the pulse, place three fingers of the right hand on the patient's radial artery at the wrist. It is immaterial whether the observer's index finger be nearer the elbow or the hand of the patient, but for beginners it is best to select the same position in all cases. If it is made a habit to examine both radials in every case, errors in diagnosis, such as failing to detect the presence of aortic aneurysm or an abnormal position of the vessel, will frequently be avoided.

When the artery is beneath the finger, the following observations should be systematically made :—

(A) **Factors which depend upon the heart.**

1. Rate of pulse.
2. Rhythm of pulse (irregularity, intermission).
3. Equality or inequality in strength of successive beats.

(B) **Factors which depend upon the vessel.**

4. The size of the artery.
5. The condition of the vessel wall (*a*, hypertrophy of media ; *b*, changes in intima, viz.—early, fibrous thickening ; late, atheroma).

(C) **Factors which depend on both heart and vessels,** the latter element generally being the more important.

6. The amount of movement during the passage of a pulse wave (volume).
7. The blood-pressure in the vessel during the beat (maximum pressure, or "force").
8. The blood-pressure between the beats (minimum pressure, or "tension").
9. The general character of the pulse wave as regards rise, maintenance, and fall of pressure ; and the presence or absence of palpable dicrotism or of other secondary waves.*

* In studying the pulse the student should always have before his mind the relation between the successive changes in pressure which can be felt with the finger, and the graphic record of these changes which can be secured by the use of the sphygmograph. The rise of pressure corresponds with the sphygmographic upstroke, the maintenance with the apex and portion of the curve following it as far as the dicrotic notch, the fall with that portion of the curve which commences with the dicrotic wave (*see* p. 193).

The **rate** of the pulse is given as so many beats per minute. It is well not to begin counting immediately the finger is laid on the pulse, as the agitation of the patient often accelerates it at first. After waiting till it resumes its normal rate, count the beats for one minute, and record the result. The beat at which the observation commences should not be counted.

The successive beats of the pulse may recur at equal or unequal intervals, giving a **regular or irregular rhythm.** In the latter case, the beats may occur in symmetrical groups, some of the simpler

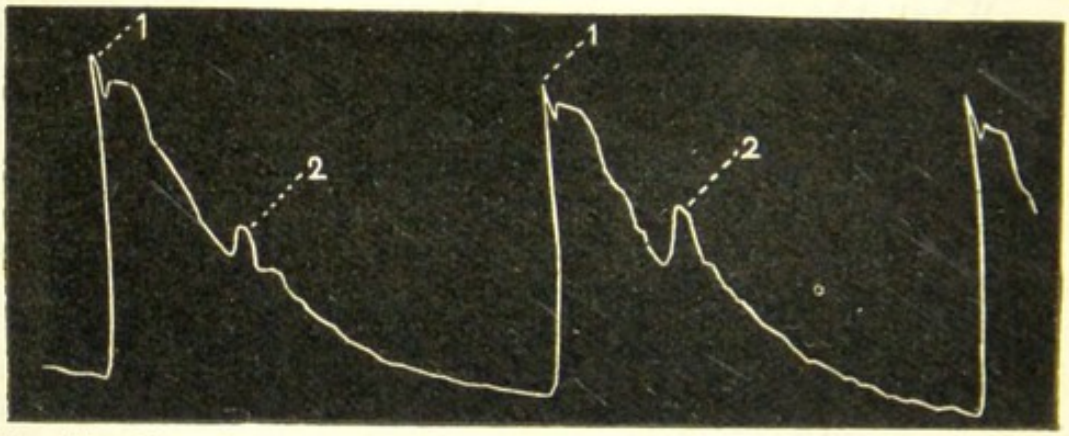


Fig. 44.—Bigeminal pulse. *(From a tracing lent by Dr. Byrom Bramwell, "Students' Guide to Examination of the Pulse."*

of which have received special names ; thus if there be two beats and a pause, recurring in regular sequence. we obtain the *pulsus bigeminus* (Fig. 44), three beats and a pause give the *pulsus trigeminus* (Fig. 45). In other cases no such symmetry occurs. Besides the varying interval that may occur between consecutive beats, the beats themselves may be unequal in **strength.** Some beats may be weaker, and the weaker beats may eventually become imperceptible —we thus arrive at pulses which may be classed under the previous heading (irregular pulses) ; this is especially true of the symmetrical types.

Recent physiological investigations, and especially the researches of Wenckebach, Mackenzie, Einthoven, and Lewis,

have thrown much light on the causes of irregularities in rhythm and force of the pulse. In many cases a pathological stimulus, arising at a point in the heart other than that from which the normal contraction starts, leads to a premature systole. Thereafter the subsequent normal stimulus occurring before the end of the refractory phase, during which the heart wall is inexcitable, fails to elicit any response, and only the next following normal stimulus succeeds in inducing a contraction. As the sequence of the normal stimuli is undisturbed by the occurrence of the pathological one, the period between the premature contraction and the following normal one is longer than the normal interval by an amount exactly equivalent to that by which the previous interval had been diminished. The contraction due to the abnormal stimulus is called an "extrasystole." Many cases of bigeminal pulse are due to the regular recurrence of such extrasystoles.

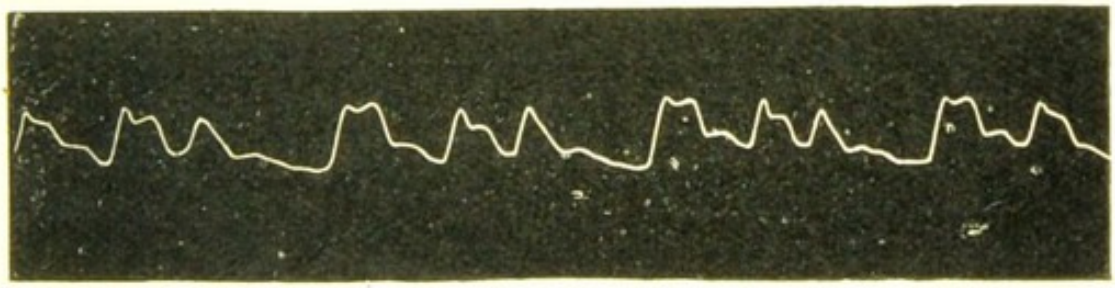


Fig. 45.—Trigeminal pulse.

In certain cases cardiac irregularity is due not to a pathological stimulus but to defective conductivity. This occurs where degenerative changes have invaded the junctional tissues.

Wenckebach classifies irregularities in the period-length of the pulse thus :—

(1) With preservation of the regular rhythm of the heart (pararrhythmia).
  (*a*) In a more or less regular manner from extrasystoles.
  (*b*) In the form of allorrhythmia (i.e. "regular irregularity") of various kinds, such as may arise from disturbances of the conduction, the contractility, or the excitability of the heart.

(2) With a change in the rhythm of the heart.
  (*a*) At definite times (e.g. variations in the period), length due to respiration, the rate being increased during inspiration and decreased with expiration.
  (*b*) In a perfectly irregular manner.

In this connection it should be observed that in many cases

of irregularity of rhythm in the pulse, the strength of successive beats will be found unequal, for the longer pause is naturally followed by a stronger beat. In such cases the unequal strength of successive beats is a direct result of the arrhythmia. Inequality in strength is of much greater importance when the rhythm is regular, or when small beats follow a long pause.

The fourth observation is directed to ascertain the **state of the vessel.** Two points should be noted: first, the size (calibre) of the vessel; second, the condition of its walls.

To determine the **calibre,** empty the vessel of blood by firm pressure, and endeavour to gauge its breadth in the flattened state. Should this be impracticable, allow the blood to return below the finger, and note the size of the cylindrical tube. When the vessel is contracted the calibre is small; when the muscular coat is fully relaxed it is distinctly larger. Be careful to ascertain that the radial artery is really under observation; in a certain proportion of cases it winds round to the back at an unusually high level, whilst the superficialis volæ continues to run in the usual site of the larger vessel. One may thus be led into error if the abnormal distribution is not recognized.

To discover the **state of the walls,** flatten the vessel and cause the skin of the patient's wrist to slip up and down over it. In health the vessel wall can rarely be felt unless the arm is thin. In disease one may feel general or local thickening, calcification, tortuosity, or irregular dilatations. These changes must be discounted when an attempt is made to estimate the blood-pressure in the vessel.

Having observed the state of the vessel, one proceeds to apply just sufficient pressure to flatten it between the beats. When this is done the increased blood-pressure that is present during the beat will cause the blood-vessel to resume its cylindrical shape;

and further, inasmuch as the vessel wall is elastic, will stretch it until the internal stress is balanced by the strained wall.

This observation enables us to estimate the **amplitude of movement** of the vessel wall during the passage of the pulse wave. As the elastic stretching of the vessel is never great, the movement chiefly depends on the resumption by the flattened artery of its cylindrical shape, and the amount of such movement is consequently greater the more dilated the vessel is. The force of the heart's action also exercises a certain influence on the amount of movement, although to a much less degree than the relaxation or contraction of the coats of the artery.

This proceeding is often described as "*observing the expansion*" of the pulse.

The next point is the determination of the **maximum blood-pressure, or force**, which, of course, occurs during the beat. In this case three fingers must be placed on the artery, so that it may be compressed both above and below the point where the pulse is being felt.

Place the finger next the wrist firmly on the vessel to prevent any pulse from the ulnar artery reaching the middle finger through the palmar arch; let the middle finger rest on the vessel with such pressure as will render the pulse most distinct, and then gradually compress the artery above this point with the remaining finger, noting the pressure employed when the pulse ceases to be felt by the middle finger. This pressure, being just sufficient to prevent the blood from lifting the finger during the beat, corresponds to the maximum blood-pressure. Only by careful practice on many healthy and diseased pulses can the student determine whether in a given case the requisite pressure is normal, excessive, or diminished.

The importance of cutting off recurrent pulsation through the palmar arch must never be overlooked. It is most likely to occur in cases where, owing to vascular dilatation, the blood-pressure is unusually low, and thus if neglected would lead to serious error, as the middle finger would continue to feel the pulse even when the upper finger exerted great pressure. As a natural consequence the observer would greatly overestimate the maximum blood-pressure in the vessel. It is worth noting that this recurrent pulse is frequently present in chlorosis.

It is rather more difficult to estimate the **minimum blood-pressure, or tension.** One thing may be remembered—namely, that in cases where the maximum pressure is low the minimum must be still lower; this rule must not, however, be extended to an assertion that a high maximum will necessarily involve a high minimum pressure.

To test for the minimum pressure, one may attempt to roll the vessel from side to side under the fingers between the beats. When the pressure is low, one cannot feel the vessel at all; if it is high, it may feel as hard as a piece of whipcord. Of course, one must be careful not to be misled by thickening of the wall of the artery.

Another method which gives good results, and which may be more easily appreciated by many, is to feel the pulse first with light, then with moderate, and finally with considerable pressure of the fingers on the artery. A pulse of low tension (i.e. with a low minimum pressure) is best felt in the first case, for the light pressure is sufficient to flatten the vessel between the beats, whilst it allows the artery to resume its cylindrical shape without much resistance during the beat, and is thus favourable to the development of the greatest possible amplitude of movement; whilst on the

contrary, where the tension is high, considerable pressure is required to flatten the vessel between the beats. But one obtains the greatest amplitude of movement precisely when the vessel is thus flattened, and so in a high-tension pulse the more firmly one presses the more forcible does the pulse appear to grow.

A normal pulse, lying as it does between these extremes, is best developed when moderate pressure is applied.

The difference between the maximum and minimum blood-pressures is often called the **"pulse-pressure."**

When all these points have been determined, one should conclude by studying the **general character of the pulse-beat.** This is divided into three periods: first, the period during which the blood-pressure is rising; second, the period at which the blood-pressure continues near its maximum; and last, the period during which the blood-pressure once more falls off.

The amount of movement having been already observed, one tries to estimate the rapidity of the rise of pressure, describing the rise as abrupt, rapid, moderate, or slow. In cases where it is abrupt it will be found that the pulse is of low tension; where it is slow the pulse is either one of high tension or an aneurysm is present. The latter condition may cause the rise to be excessively gradual. It is only in aneurysm that the rise of pressure may be as gradual as the subsequent fall.

As regards the period at which the pressure remains near its maximum, the point to be observed is whether the pressure is well sustained, or whether, on the other hand, it has no sooner attained its highest value than it begins to fall off again with rapidity.

In the third period one observes whether the fall of

pressure is swift or gradual. During the fall, instead of continuous decrease, there may be oscillations of pressure, which, in marked cases, are quite perceptible to the finger as distinct impacts following the primary stroke of the pulse. Of these the most noteworthy is that known as the **dicrotic wave.** It is best marked in pulses of low tension (provided there is not aortic regurgitation), and is most readily felt when the finger is very lightly applied to the vessel. Where not readily perceived, it is often accentuated by occluding the vessel on the distal side by the lower finger, whilst the middle and upper ones remain lightly applied.

The secondary wave which the finger detects with the next greatest frequency is the **tidal or pre-dicrotic wave.** It occurs in certain pulses of high tension where the blood escapes slowly from the contracting ventricle, and is very characteristically present in aortic stenosis. In opposition to the dicrotic wave, it is best felt when considerable pressure is applied to the artery, and thus there is no difficulty in discriminating between the two.

Besides these, in pulses of high tension there are frequent fluctuations of pressure at a period subsequent to that in which the dicrotic wave occurs.

These can sometimes be detected by the fingers, but it requires much practice to recognize them. They are visible enough in good sphygmographic tracings.

The **typical pulse** of a healthy adult man should be described in some such terms as the following :—

The rate is 70 per minute. The beats are regular in rhythm and equal in strength. The expansion is moderate in amount. The vessel is not tortuous, its walls are not rigid or thickened, and between the beats it is just possible to feel it. It is of medium size. The passage of the pulse wave can be arrested by moderately firm pressure, and the beat is best felt when a

medium degree of pressure is applied : in other words, the force and tension are both moderate. With regard to the general character of the pulse, the rise of pressure is neither abrupt nor very gradual, it is fairly well sustained, and its fall is gradual but not very tardy. No conspicuous oscillations of pressure are discernible during the time of subsidence.

The advantage of an instrument which could be used in clinical work for the accurate measurement of the arterial blood-pressure both during and between the beats has long been recognized. We are now in possession of several which, though they cannot be wholly free from risks of error, are yet sufficiently trustworthy to offer a very material assistance in the estimation of blood-pressure, and, whilst supplementing and confirming observations which have been made by the finger, have the additional advantage of yielding their results in a form capable of being definitely recorded.

Of these instruments the most suitable for clinical use are one or other of the modifications of Riva-Rocci's sphygmomanometer for the estimation of the maximum pressure, Dr. H. French's model * being the most conveniently portable ; Oliver's hæmomanometer, which can be used to estimate both maximum and minimum pressure ; and Pachon's sphygmometric oscillometer, which also serves to estimate both pressures, but is specially designed for the determination of the minimum pressure.

For hospital work, probably the best instrument is G. A. Gibson's sphygmomanometer, which by means of a revolving drum takes a permanent record, and which serves to estimate both the systolic and diastolic pressures.

1. **Riva-Rocci's sphygmomanometer** (modified). This consists of a mercurial manometer, an armlet for compressing the upper arm, and a double-valved bulb to inflate the armlet. The armlet should be about 4½ in. broad, as narrow armlets introduce very serious errors, especially in the case of high-tension pulses. A valve, by means of which the pressure in the armlet can be gradually reduced, is attached to the manometer, and the various parts are connected by means of rubber tubing.

Method of use : Fix the armlet securely on the upper arm,

* Supplied by Messrs. Down Bros., Ltd., St. Thomas's Street, London.

and gradually raise the pressure until the pulse can no longer be felt at the wrist. Note the manometer reading at the moment the pulse disappears. Increase the pressure a little, and then, by means of the valve, again reduce it very gradually, until the return of the pulse is observed, when a second reading of the manometer is taken. As it is easier to detect the first return of the pulse than its final disappearance, the second reading is, as a rule, the more accurate of the two, but the first one is a very useful check in case of a faulty observation.

2. **Oliver's hæmomanometer** is a modification of the Riva-Rocci instrument. The column of mercury is replaced by a spirit index which shows the changes that

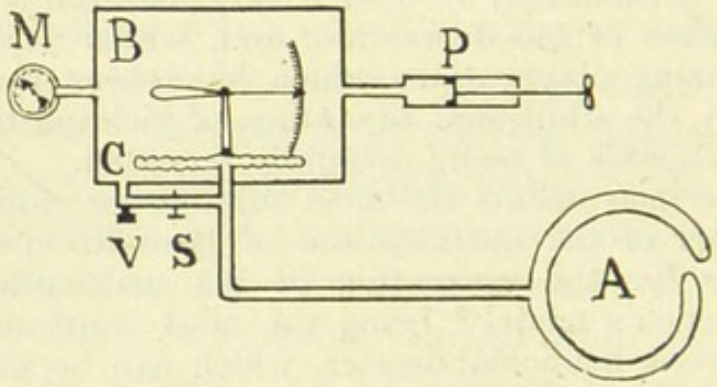


Fig. 46.—Pachon's sphygmometric oscillometer.

A, armlet; B, airtight metallic chamber; C, sensitive manometric capsule and index; M, ordinary manometer showing the pressure within B; P, pump; S, separator to disconnect B from A and C; V, valve to allow escape of compressed air.

occur in the volume of air in a closed chamber as the pressure in the interior of the system is varied, and which is graduated to give readings in millimetres of mercury. The air-pressure is raised or lowered in the armlet by means of a bellows reservoir which is compressed or expanded by turning a screw. This forms a marked improvement on the ordinary bulb.

The chief advantage of the apparatus is that, owing to the small inertia of the air and spirit index, and to the openness of the scale, it is easy to determine the pressure at which the excursions of the index attain their widest range, and thus to arrive at an approximate estimate of the minimum blood-pressure.

3. **Pachon's sphygmometric oscillometer.*** (Fig. 46).—This consists of a metal chamber connected with

* Supplied by G. Boulitte, 7, Rue Linné, Paris.

the armlet, with the pressure pump, and with an ordinary metallic manometer which indicates the air-pressure within it. Wholly enclosed in the metal chamber is a sensitive manometric capsule with its pointer and scale, the latter being visible through an air-tight window in the chamber wall. The external surface of the capsule is thus always subjected to the same pressure as that of the air within the chamber. The interior of the capsule communicates directly with the air in the armlet by an independent channel. By means of a separator the metal chamber can be disconnected from the armlet without altering the pressure of the air contained in it, and the pointer of the enclosed capsule then moves freely with any oscillations of pressure caused in the armlet by the expansion and contraction of the artery in the

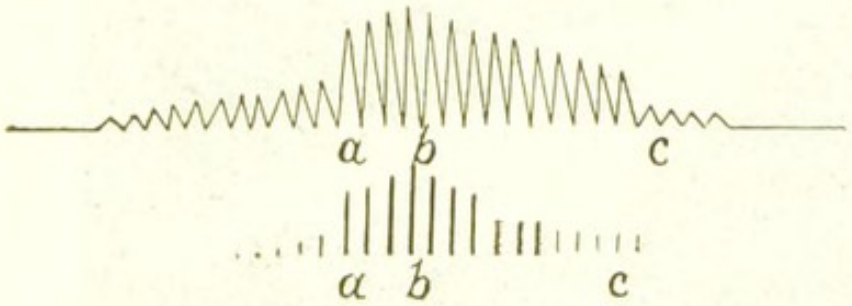


Fig. 47.—The upper part represents the oscillometer movements of the instrument shown in Fig. 46, the lower part the corresponding auscultatory phenomena. (*Modified from Gallavardin.*)

limb to which it is applied. Before any alteration is made in the pressure within the chamber its connection with the armlet must be re-established. Under these conditions it is found that, as the pressure in the system is gradually raised, the oscillations of the pointer are at first very slight. At a certain point, however, where the pressure approximates to the diastolic blood-pressure, the movements suddenly become much larger when the chamber is disconnected, and these large oscillations gradually attain a maximum as the pressure is further increased. Thereafter they slowly fall off, and eventually disappear abruptly when the air-pressure reaches that of the systolic blood-pressure. These movements are diagrammatically represented in Fig. 47, where the large oscillations lie between *a* and *c*. The part to the left of *a* represents the small movements observed when the air-pressure is distinctly less than the diastolic blood-pressure, and that to the right of *c* the disappearance of the oscillations when the air-pressure exceeds the systolic blood-pressure. In practice, while the maximum blood-pressure is usually easily

determined by this apparatus, it is doubtful whether the true minimum blood-pressure is to be taken as corresponding to the manometer reading observed when the large oscillations reach their greatest amplitude at *b*, or—as Professor Pachon himself suggests—at the point *a*, where the first large oscillation is noted as the pressure in the system is raised.

4. **G. A. Gibson's recording sphygmomanometer.**—This consists of an armlet and compression pump

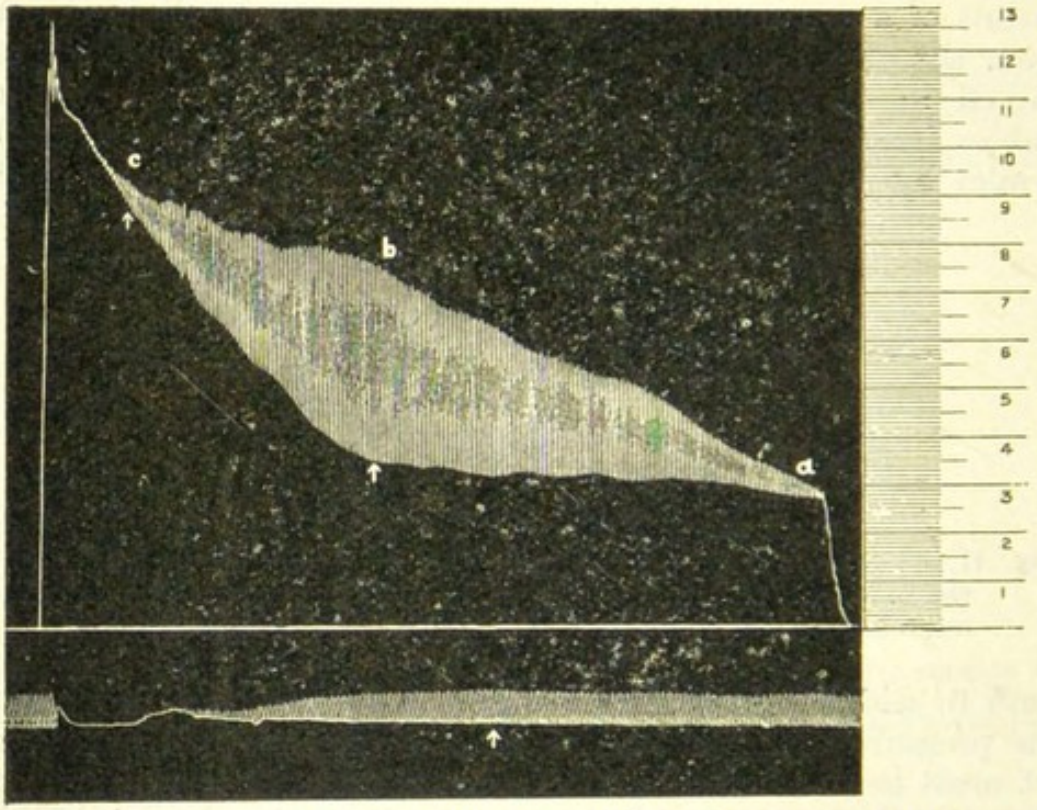


Fig. 48.—Reduced tracing from recording sphygmomanometer.
(Supplied by Dr. G. A. Gibson.)

Below the base line of the manometer-tracing is the pulse-tracing. The arrow beneath it indicates the point where it attains its full amplitude: some observers take this point as corresponding to that of minimum blood-pressure. The scale shows the systolic pressure to be 180 and the diastolic 110 millimetres.

connected directly with a mercurial manometer. A float provided with a recording needle is placed in the open limb of the manometer and traces the pressure curve on a rotating drum. A base line is first marked out by allowing the drum to revolve whilst the pressure is at zero; the pressure of any point on the curve which is subsequently traced is then obtained by measuring the height of the point above the base line and multiplying the result by two as the mercury is depressed in one limb of the manometer to the same extent as it is raised in the other.

A small pneumatic pad or tambour placed on the radial artery at the patient's wrist, or on the brachial just above the elbow, is connected with a recording tambour which inscribes the beats of the pulse on the drum directly below the pressure-recording needle. The drum being set in motion, the pressure is rapidly raised to a point above that at which the pulse-beat is arrested, and thereafter the compressed air is allowed gradually and continuously to escape. The point corresponding to the systolic blood-pressure is marked by the first returning beat of the pulse, and almost at the same moment the pressure-recording needle will be seen to oscillate more freely than before. The oscillations increase in amplitude until a maximum is reached, and thereafter they again diminish, and eventually become quite small. The minimum pressure is assumed to correspond with the largest oscillation, and is measured by the height (doubled) of the centre of that oscillation above the base line. A tracing, reduced to the proportion of one-third, is shown in Fig. 48, where *c* represents the maximum blood-pressure, *b* the minimum blood-pressure, and *a* the lower limit of the large oscillations.

Instead of using a recording apparatus, or reading the range of the large oscillations on a manometer, one can often arrive at an approximate estimate of both the maximum and minimum blood-pressures by combining the use of an ordinary Riva-Rocci instrument with auscultation. The method is as follows: Apply the Riva-Rocci instrument in the ordinary way, but place the chest-piece of a binaural stethoscope or phonendoscope upon the brachial artery just above the bend of the elbow. Raise the pressure in the armlet to a point above that at which the pulse at the wrist ceases to be felt, and then, whilst auscultating, gradually reduce the pressure. It will be found that, as the pressure falls, faint arterial sounds are first heard, then these sounds become slightly louder and are associated, as a rule, with short and soft systolic murmurs. At a slightly lower pressure the sounds will become much louder and sharper, and this increase in intensity is usually abrupt and highly characteristic: the loudness quickly reaches a maximum and then falls off more or less rapidly. Finally the loud sounds disappear, sometimes suddenly, at other times rather more gradually, and are replaced by sounds which are fainter and different in quality, and these in turn soon vanish as the pressure is lowered still further. The first faint sounds accurately mark the return of blood during systole, and thus indicate the maximum blood-pressure; the loud and sharp sounds correspond to the large oscillations of

the sphygmometric oscillometer and afford the same data for the determination of the minimum blood-pressure. (*See* Fig. 47.)

In some cases the mere application of the finger to the brachial artery yields information which closely resembles that obtained by the stethoscope. Usually this procedure is less sensitive than the auscultatory method, but occasionally it proves more exact.

In all the above determinations it is essential to work as quickly as is compatible with accuracy, as compression of a limb induces a rise in blood-pressure. This rise is estimated as amounting to 5 mm. during the first minute, and in long observations under exceptional conditions of blood-pressure may attain as much as 20 mm. In view of this fact an observation should only be repeated after the lapse of a considerable interval.*

It has been shown that instruments whose readings depend on compression of a limb by means of a cuff are fairly reliable, and that the allowance for errors induced by the resiliency of the tissues and the rigidity of the arterial walls does not in ordinary cases exceed 10 mm. It should, however, be noted that in cases where the vessel wall is much thickened the error may be considerably greater. Where there is marked œdema the value of the readings can only be accepted with considerable reserve; and in cases where, as in tetanus, the muscles cannot be relaxed, no reliance can be placed on the records obtained.

The arterial pressure should be observed when the patient, free from excitement, is quietly resting. It should be taken in both the recumbent and standing postures, in order to determine whether the compensation for the influence of gravity is perfect. The pulse-frequency should be noted in each case, and the point at which the compression is applied should be placed on the same level as the heart.

Several instruments have been designed to estimate the blood-pressure in the veins, but, for ordinary work, a fair approximation can be made by slowly raising and lowering the hand whilst watching the filling and emptying of the veins on its dorsum. They will gradually be observed to collapse when the hand is raised to a height of about 5 to 10 cm. above the sterno-xiphoid articulation, and to refill when brought slightly below that level.

* *See*, however, Gallavardin and Haour, *Arch. des Mal. du Cœur*, v. 81 (1912).

VII. THE USE OF THE SPHYGMOGRAPH

For permanent record, and also to aid in the analysis of details, it is desirable to employ a sphygmograph as well as to feel the pulse in all cases where the state of the heart and vessels is an important factor. Numerous types of sphygmograph are now in use, but only Marey's and Dudgeon's will be described here, these being the ones most frequently employed.*

In **Marey's sphygmograph** the pad which rests on the artery acts on a long lever, the farther end of which traces the pulse-curve on smoked paper that is held in a frame driven by clockwork. In **Dudgeon's** the same result is obtained by connecting two shorter levers in such a manner as to magnify the movement of the pad fifty times. The end of the lever in Marey's instrument describes the arc of a circle; in Dudgeon's, owing to the mechanical device employed, the style moves backwards and forwards in a straight line. Of the two instruments, Marey's sphygmograph gives the more accurate trace, but Dudgeon's is easier to apply.

The following description of the method of using each will aid the student in applying them :—

1. **Marey's instrument.**—The patient should sit or lie down. Place the arm support on a table of suitable height beside the patient. Loosen any tight garments about the patient's arm, which must be bared to above the elbow. Place the arm supine on the support, with the back of the wrist at the highest part. Semiflex the fingers. Mark the line of the

* A modification of Dudgeon's sphygmograph has been introduced by Jaquet, and has the advantage of possessing a time-marker actuated by a separate mechanism which enables accurate measurements to be made of the exact duration of the various events recorded in the tracing. As the recording paper often advances with considerable irregularity in speed, this is an important improvement. A larger form of Jaquet's instrument is also made, in which by the aid of tambours the apex beat of the heart, or the respiratory curve, and the jugular pulse can be recorded simultaneously with the radial pulse.

radial on the skin, and draw a cross line where the pad of the sphygmograph is to rest. Wind the clockwork and adjust the paper on the instrument.* Place the pad of the sphygmograph very accurately in position, with the lever pointing up the arm towards the elbow, and fix the instrument in its place by non-elastic bands. Adjust the pressure of the pad, by means of the screw or milled head, until the lever affords the maximum range of movement. Adjust the level of the style, after re-coupling it with the pad, so that the movement of the lever takes place opposite the smoked paper, and see that the style is in contact with, but does not press too heavily on, the latter. Before varnishing the trace, note patient's name, the date (and time of day), whether right or left radial, pulse-rate, respiration, and approximate pressure employed.† Ordinary quick-drying negative varnish, as used by photographers, is employed to fix the trace, the paper being dipped into a jar of varnish, or laid, trace upwards, in a saucer containing some.

2. **Dudgeon's instrument.**—(1) Wind up the clockwork.

(2) Insert one end of the smoked paper (smoked side uppermost) on the right-hand side of the instrument between the roller and the small wheels.

(3) Make the patient hold out either hand open, and in any easy position, palm upwards, the fingers pointing towards you, and tell him not to move the wrist or fingers.

(4) Ascertain and mark the precise spot where the radial artery beats at the wrist.

(5) Slip the band, the free end of which has been drawn through the clamp, over the patient's hand.

(6) Adjust the pressure of the spring.

(7) Place the pad on the artery, the clockwork case being nearest the elbow.

(8) Retain the instrument in place with the right hand; tighten the band sufficiently with the left, and clamp by means of the screw with the right hand. When the band is correctly tightened, the needle will oscillate over the paper. If the tightness is nearly but not quite correct, bend the hand backwards at the wrist to increase the tension, or forwards to diminish it.

* The paper is best smoked over a small piece of burning camphor, which may, if necessary, be moistened with a drop of alcohol.

† The nominal pressure as given by the index on the screw is not even approximately correct, but if the same instrument is always used the results are comparable to some extent.

(9) Set the paper in motion by releasing the catch that controls the clockwork.

(10) Let the paper run through unassisted, and catch it in your hand as it passes from the instrument. Generally the patient's hand must be supported while the tracing is taken.

(11) Stop the clockwork as soon as the paper has passed.

In a pulse-tracing, rise of blood-pressure will be represented by an upstroke, and fall by a downstroke.

Bearing this in mind, the student will readily understand the main outlines of a healthy pulse-tracing. The pressure rises fairly rapidly; therefore the upstroke, when the paper is driven forward at the usual speed, is nearly, but not quite, perpendicular. The percussion wave is quickly followed by what is known as the tidal (or predicrotic) wave; these are not separately distinguishable by the finger in health; the sphygmograph, however, indicates their existence by a notch at the beginning of the downstroke. Thereafter the pressure begins to fall off, but at the moment when the aortic valves close the decrease of pressure is arrested, and a positive (dicrotic) wave is propagated into the vessels; this condition is recorded by a small break in the descent of the downstroke of the tracing. The foot of the notch immediately before the dicrotic upstroke indicates the time when the aortic valves close. After this rise the line again curves downwards, often exhibiting slight secondary oscillations, until a new upstroke marks the arrival of the next pulse-wave. Ordinarily the blood-pressure requires much longer to fall than to rise, hence the downstroke is much less vertical than the upstroke (Fig. 49).

A line joining the lowest points of the upstrokes of successive pulse-beats on the tracing is often known as the respiratory line. In health it is usually straight, but where the blood-pressure is low and the patient is suffering from dyspnœa it becomes undulating, rising

to a higher level during expiration and sinking again during the period of inspiration (Fig. 50).

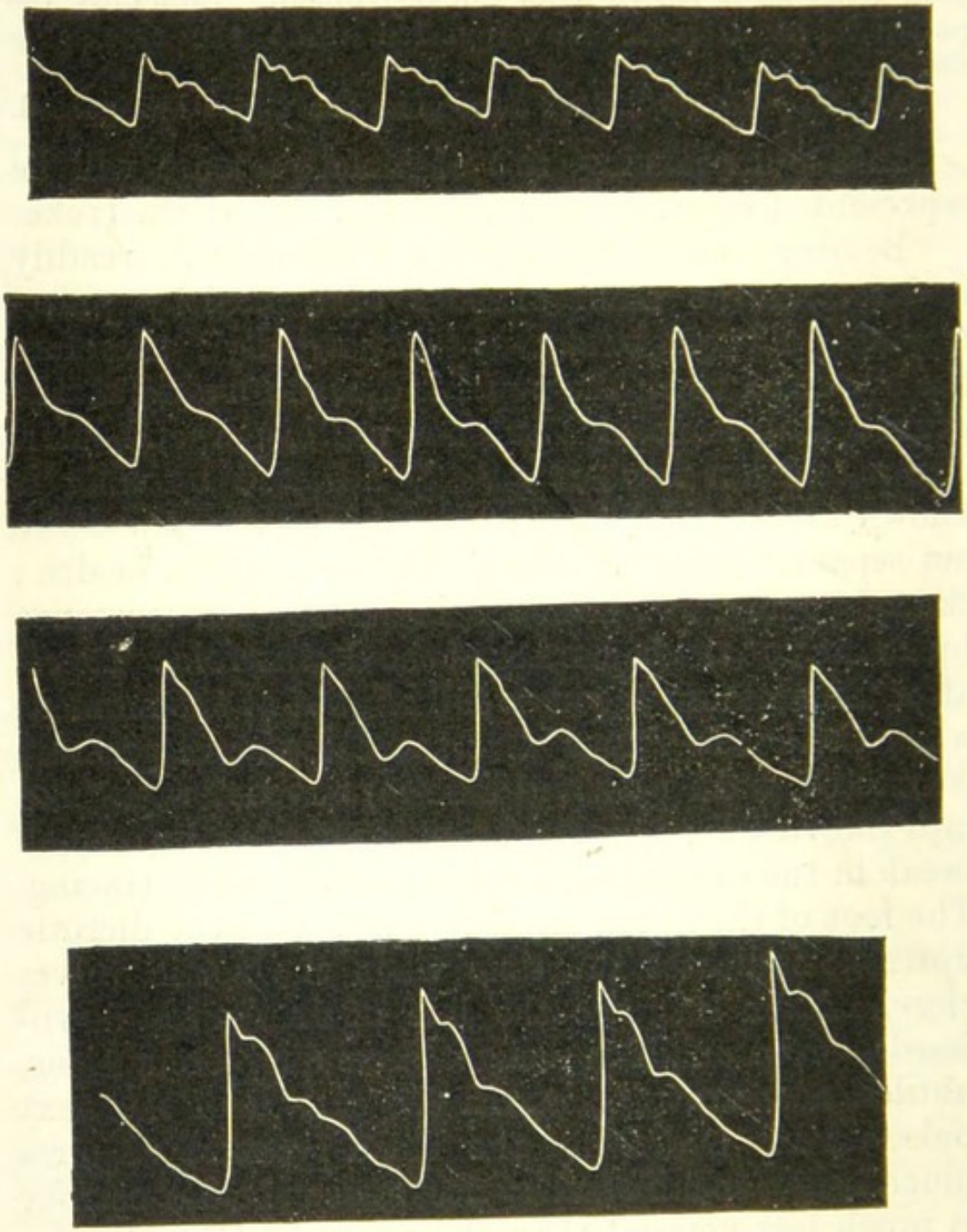


Fig. 49.—Normal forms of pulse. (*Mahomed.*)

In health a pulse-tracing taken with suitable pressure has a sharp apex, a small tidal wave, and a moderately distinct dicrotic wave. A rounded apex, in

most cases, means either excessive pressure of the spring, or bad application of the instrument (*see*, however, p. 197).

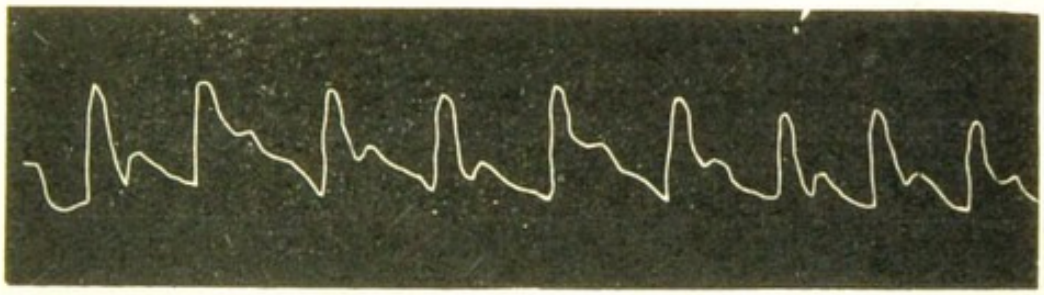


Fig. 50.—Low-tension pulse.

The upstroke is longer and steeper than usual when the ventricle discharges a larger volume of blood than

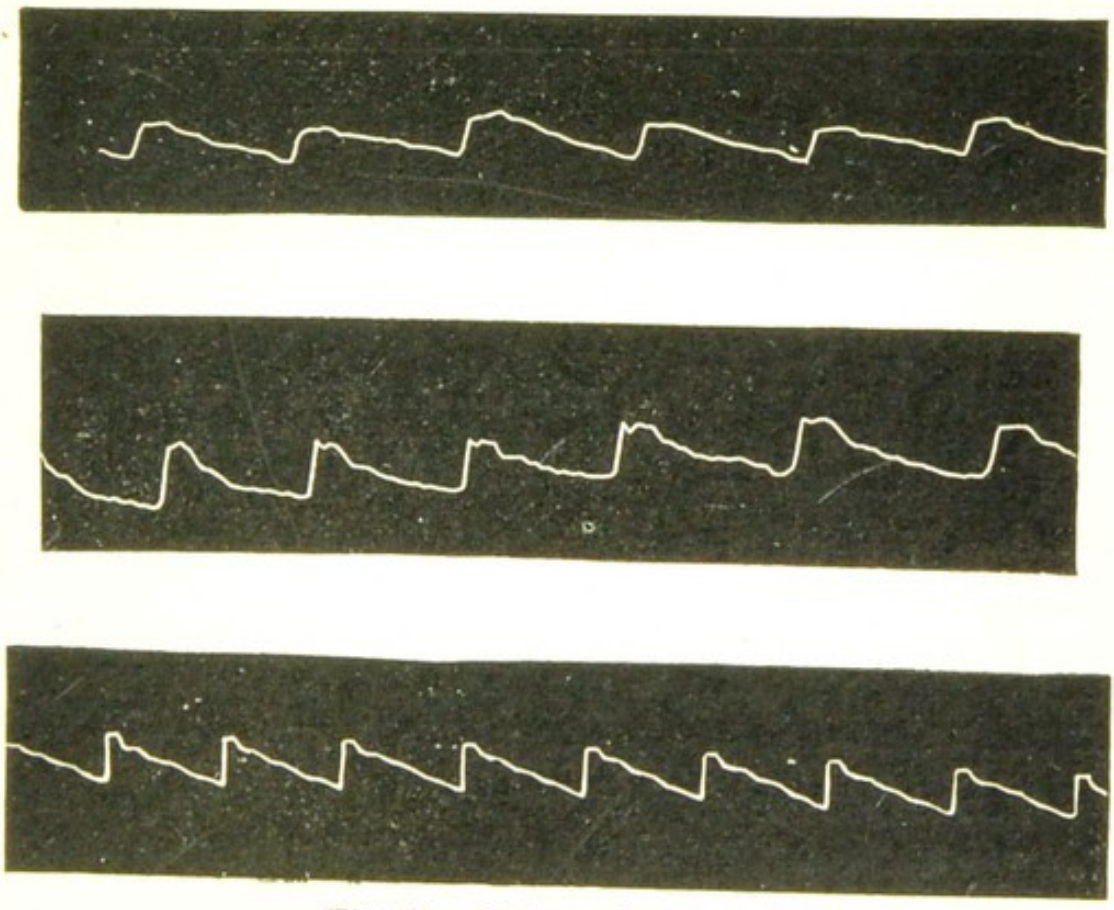


Fig. 51.—High-tension pulse.

normal into the arteries, and when the arterioles are dilated (low tension) (Fig. 50).

The upstroke is shorter and less steep when the heart acts feebly or when the aortic ostium is stenosed, so that less blood than usual is delivered in a given time, and also when the blood-pressure is high and undue opposition is thereby offered to the outflow from the heart (Fig. 51).

In conditions where the minimum blood-pressure is

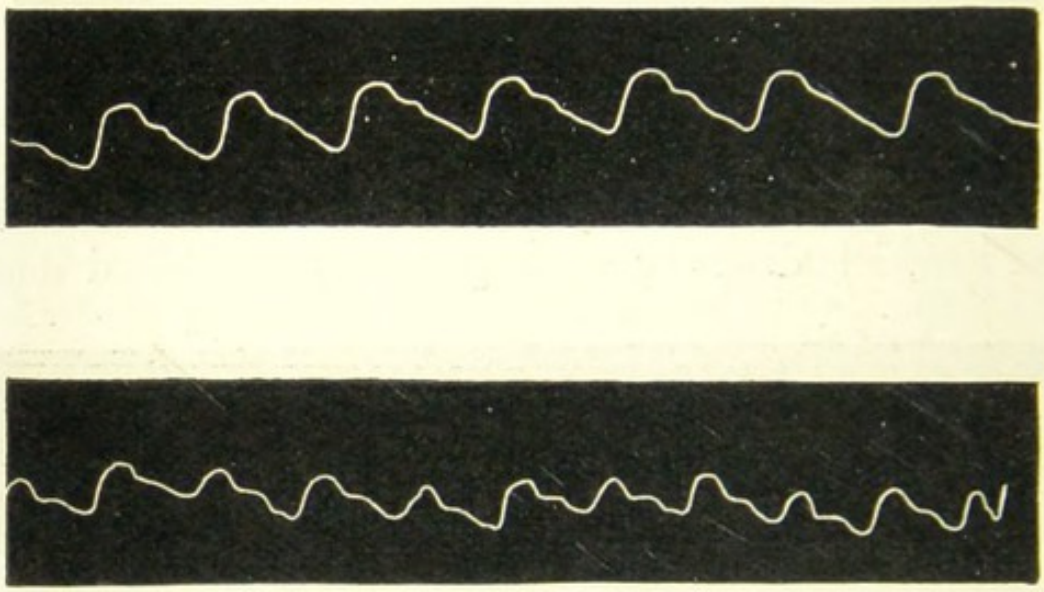


Fig. 52.—Aortic stenosis.

low, the dicrotic wave is well marked; where it is high, the dicrotic wave is small, and secondary oscillations are present.

The following conditions present characteristic tracings :—

1. **Aortic stenosis.** Small amplitude, sloping upstroke, tidal wave well developed, and often higher than primary apex (anacrotic pulse) (Fig. 52).

2. **Aortic incompetence.** Great amplitude, abrupt upstroke, rapid fall, little or no dicrotic wave. This is known as the water-hammer, collapsing, or Corrigan pulse (Figs. 53, 54).

3. **Mitral disease.** Small amplitude, with moderately steep upstroke, secondary waves rather

slight, successive beats unequal and irregular, especially when failure of heart is threatening to set in. In mitral stenosis the vascular tension tends to be higher than in mitral incompetence* (Fig. 55).

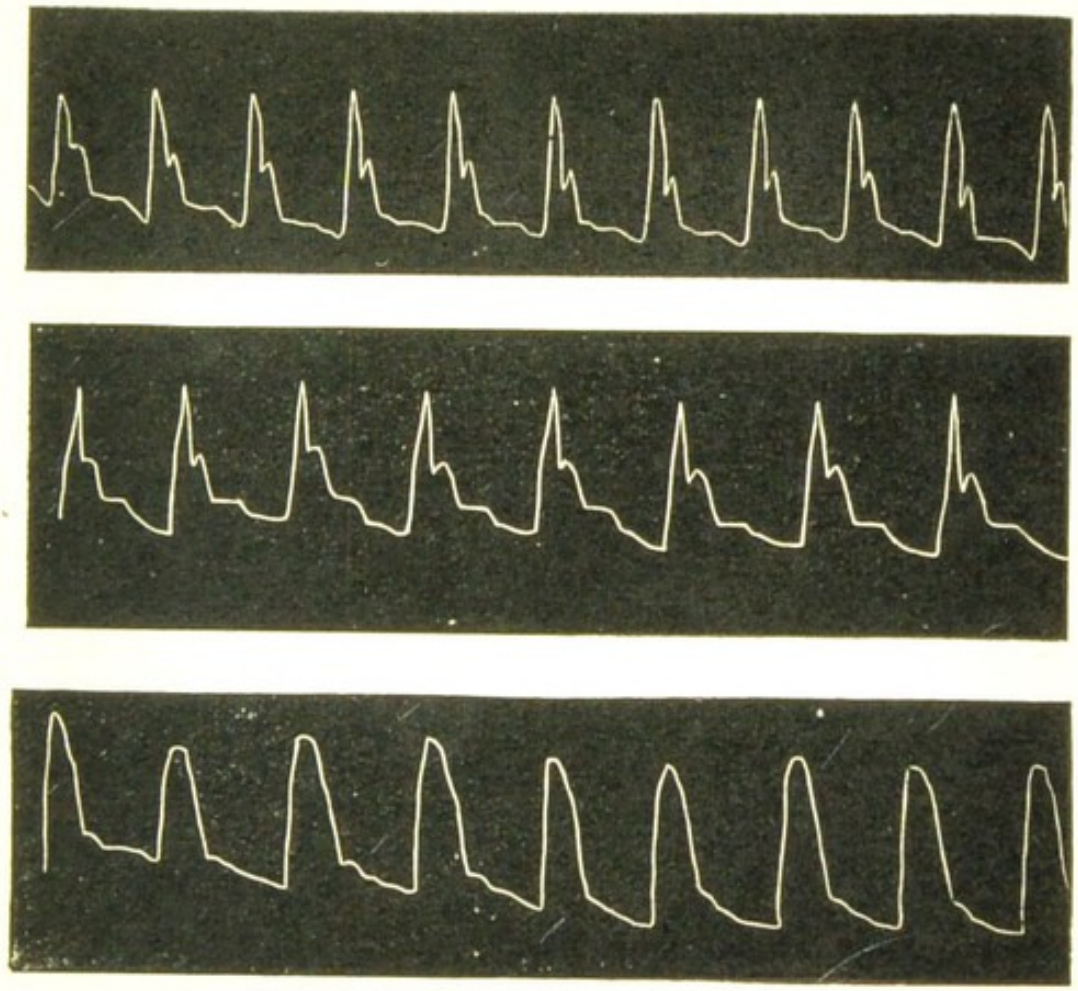


Fig. 53.—Aortic incompetence.

4. **Aneurysm** of the ascending or transverse aorta generally affects the pulse in the implicated radial, where the impulse is delayed, whilst the rise is gradual, the amplitude less, and the apex rounder than in the unaffected radial (Fig. 56).

* The characters of the pulse in mitral disease are very variable, and space forbids any complete analysis of the subject here. In general, however, it may be said that the irregularities are most often due to extrasystoles, but that in certain cases they arise from slight degrees of heart-block, as is evidenced by the lengthening of "the *a-c* interval" (p. 206).

5. **Arterial atheroma** of the great vessels, by abolishing the modifying effect of their elasticity, renders the tracing similar to one taken from the pressure curve of the left ventricle. This is known as the senile pulse (Fig. 57). Aortic atheroma combined with a moderate degree of incompetence gives a pulse which is like that of atheroma, but with sharper apex.

Several types of pulse have received special names; of these the following are amongst the more important :—

1. **Pulsus bisferiens.** The tidal wave is felt separately from the primary impact. The tracing is characteristic, and is often observed in cases of aortic stenosis (Fig. 58).

2. **Pulsus dicroticus.** The dicrotic wave is exaggerated (*see* p. 184) (Fig. 59).

3. **Pulsus anacroticus.** The tidal wave is more forcible than the percussion wave. Consequently the latter, instead of appearing at the summit of the sphygmographic tracing, produces an indentation in the upstroke of the curve whose apex is formed by the tidal wave. The condition is found in pulses where the tension is high, and, like the pulsus bis-

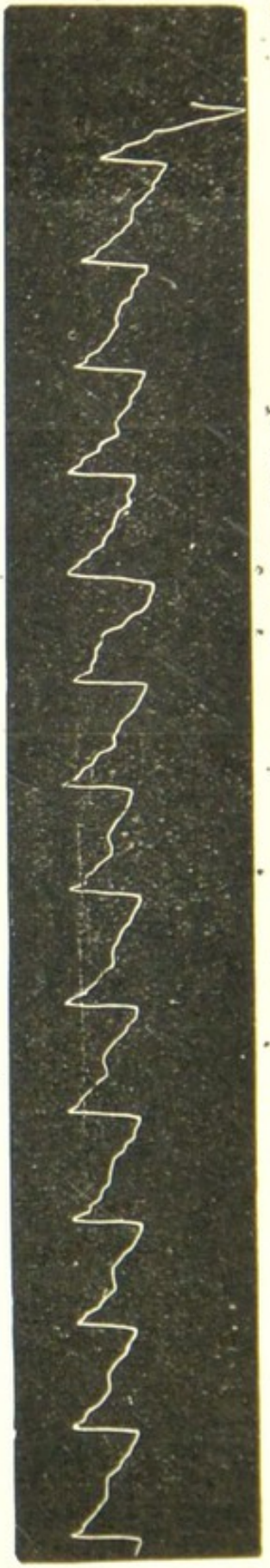


Fig. 54.—Aortic incompetence : compensation established.

feriens, is frequently associated with aortic stenosis. It is most important to distinguish from the true anacrotic pulse those cases where one finds that, from

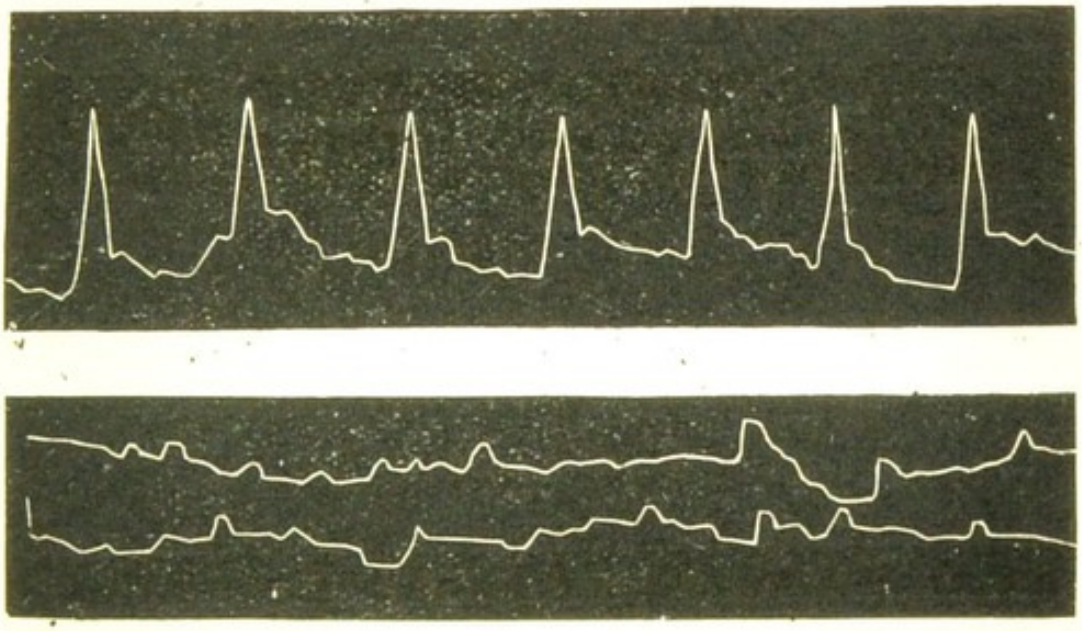
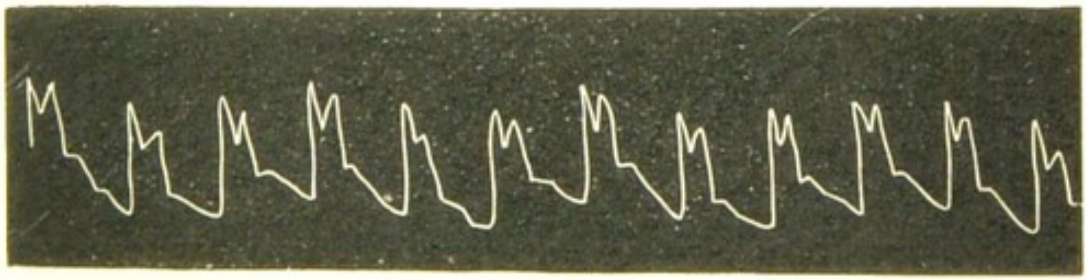


Fig. 55.—Mitral incompetence.

Left side.



Right side.

Fig. 56.—Pulse in aneurysm (left subclavian).

the rapidity of the heart's action and extreme low tension, the pulse exhibits hyperdicrotism.

4. **Pulsus celer.** The pressure is ill sustained; the up and down strokes are therefore abrupt.

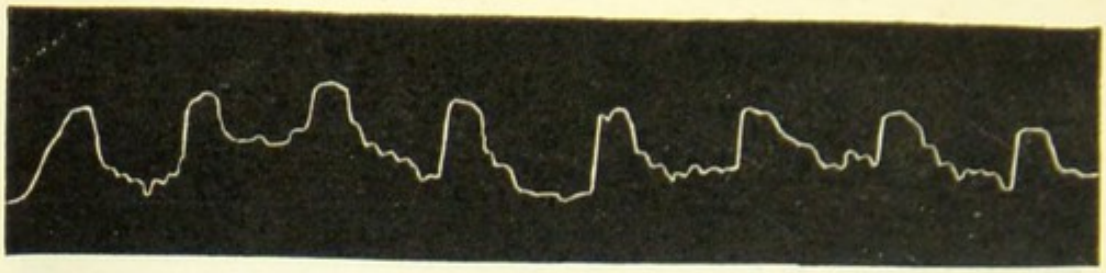


Fig. 57.—Aortic atheroma (senile pulse).

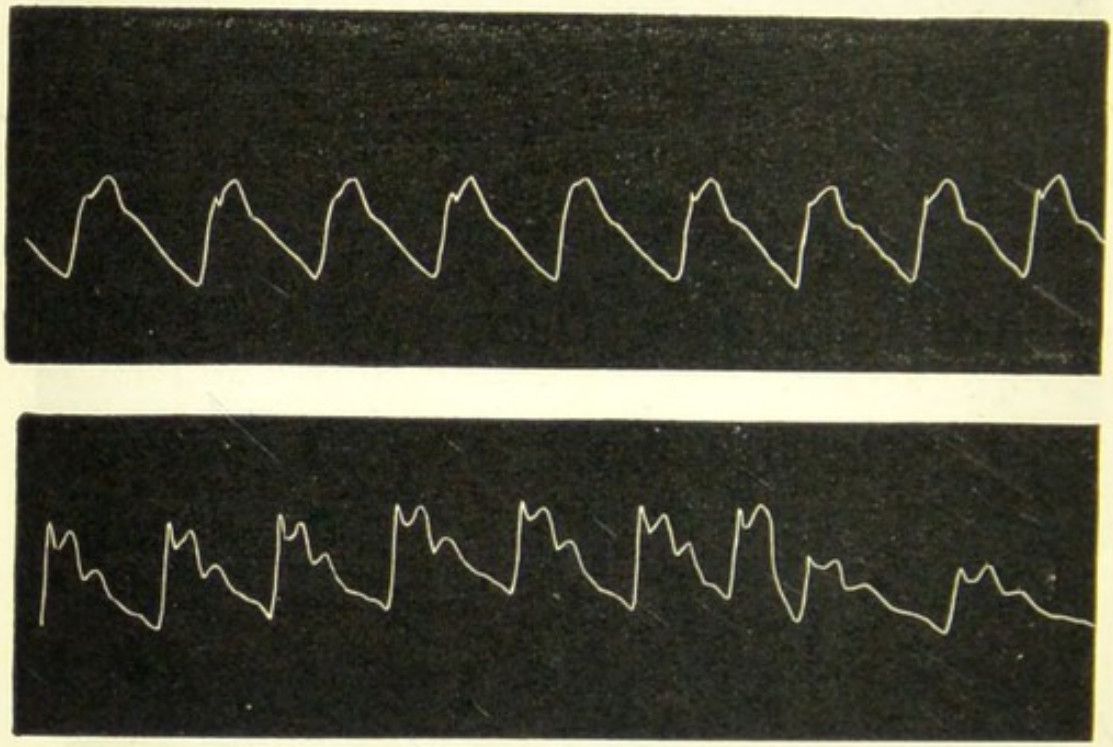


Fig. 58.—Pulsus bisferiens.

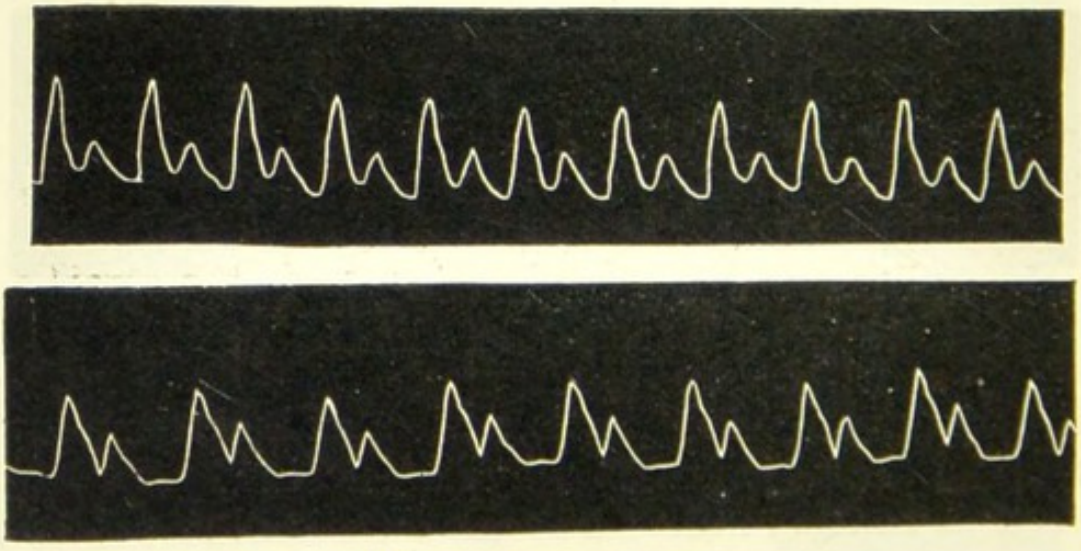


Fig. 59.—Dicrotic pulse.

5. **Pulsus tardus.** The rise of pressure is gradual, the summit is well sustained, and the fall of pressure is slow.

6. **Pulsus bigeminus.** There are two beats and a pause. The two beats may be alike, or they may differ in force (Fig. 44).

7. **Pulsus trigeminus.** Three beats and a pause (Fig. 45). It must be clearly kept in mind that the bigeminal and trigeminal pulses are simply names for clinically observed sequences of pulse-beats. They do not connote any particular state of cardiac derangement, but result from various types of cardiac irregularity which differ in nature and importance.

8. **Pulsus intermittens.** A pulse-beat is absent from time to time in the tracing. It does not follow that the cardiac systole is also entirely absent. Many cases of intermittent pulse are due to special types of "extrasystole."

9. **Pulsus alternans.** The beats are regular in time but unequal in strength. When the alternation persists for a considerable period, it indicates a disorder of the contractile power of the heart muscle, and not merely a passing disturbance of the regulating nervous apparatus. It should be regarded as a grave condition indicating serious embarrassment of the heart, either as a result of previous damage to the heart muscle or of a demand for effort which it can barely meet.

10. **Pulsus frequens.** The pulse is very rapid. The increase in rate may be due to very various cardiac conditions; in some cases it may be traced to the nervous mechanism, in others to undue irritability of the muscle, and in yet others to the presence of repeated pathological stimuli such as have been referred to under "extrasystole." In extreme cases the sequence of contractions may be so rapid that the

fall in pressure succeeding the dicrotic wave is not developed, and the next upstroke of the pulse is superposed upon the summit of the dicrotic wave. In this way the so-called **monocrotic pulse** is produced.

11. **Pulsus rarus.** The pulse is abnormally slow. In some cases the retardation is due to the nervous mechanism of the heart, in others to a failure in the conductivity of the heart muscle. Of the latter a very serious type is that where the auriculo-ventricular bundle of His, which carries the impulse from the auricles to the ventricles, fails to conduct normally, and the ventricular contraction is consequently either delayed or wholly absent, producing in extreme cases the condition described as "heart-block."

12. **Pulsus paradoxus.** The pulse becomes smaller or disappears at the end of inspiration when the patient breathes deeply. It occurs in pericardial adhesion.

13. The pulse is described as **wiry** when the vessels are contracted and the heart-beat is rapid and moderately strong. This may occur in peritonitis. When the heart gets weak, whilst the other conditions continue, the pulse grows **thready,** but at the same time the blood-pressure generally begins to diminish.

14. The pulse is said to be **running** when the vessels are relaxed and the heart's action is weak and fairly rapid.

VIII. THE VENOUS PULSE

In a considerable number of cases where the circulation of blood through the right side of the heart is interfered with, either from valvular disease or from increased blood-pressure in the pulmonary circulation, the embarrassment manifests itself by distension or pulsation in the veins. From their size, nearness to

the heart, and comparatively superficial situation, the veins of the neck offer special facilities for the study of these phenomena.

Whilst inspecting the root of the neck the observer has already had an opportunity of noting these appearances where they exist, but the several varieties and degrees of disturbance in the venous circulation must now be more completely distinguished. A mere flicker of pulsation at the root of the neck when the patient is recumbent is common enough even in perfect health, and must not, in the absence of further evidence, be regarded as indicating any disease of the heart. When, however, the pulsation passes farther up, the case is entirely altered. Two points must then be noted—first, whether there is actual regurgitation, or merely retarded emptying of a full vein during the beat of the heart; and second, the exact moment at which the pulsation occurs with reference to the apex beat. Sometimes there is no difficulty in distinguishing between **regurgitation** and simple **undulation** in the vein. In bad cases of tricuspid incompetence, a mere glance at the patient will at once reveal the nature of the phenomenon. Where the conditions are less urgent, and the distinction is less readily determined, a simple plan is to empty the vein from below upwards by running the finger along it, and then to keep its upper extremity closed by the pressure of the finger, so that no blood can enter it from the periphery.

In cases of simple undulation, the vessel either remains empty and collapsed, or at most refills very gradually as small collateral branches discharge their contents into it. Where, however, there is true pulsation, the valves at the root of the neck have ceased to be competent. As a consequence of this, coupled with the overloaded state of the right heart, a backward wave of blood is forced into the vein, which refills from

below with a series of bounds corresponding to the beats of the heart, and after three or four pulsations is again distended and pulsating as vigorously as ever, although the observer's finger continues to maintain the peripheral closure.

Generally, venous pulsation at the root of the neck is best observed when the patient is lying down with the head only slightly, if at all, raised on a pillow. In extreme cases, however, it may happen that in this attitude the veins remain so distended throughout the whole cardiac cycle that the phenomena are thereby masked, and in such instances the pulsation may return when the patient once more assumes the erect position.

The venous pulse occurs in two principal forms, known respectively as the auricular and the ventricular type of venous pulse. In the **auricular type** a wave appears in the vein at the instant of auricular systole. Soon after this wave has reached its summit, and when it is beginning to fall off, it is reinforced by a second wave which corresponds in time to the occurrence of the carotid pulse, and which is probably due, at least in part, to that cause. Thereafter the pressure again falls, and often reaches its minimum towards the middle of ventricular systole. The pressure once more rises near the end of ventricular systole, the exact time of this second rise depending on the rate at which the auricle and veins fill up with blood, when the outlet into the ventricle no longer exists, or when regurgitation occurs; it then falls off somewhat during ventricular diastole, to rise once more at the commencement of the next cardiac cycle with auricular systole. These conditions may be traced in relation to the events of the cardiac cycle, when the sequence will resemble that of Fig. 60, where the upper tracing represents diagrammatically the movement of

the recording lever of a suitably adjusted tambour applied to the pulsating vein, whilst the lower part indicates the events in the cardiac cycle which correspond to the various parts of the curve so obtained.

The details of individual tracings differ greatly; in some one part of the curve, in others another part, being specially emphasized. It therefore becomes necessary, in order to identify the various oscillations in the

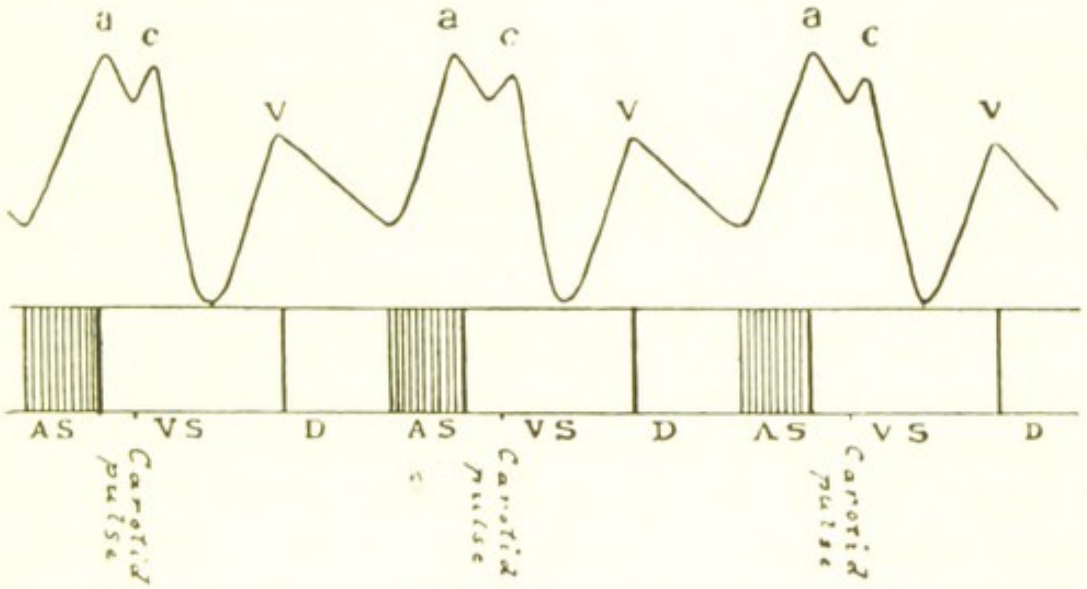


Fig. 60.—*See* text.

venous tracing, to take a simultaneous tracing of the radial pulse on the same strip of paper. In all doubtful cases a further double tracing should be made of the radial and carotid pulses, so that their time relation to each other may be ascertained. As a general rule, the carotid wave will be found to occur about one-tenth of a second earlier than the radial pulse. A wave preceding the carotid wave by about one-fifth of a second is ordinarily due to auricular systole; and a wave following it, to the accumulation of blood in the veins and auricle during ventricular systole.

The **ventricular type** of venous pulse is characterized by the absence of the wave which occurs during auricular systole, and by the fact that the principal

summits of such waves as are present fall within the period of ventricular systole. The occurrence of this form of venous pulse is proof either that the auricle is unable to contract and propel its contents into the ventricle, or that, in consequence of some derangement of the mechanism of the heart-beat, the auricle and ventricle are contracting simultaneously.

In venous pulse-tracings the various waves are usually designated by the following letters. The auricular wave is marked *a*. The carotid pulse-wave is marked *c*. The wave occurring during ventricular systole is marked *v*. The trough or negative wave between *a* and *c* is marked *x*. The trough between *c* and *v* is marked *x'*. The trough between *v* and *a* is marked *y*. A small wave is sometimes noted between *v* and *a*, and has been specially investigated by Fredericq, Hirschfelder, and Gibson. Its causation is uncertain, and it is usually designated by the letters *b* or *h*. This *b* wave must be distinguished from an *a* wave which has not been followed by a ventricular contraction. The interval which separates the commencements of the *a* and *c* waves is known as the *a-c* interval. It is of importance as determining the time which elapses between the commencements of the auricular and ventricular contractions. If this is found to exceed 0·3 second, one is justified in assuming that the ventricular systole is delayed, and one may infer that the conducting mechanism of the heart has become impaired. If, however, there is any doubt of the character of the wave which is supposed to be the *a* wave, a diagnosis should not be made until an electrocardiogram has been taken. The summit of the *v* wave is important as marking the commencement of ventricular diastole, for as soon as the tricuspid valve opens the wave begins to fall.

The venous pulse is best studied by means of Mackenzie's

ink polygraph.* This consists of a metal case containing two independent pieces of clockwork, one of which actuates a time-marker indicating fifths of a second, the other drives a long strip of paper beneath the writing pens at a rate which can be controlled by the observer. The movements of the radial pulse are transmitted to the polygraph through a pad attached to a spring, similar to that of a Dudgeon sphygmograph, which is applied to the wrist by a small splint. The movements of the pad are directly transmitted to a tambour which is attached to the splint, and thence by means of rubber tubing to a receiving tambour fixed to the polygraph, and provided with a lever and writing pen. The venous pulse is transmitted to a similar recording tambour connected by tubing with a small shallow cup or "receiver," which is applied to the pulsating area. There are thus simultaneously inscribed on the strip of paper a time record and two tracings, one of the arterial, the other of the venous pulse. In using the instrument the two writing pens must be made to move across the strip of paper before the clockwork is started, in order that a line may be marked by each from which measurements can subsequently be made. As the pens do not move in a straight line, but along the arc of a circle, the necessary allowance must be made for this if the measurements are to be accurate.

The receiver which is used to transmit the jugular pulsations must be very lightly applied. If it is held too firmly against the neck the tracing will show the carotid and not the venous pulse.

In certain circumstances a venous pulse of totally different origin may be present. This is known as the **centripetal venous pulse**, and is due either to great dilatation of the arterioles, so that the arterial pulse passes through the capillaries and is visible even in the veins, or to an aneurysmal varix, whereby direct communication occurs between an artery and a vein, and the pulse-wave of the artery reaches the vein by this channel. The observer can have no great difficulty in recognizing the nature of such centripetal pulsation.

* Supplied by Mr. Shaw, instrument maker, Padiham, Lancashire.

IX. SYMPTOMS AND SIGNS OF THE PRINCIPAL DISEASES OF THE HEART

1. **Valvular diseases.** (*a*) **Aortic incompetence.**—The patient complains of attacks of giddiness, is often pale, and his arteries pulsate. Capillary pulsation may be observed. The apex beat is displaced downwards and outwards, and has a heaving character. The left border of the heart is farther out than usual. A murmur accompanies the second sound, as has been elsewhere described, and the pulse exhibits a "water-hammer" character.

(*b*) **Aortic stenosis.**—Here the apex beat is rather weak, and the displacement less notable than in the last condition. The arteries are small, and the tension of the pulse, before compensation is destroyed, is somewhat high. Vertigo or fainting fits are not uncommon. The murmur is described on p. 167. The aortic second sound is very weak.

(*c*) **Mitral incompetence.**—This condition is often associated with dyspnœa, which is the first symptom of which the patient may complain. The face is apt to be cyanotic. The apex beat is of moderate strength, and is frequently displaced outwards. On auscultation, in addition to the characteristic murmur, one finds marked accentuation of the second sound in the pulmonary area.

(*d*) **Mitral stenosis.**—Here the stress falls chiefly on the left auricle and right side of the heart. The apex beat is therefore only slightly displaced outwards, and is not unduly vehement. The murmur is very frequently accompanied by a thrill, and in many cases the second sound as heard at the apex is reduplicated. The pulse is at first not notably affected, but gradually becomes irregular and feeble as compensation fails.

(*e*) **Tricuspid incompetence** which is usually

secondary to mitral disease, is associated with dilatation of the right auricle, and consequently with outward displacement of the right border of the heart, with venous and hepatic pulsation, and with dropsy. The murmur has already been described ; the second sound in the pulmonary area is weak. The patient usually suffers from very decided dyspnœa.

2. **Non-valvular diseases of the heart.**—In certain diseases the heart has an increased amount of work imposed upon it. In these circumstances, unless the work is too great for its reserve energy, it hypertrophies, the hypertrophy being usually associated with some degree of dilatation. Eventually such cases terminate in failure of compensation, with its accompaniments of dyspnœa, urinary deficiency, dropsy, and cyanosis. But another large group of cases exists where the disease originates in the nerve mechanism of the heart, or in the myocardium. Such diseases manifest their presence by disturbances in the rate and rhythm of the heart, and our knowledge of their course has been greatly advanced by the use of the methods of examination that are afforded by polygraphic tracings and electrocardiograms.

Three conditions of great clinical importance demand special reference, namely, extrasystoles, heart-block, and auricular fibrillation.

Extrasystoles are the expression of premature contractions of the heart muscle, and may arise in the ventricle, the auricle, or the junctional tissues between the auricle and ventricle. When the extrasystole is of *ventricular* origin the tracing of the radial pulse shows either a premature beat which is followed by a longer pause, so that the next beat falls at the point at which it would have occurred had there been no disturbance of the ventricular rhythm, or else the abnormal beat is not represented at all in the radial pulse-tracing, but the interval between the beats which have reached the artery is twice its usual length. The venous curve shows that the auricular waves have followed one another at regular

intervals, without being disturbed as regards their time relations by the premature ventricular contraction; but if it happens that a ventricular systole occurs at the same time as the auricular contraction, the auricular wave will be unusually large and prominent.

When the extrasystole is of *auricular* origin the tracing of the radial pulse exhibits the same characters as in the case already discussed, except that the compensatory pause is usually absent. The venous curve shows that the carotid wave is preceded by the auricular wave in the normal way, but the auricular wave occurring prematurely may become blended with the *v* wave, which then disappears as a separate wave from the tracing, whilst the *a* wave becomes unusually prominent.

When the extrasystole occurs in the *junctional tissues* the most important feature is a disturbance of the length of the *a–c* interval in the venous curve; but the appearances vary greatly, and depend on the exact point in the junctional tissues where the abnormal stimulus originates. Much experience is needed to arrive at reliable conclusions in this group of cases.

The manifestations of **heart-block** vary greatly, according as the block is complete or partial. The radial pulse is generally slow, the systolic blood-pressure high, and the range of pressure between systole and diastole considerable. In the venous pulse-curve the *a* waves occur at regular intervals, but in the case of partial heart-block only some of them are associated with resultant *c* and *v* waves, whilst in complete heart-block the waves due respectively to the auricular and ventricular contractions occur regularly in respect to others of their own kind, but with no correspondence at all as regards the relation of auricular and ventricular systoles.

Auricular fibrillation is characterized by complete arrhythmia of the pulse, while the venous pulse-curve assumes the ventricular type, and shows in many cases numerous and rapid oscillations during the diastolic period.

Pericarditis is characterized by pain in the chest, more or less fever, and by friction of a to-and-fro character when effusion is scanty. When more fluid has been poured out, the area of dullness is triangular and oversteps the 2nd left interspace. The first indication of an increase in the area of dullness is often found to the right of the sternum immediately above

the point where the liver and heart dullness meet. The apex beat is internal to the left limit of dullness, and may be weak or quite unable to be felt. The pulse may be greatly affected, and it is by this disease that the "pulsus paradoxus" (p. 202) is produced.

Intrathoracic aneurysm of the aorta leads in some cases to a tumour at the upper part of the chest, which is dull on percussion, and in which expansile pulsation can often be observed. It may also affect the character and synchronism of the pulses at the two wrists. The auscultatory phenomena are very variable. It produces numerous symptoms of involvement of different structures upon which it comes to press as it enlarges.

Cardiac asthma.—This condition is characterized by attacks of severe dyspnœa, often lasting for several hours on end. The patient is so breathless that he requires to sit up, and may have to call the accessory respiratory muscles into violent exercise. It is distinguished from bronchial asthma by the state of the heart, which is generally dilated, while the apex beat is weak. The pulse also is small, rapid, and irregular, and the dyspnœa is not of the pure expiratory type which characterizes asthma of respiratory origin.

CHAPTER V

CLINICAL EXAMINATION OF THE BLOOD

Enumeration of Red Blood-Corpuscles

This may be done by means of either a Thoma-Zeiss hæmocytometer or by Strong's method. The former is the method more commonly used; we shall describe it first.

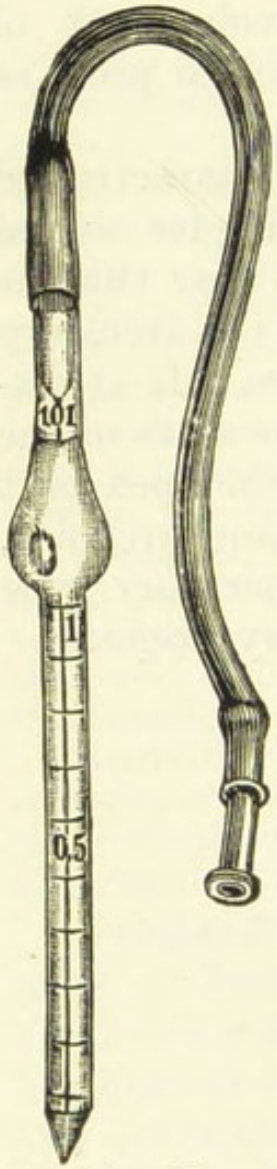


Fig. 61.—Hæmocytometer pipette (*Thoma-Zeiss*).

1. **Thoma-Zeiss hæmocytometer.**—The instrument consists of a mixing pipette (Fig. 61) suitably graduated and a counting slide. Cleanse the lobe of the patient's ear with ether and dry it. Make a puncture on the lower border of the lobe by means of an ordinary surgical needle. The needle should be inserted with a sudden stab—not slowly—and the blood must flow freely. On no account must the blood be *squeezed* out, as it is then always diluted by lymph squeezed out of the tissues. Slowly suck up blood by means of the pipette till either the mark 0·5 or 1 is reached. If one should happen to go a little beyond the 0·5 mark, the column of blood should be gently blown down to the proper point. If blood has been sucked past the mark 1, it has reached the mixing chamber, and the process must be begun over

again. Having charged the pipette, *wipe the end of it* on a clean cloth, and plunge it at once into the diluting fluid (Appendix, 18), which should be standing ready in a small, wide-necked bottle with the stopper out. Suck up the diluting fluid as far as the mark 101. While this is being done, the pipette should be gently rotated so as to start the mixing. Seize the pipette firmly by its ends between the forefinger and thumb, and shake thoroughly for about one minute. This induces a thorough mixing of the blood with the fluid. It must be remembered that the column of diluting fluid which occupies the capillary part of the pipette does not enter

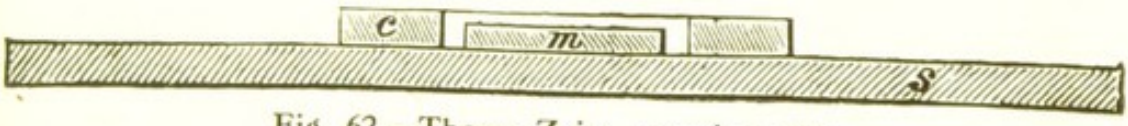


Fig. 62.—Thoma-Zeiss counting slide.
s, slide; *m*, platform; *c*, wall of trench.

into the mixture. Hence, if blood is sucked up to 0·5, the dilution produced is in the proportion of 1 in 200, whereas if blood is taken up to the mark 1 the dilution is only 1 in 100. The former degree of dilution is to be preferred in most cases. The finger should now be removed from the pipette, and the diluting fluid in the capillary tube blown out. After a few drops of the diluted blood have been shaken out, a small drop is transferred to the counting slide (Fig. 62). The latter consists of a small platform (*m*) bounded by a trench which is surrounded by a glass slab (*c*). On the surface of the platform microscopic squares are ruled, each having an area of $\frac{1}{400}$ sq.mm. Special cover-glasses,* carefully ground, are supplied, which rest upon the glass slab, a space being left between the under-surface of the cover and the surface of the platform, which space is exactly $\frac{1}{10}$ mm. in depth.

The drop of diluted blood should be placed in the

* Ordinary cover-glasses must not be used.

centre of the platform, and should be of such a size that when the cover-glass is placed in position the drop is flattened out so as to cover most of the surface of the platform, *but yet without any of it flowing over the edge into the trench*. It requires a little experience to enable one to take just the proper size of drop. It is important that the cover-glass should lie quite flat upon the glass slab. This can best be achieved by previously washing both it and the slab with caustic potash, so as to remove all grease, and then rubbing them with soft chamois leather. The cover must be lowered into position by means of a needle. One recognizes that the cover-glass is lying properly by the appearance of concentric colour (Newtonian) rings between it and the slab; they should be produced by gently stroking down the cover-glass with the handles of two mounted needles. The rings should remain when the cover is simply *lying* on the slab without any pressure being exerted; they are best seen by looking horizontally along the surface of the cover. Unless the rings are seen, one cannot be sure that the space between the cover and the platform is exactly $\frac{1}{10}$ mm. in depth. Having placed the drop in position, and the rings being visible, one should set the preparation aside for two minutes or so, to enable the corpuscles to settle. It should then be examined with the low power to see whether any air bubbles or foreign bodies are present, and whether the corpuscles are distributed with fair uniformity throughout the field, after which the high power (No. 2 eye-piece and $\frac{1}{6}$-in. objective) is used for counting. The microscope must be vertical and should be provided with a condenser and a diaphragm. The light should be gradually cut off until the red cells become clearly visible. The little squares will be seen to be marked off into sets of sixteen by double ruling (Fig. 63). Should the lines marking off the

squares be only dimly seen, it may be necessary to intensify them. This is best done by rubbing the surface of the platform with a little finely powdered graphite—e.g. the scrapings from a very soft lead pencil—and then polishing it with soft chamois leather.

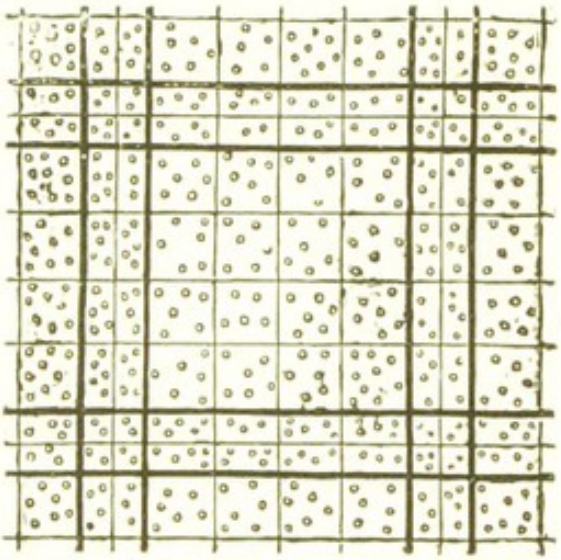


Fig. 63.—Microscopic view of Thoma-Zeiss counting slide, showing divisions.

For enumeration of the red cells, at least four sets of sixteen squares should be counted. The squares in each set should be gone over systematically in horizontal rows of four at a time. Of the corpuscles which lie *upon* the lines bounding the row, only those on the upper and on the left-hand lines should be counted. The number of corpuscles in each of the four sets should be approximately equal.

Calculation.—Count the corpuscles in each of the four horizontal rows from above downwards. The total is the number of corpuscles in sixteen squares. Count in this way four sets of sixteen, and divide the total by sixty-four, which gives the average of corpuscles in one square. But the dimensions of this square are $\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000}$ c.mm. Therefore, if there be x corpuscles in this dimension, there will be 4,000 x in 1 c.mm. But the blood was diluted 200 (or 100) times. Therefore, in 1 c.mm. of blood there will be 4,000 $x \times$ 200 (or 100) corpuscles.

Suppose, for example, that one finds a total of 384 corpuscles in the sixty-four squares. This gives an average of six corpuscles per square, or $6 \times 4{,}000$; i.e. 24,000 per c.mm. of *diluted* blood,

or 4,800,000 per c.mm. of pure blood, if the dilution was 1 in 200.

The constant error in the Thoma-Zeiss instrument is less than 1 per cent. of the total result. The variable error depends upon the number of corpuscles counted. By counting 200 corpuscles it amounts to 5 per cent. of the total; by counting 5,000 it amounts to only 1 per cent. To count the whole 256 squares takes a novice about half an hour. This usually means counting 1,200 to 1,600 corpuscles. The coefficient of error is then about 2 per cent.

2. **Strong's method (modified).**—The necessary instruments consist of a capillary tube provided with two marks placed close together (the tube at the lower mark contains 5 c.mm., from the upper mark delivers 5 c.mm.), a pipette graduated for 995 c.mm., and a small bottle provided with a well-fitting stopper. Take up 995 c.mm. of diluting fluid (Appendix, 19), and transfer it to the bottle. Draw up 5 c.mm. of blood to the lower mark in the capillary tube, wipe the outside of the tube free from blood, blow out the blood into the diluting fluid, and wash out the inside of the tube by sucking the mixture of blood and fluid several times up and down the tube. The blood is now diluted in the proportion of 1 in 200. Shake the bottle thoroughly and transfer a drop of the mixture to the Thoma-Zeiss counting slide. Proceed as described under the Thoma-Zeiss hæmocytometer.

The advantages of this modification are that the mixture of blood and diluting fluid is more intimately made, that in the stoppered bottle the mixture can be readily transported and counted at leisure, and the same mixture can be used for an accurate estimation of the leucocytes (*see* p. 217).

The **normal number of red corpuscles** is about 6,000,000 per c.mm. In some full-blooded

adults this number may be exceeded. In females, even in health, it is usually rather smaller (about 5,500,000). The normal number of red corpuscles is surpassed in cases of prolonged cyanosis.

The number is reduced in all forms of anæmia. In chlorosis, however, the number of corpuscles may be normal, or nearly so.

Enumeration of Leucocytes

1. **By the Thoma-Zeiss hæmocytometer.** —A special pipette is supplied for this purpose with the Thoma-Zeiss instrument. It is used in precisely the same manner as the red-corpuscle pipette, but permits of a lesser degree of dilution of the blood. The best diluting fluid to employ is one containing 1 c.c. of glacial acetic acid in 100 c.c. of water, to which enough of a watery solution of methyl green or gentian violet has been added to give the mixture a decided colour. The advantage of this mixture is that it dissolves all the red cells while it stains the nuclei of the white. One can thus easily count the whites, and at the same time note roughly the relative numbers of the unipartite and multipartite nucleated varieties.

It is important that a large drop of blood should be allowed to exude before one begins to fill the pipette. The blood should be sucked up to the mark 0·5, the end of the pipette wiped, and diluting fluid taken up to the mark 11.

Owing to the relatively large calibre of the pipette, the blood is apt to run out of it. It is well, therefore, to keep the pipette in a horizontal position as soon as one has filled it with blood.

The blood and the fluid are mixed as already described. This produces a dilution of 1 in 20. A drop is then placed on the counting slide with the same precautions that were observed in the case of the red cells.

In this case the whole sixteen sets of sixteen squares should be counted, or 256 squares in all. Instead of going over the squares in rows of four, a whole set of sixteen can easily be counted at one time. A movable stage greatly facilitates the enumeration.

One should note on a piece of paper the number of leucocytes with multipartite and with rounded nuclei respectively in each set of sixteen squares. In this way one gets a rough idea of the proportion of each variety present, and by adding them together the total number of white corpuscles is obtained. The *calculation* is made in the same way as that of the red corpuscles, it being borne in mind that each of the 256 squares counted represents $\frac{1}{4000}$ c.mm. of diluted blood, and that the dilution is much less than in the enumeration of the reds (1 in 10, or 1 in 20). For example, if there be 20 leucocytes in the 256 squares, this represents an average of $\frac{20}{256}$ per square, or $\frac{20}{256} \times 4{,}000$ per c.mm. of diluted blood, or 6,250 per c.mm. of pure blood if the dilution is 1 in 20. This is about the normal number.

In leukæmia, where a very large excess of leucocytes is present, one can easily count the red and the white cells in the same drop. For this purpose a 3 per cent. solution of common salt just coloured with a gentian violet is to be preferred for diluting the blood. This stains the nuclei of the whites, and at the same time preserves the reds. Toisson's solution (Appendix, 20) may be used similarly. The dilution and calculation are the same as for the red cells.

2. **By Strong's method.**—The same mixture and the same capillary tube are used as for the enumeration of the red cells. The stoppered bottle is well shaken, and 5 c.mm. of the mixture is drawn to the upper of the two marks in the capillary tube. The end of the tube is wiped and held lightly against the centre

of a clean slide, the tube being at right angles to the slide. The contents of the tube are gently blown out on to the slide in the form of a drop. The drop is allowed to dry, is then stained for five minutes in filtered hæmalum (or in any simple nuclear stain), washed in tap-water, again dried, and mounted in cedar-wood oil. The leucocytes are stained blue, the red cells being unstained. All the leucocytes in the drop are then counted in the following manner: A metal disc with a central square aperture is placed in the eye-piece of the microscope. The metal disc may be replaced by a paper or cardboard one of home manufacture, all that is necessary being to obtain a square field. The $\frac{1}{6}$-in. objective is used, and the microscope should be fitted with a mechanical stage. The edge of the drop is easily defined, and by moving the stage the leucocytes in the top segment of the drop are counted from left to right. By marking a red cell the drop can be moved down exactly one square field, and by proceeding backwards and forwards across the drop no leucocytes need be omitted. The total number of leucocytes thus counted comprises those present in 5 c.mm. of blood diluted 1 in 200. To arrive at the number of leucocytes per c.mm. of undiluted blood, the number counted should therefore be multiplied by 40. In cases of leukæmia a further dilution is necessary, and for this two additional pipettes are provided, one to contain 495 c.mm. and the other 55 c.mm. From the original mixture 55 c.mm. are taken and added to 495 c.mm. of diluting fluid in a stoppered bottle; 5 c.mm. of this mixture are put up and counted, and the number found is multiplied by 400.

After use, the diluting pipettes should be thoroughly cleaned. A little trouble in this is repaid by saving of time and annoyance when next they come to be used. They should be washed out (1) with distilled

water, (2) with absolute alcohol, and (3) with ether. A stream of air should then be blown through till one is sure that the glass ball in the chamber moves freely without tending to adhere to the sides. To save time in these manipulations, the rubber tube may be taken off and the fluid blown out through the wide end of the pipette. Coagulated blood may be removed from the capillary tube by means of a horse-hair. If the blood adheres firmly to the pipette, it may be removed by repeated rinsing with strong alkali or acid, or it may even require to be digested away with pepsin.

The **number of leucocytes in normal blood** is about 6,000 per c.mm., or 1 to 600 reds. The number varies, however, within considerable limits even in health. The normal proportion of leucocytes with divided nuclei to those with rounded nuclei is about 2 to 1.

A *physiological leucocytosis*, in which the small cells with rounded nuclei (lymphocytes) are both absolutely and relatively increased, occurs in infancy and after meals. In the *pathological leucocytosis* met with in acute inflammatory conditions, the increase affects chiefly the polynuclear neutrophil cells, and these may come to be ten or more times as numerous as the others. A relative increase in the number of the large lymphocytes occurs in malaria. The condition of the leucocytes in *leukæmia* will be referred to later.

Estimation of Hæmoglobin

For the estimation of the amount of hæmoglobin one has the choice of several instruments :—

1. **Gowers' hæmoglobinometer.** — Place a couple of drops or so of distilled water in the little graduated test tube supplied with the instrument. Get a large drop of blood from the ear, and fill the pipette with it up to the mark. Then dip the end of the

pipette into the distilled water in the tube and gently blow out the contained blood. Mix, and go on adding water drop by drop, comparing the colour from time to time with that of the standard tube. The latter is filled with tinted gelatin, and represents the colour of

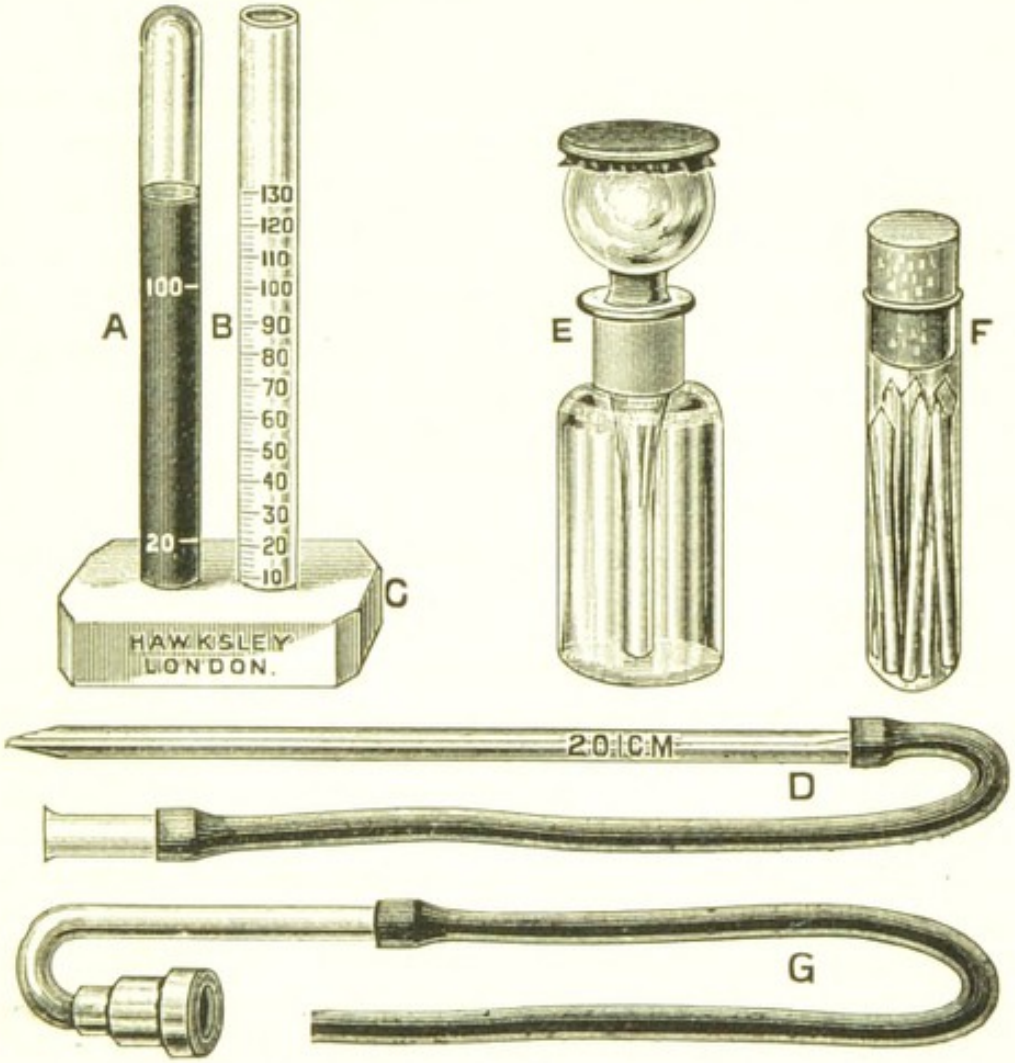


Fig. 64.—Haldane's hæmoglobinometer.

A, standard blood solution; B, graduated mixing tube; C, rubber stand; D, pipette; E, bottle for distilled water; F, lancets; G, tube and cap for fixation to gas-burner.

blood which contains a normal amount of hæmoglobin when diluted in the proportions effected by the instrument. The comparison should be made both by transmitted light, care being taken to hold both tubes level with the eye, and by reflected light, the tubes being held side by side against a sheet of paper. Good day-

light is indispensable. Stop adding water when the tint in the two tubes is the same, and read off the level at which the mixture stands in the graduated tube. If this be at (say) 60, then the blood contains 60 per cent. of hæmoglobin. The mean of the dilution which is just too much and that which is just too little is the correct point.

2. **Haldane's hæmoglobinometer.** — Haldane has modified Gowers' instrument by using as a standard of comparison, instead of gelatin tinted with picrocarmine, a 1 per cent. solution of blood containing the average percentage of hæmoglobin found in the blood of healthy men, and saturated with carbonic oxide. It has an oxygen capacity of 18·5 per cent. as determined by the ferricyanide method, and is both definite and permanent.

The instrument is shown in Fig. 64. It is used as follows :—

Sufficient water is first placed in the graduated tube to dilute the blood as far as safely possible. A puncture is then made in a finger or the lobe of an ear, and the capillary pipette (which must be clean and dry) at once filled to a little beyond the mark 20 from the drop of blood obtained. The point of the pipette is wiped, and dabbed on any convenient surface until the contained blood stands exactly at the mark. The blood is then gently blown out into the graduated tube, where it sinks ; the pipette is rinsed with the water in the graduated tube and withdrawn. The piece of rubber tube attached to a gas-burner is now introduced into the graduated tube to near the level of the water, and gas allowed to pass for a few seconds. As the tube is withdrawn (with the gas still passing) the end is closed with the forefinger, and the liquid made to pass up and down in the tube—not violently shaken—at least a dozen times, so as to saturate the hæmoglobin with CO. During this manipulation the tube is held in a handkerchief, otherwise it will become heated and liquid will spurt out when the finger is withdrawn. Water is now added drop by drop with the pipette stopper, the tube after each addition being inverted, until the point is reached at which the tints of the liquids in the two tubes are just equal. In judging of the equality the tubes should be

held against the light from the sky, or, if artificial light be used, from an opal glass shade. It is also absolutely necessary to transpose the tubes repeatedly, otherwise serious errors may arise. The level is read off on the graduated tube after half a minute has elapsed since the last drop added was mixed with the rest of the liquid by inverting the tube. The observation is repeated after the addition of another drop of water, and if necessary another, until the point is reached when the tints are again unequal. The true result is the mean of the readings giving equality. The error in any single determination ought not to exceed 1 per cent.

The result obtained is the percentage actually present of the average proportion of hæmoglobin in the blood of healthy adult men. The blood of healthy men, however, contains more hæmoglobin on an average than the blood of healthy women and children. Women give an average of 89 per cent., and children of 87 per cent., of the proportion in men. The results may be expressed in terms of oxygen capacity (the number of volumes of oxygen taken up in combination from air by 100 volumes of blood), if it be borne in mind that 100 per cent. on the scale corresponds to an oxygen capacity of 18·5.

The advantages of the modifications introduced into the original method of Gowers are : (1) That the standard solution is a definite one, so that an instrument can be verified at any time by making a determination with ox-blood of which the oxygen capacity has been determined by the ferricyanide method ; (2) that the standard solution is permanent ; (3) that the apparatus can be used with equal correctness by daylight and by artificial light.*

3. **Oliver's hæmoglobinometer** (Fig. 65).—The apparatus adopted by Dr. Oliver is founded on the colorimetric principle. But the applications of that principle are modified in these ways :—

(1) Double transmission of light (or reflected light) is used instead of single transmission.

(2) A standard white background is selected on which the solution of the blood and the standard colours rest.

* If the capillary pipette becomes blocked or soiled by coagulated fibrin, use the brass wire in the case from the wide end of the pipette, or a horse-tail hair, to remove it. (On no account use iron or steel wire.) To dry the pipette, suck air through it. Always examine the pointed end of the pipette before using it : it should be smooth and rounded ; if sharp or angular it is broken.

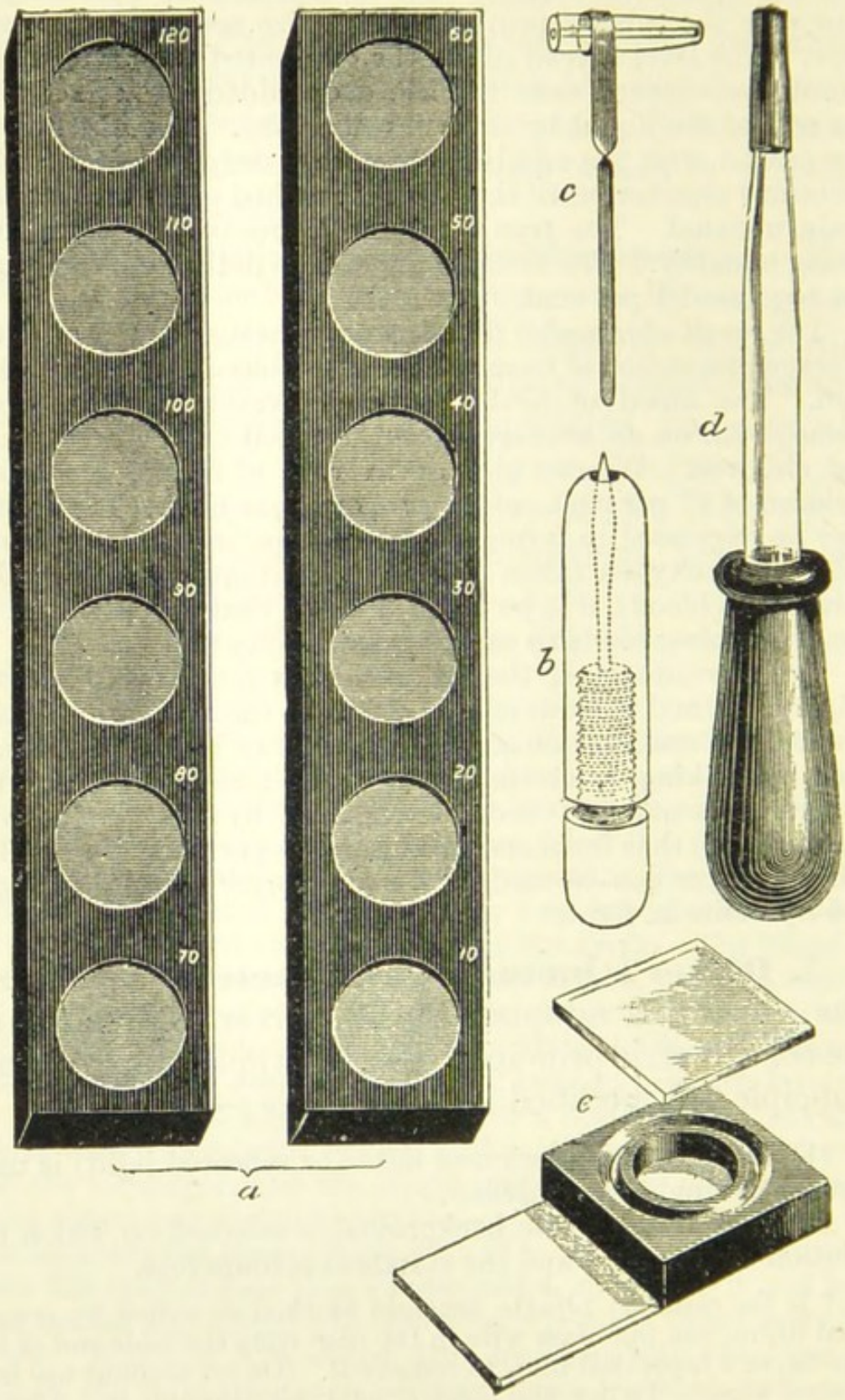


Fig. 65.—Oliver's hæmoglobinometer (*see* text).

(3) The standard is presented as a series of definite gradations.

(4) The colour of the blood solution is compared with that of the standard in camera.

The apparatus consists of an automatic blood-measurer, a mixing pipette, the blood cell and cover-glass, the sets of standard gradations, the riders, the camera tube, the light, a bottle of antiseptic fluid, the lancet, needles and thread.

The automatic blood-measurer (*c*) has a capacity of 5 c.mm., and fills readily by capillary attraction. The handle is useful for stirring together the blood and water in the blood cell.

The mixing pipette (*d*) is provided with a rubber nozzle which fits over the polished end of the blood-measurer, and ensures the complete rinsing out of the blood with the first few drops of water.

The blood cell (*e*) is of more than sufficient capacity to ensure the complete liberation of the hæmoglobin. It yields a blood solution of rather less than 1 per cent. when filled level with the rim. It is itself the measure of the amount of water to be added, and it is quite easy to fill it accurately.

The standard gradations (*a*) are arranged as circular discs in two slabs—six in each ; and they represent the divisions of 10 degrees of the scale from 10° to 120° inclusive.

The riders are small squares of tinted glass provided for the reading of the degrees intervening between each of the standard gradations.

The daylight standard is less adapted to the finer readings than the candle-light one, because the value of each rider when used with it is doubled, i.e. No. 1 rider becoming equal to 2° in the upper half of the scale and 4° in the lower half ; therefore the candle-light standard is preferable for such readings, and when it is used each rider has an equivalent value in the six stronger grades = 1°, and a double value in the six weaker grades = 2°.

The light.—For observation by artificial light it has been found that the small-sized wax candles known as " Christmas candles " are the most satisfactory in affording the most suitable intensity of light, and one, moreover, which is sufficiently uniform.

How to make an observation.—The bore of the blood-measurer is first of all dried out by passing through it a needle threaded with darning-cotton, and then the polished point is presented to the drop of blood. Care must be taken to see that the pipette is really quite filled, and, if it has been necessary to re-apply it to the drop, it should be observed whether there is

a break in the column of blood. The rubber nozzle of the mixing pipette, charged with water, is adjusted over the polished end of the pipette, and the blood is washed into the blood cell by pressing through the water drop by drop. The handle of the pipette is then used as a stirrer, and the further additions of water are made to impinge on it, for it serves to graduate the size of the drops required accurately to fill the cell. A final thorough mixing with the handle will be required, and perhaps another slight addition of water may be necessary to secure a level filling. The cover-glass is then adjusted, when a small bubble should form, a sure sign that the cell has not been overfilled. The blood cell is now placed by the side of the standard gradations, and the eye quickly recognizes its approximate position on the scale. Then the camera tube will more accurately define it. If it is found that the blood solution is matched in depth of colour by one of the standard grades, the observation is at an end; but if it is observed to be higher than one gradation but lower than that above it, the blood cell is placed opposite to the former and the riders are added to complete the estimation.

In all tintometric observations, take a standard time for looking down the camera tube (a ten-seconds observation is most convenient).

4. **Tallqvist's method.** — In this method, which has the merit of great simplicity and ease of application, a drop of blood is allowed to fall upon a piece of standard blotting-paper and compared in good daylight with a paper colour scale of tints ranging from 10 to 100 per cent., the blood-stain being moved along the scale till a match is found.* The comparison should be made as soon as the stain has lost its humid gloss and before it is thoroughly dry (Cabot). The method, however, gives only roughly approximate results and cannot be recommended.

One can also state the percentage of hæmoglobin in terms of the amount contained in each corpuscle, this being known as the **colour index.** Thus, if the number of red cells be 20 per cent. of the normal and the hæmoglobin 10 per cent., then the hæmoglobin

* The scale is supplied by Messrs. Allen & Hanburys.

value of each corpuscle is $\frac{1}{2}\frac{0}{0}$ or half normal. In calculating the percentage of red cells, 5,000,000 is for convenience commonly reckoned as the normal number of red cells per c.mm. or 100 per cent. The importance of this method of expressing the facts is seen when one recollects that the total amount of hæmoglobin in the blood may be diminished while the amount in each corpuscle is really above the normal. This happens in some forms of anæmia.

The last point to be remembered in making blood estimations is that, as far as possible, all observations on the same individual should be carried out under the same conditions as regards time of day, taking of food, etc. This is important, as it is found that the composition of the blood is temporarily altered by the taking of food, or by the occurrence of profuse sweating, diarrhœa, etc.

Microscopical Examination of Blood

Blood may be examined (1) fresh, (2) stained.

1. **Blood examined fresh.**—This simple procedure should never be omitted, since much valuable information may be derived from it. To obtain the blood, hold a cover-slip lightly on a slide, one edge of the cover-slip being exactly flush with one edge of the slide. Hold these edges against a drop of blood; the blood will then flow between slide and cover-slip. Examine at once with the diaphragm of the microscope partly shut down.

In the case of normal blood, the red corpuscles will be observed to range themselves in rouleaux as one watches, clear spaces being left between in which the white cells and little clumps of aggregated platelets may be seen. Any abnormality in the shape or size of the red cells or in the formation of rouleaux should be noted. Distorted red cells—pear-shaped, indented,

budded, etc.—are known as poikilocytes ; red cells larger than normal are called megalocytes ; smaller than normal, microcytes. All these abnormal shapes and sizes may occur in any form of severe anæmia. One can also see if any large excess of white corpuscles

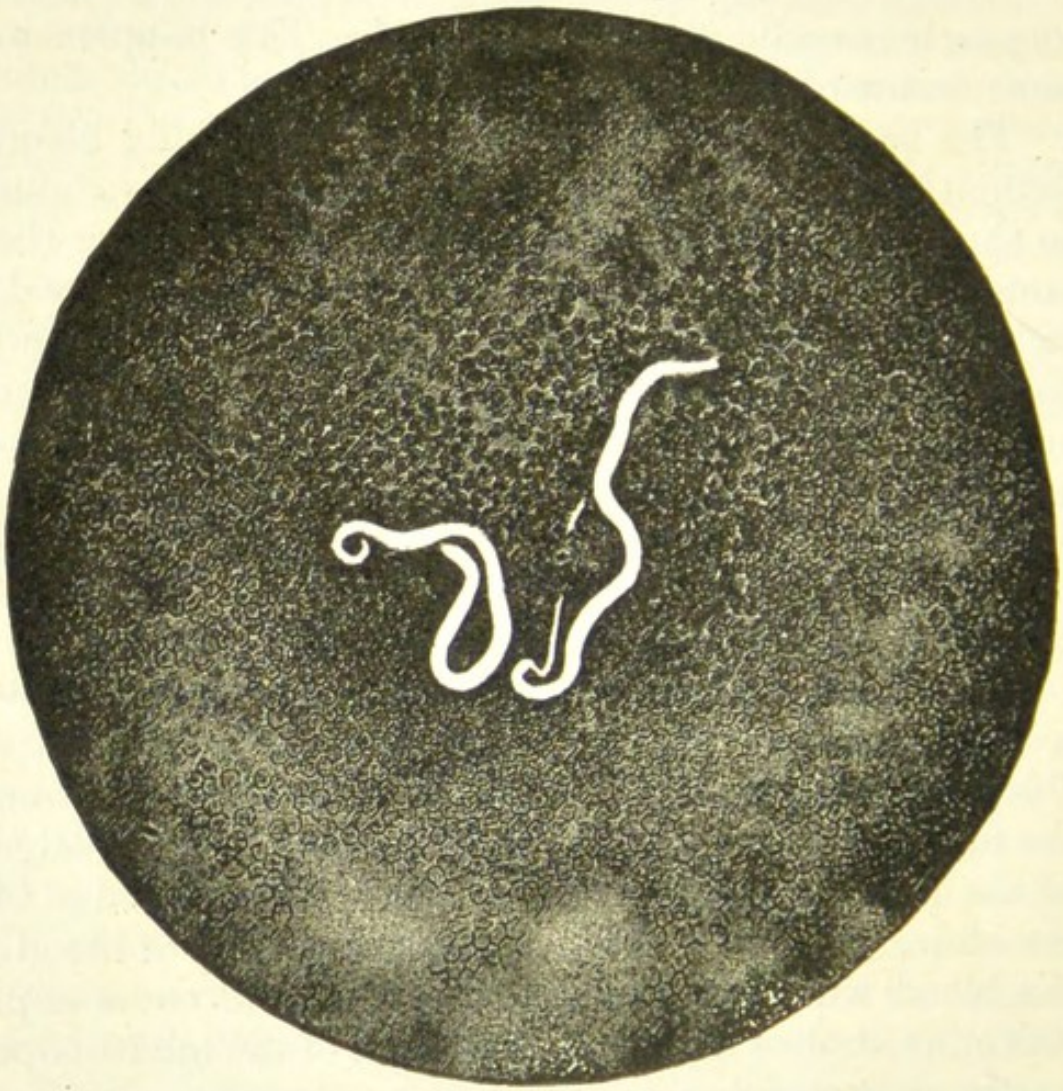


Fig. 66.—Filaria nocturna ; × 160. (*After Patrick Manson.*)

is present. The presence of abnormal elements should be noted. Among these are abnormal varieties of white cells, more easily recognized, however, in stained specimens.

Sometimes particles of pigment can be noticed amongst the corpuscles. This condition, known as

melanæmia, is found occasionally in chronic malaria. During a malarial attack the parasites can readily be seen and the activity of their pigment granules noted.

The **spirillum** of relapsing fever and the **trypanosoma** of sleeping sickness can be recognized by this method, as also the **Filaria sanguinis hominis.** This last can be seen, with a low power, moving about

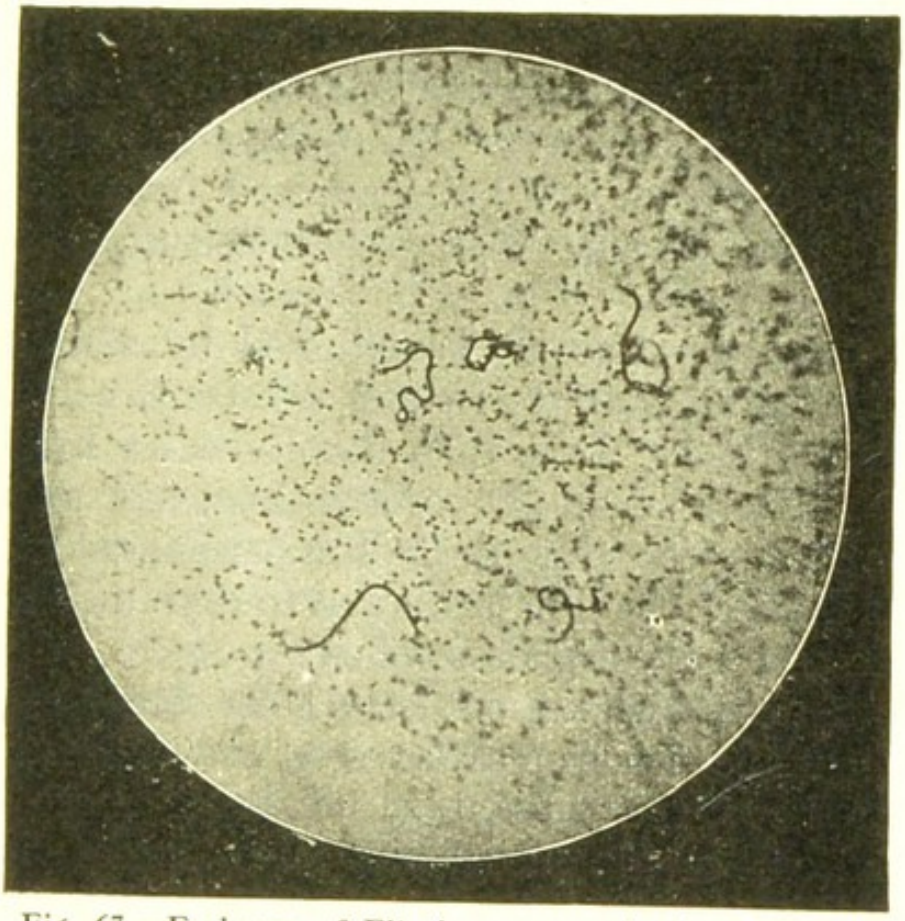


Fig. 67.—Embryos of Filaria nocturna in blood; × 50.
(*From an original photomicrograph.*)

among the red cells. The parasites average about $\frac{1}{75}$ in. in length, and are about as broad as a red blood-corpuscle. (Figs. 66, 67.) They remain alive for a surprisingly long time even at ordinary temperatures, especially if the preparation is kept from drying by being sealed with a little vaseline. The following is the method recommended by Manson for their demonstration :—

Spread out a thick drop of blood on a slide by means of a needle, and allow it to dry. It may then be preserved indefinitely. When the parasites are to be demonstrated, immerse the slide in a solution of 1 drop of saturated alcoholic fuchsin in 1 oz. of water. Stain in this for one or two hours. If, on examining the film, it is found that the blood is very deeply stained, one must decolorize by means of dilute acetic acid (4 drops of acetic acid to 1 oz. of water). The specimen may be examined either wet or dry, and with or without a cover. On searching it with a low power, the filariæ will be recognized by their being very deeply stained. The preparation is apt to fade after a few days.

A more rapid result is obtained by staining the film for half a minute in a 2 per cent. solution of methylene blue. It is then decolorized a little with dilute acetic acid as above described, and examined with a low power while wet. If a permanent preparation is desired, the film is allowed to dry and a drop of balsam and a cover-glass applied.

2. **Examination of blood in films.** — Films may be made either on slides or on cover-glasses. The former have the advantage of being more easily cleaned and manipulated, but cover-glasses give the best results in skilled hands. The slides should be of colourless glass, thin, and with ground edges. The cover-glasses should be $\frac{3}{4}$ in. square, and as thin and flexible as possible. It is important that both slides and cover-glasses should be entirely free from grease. To ensure this the cover-glasses should be dropped one by one into an enamelled iron dish containing 10 per cent. chromic acid and boiled for twenty minutes. They should then be tipped into a shallow basin, and water allowed to run on them till the washings are colourless. After this they are covered with spirit, and

finally transferred with forceps to a wide-necked stoppered bottle containing absolute alcohol. When required for use they should be picked out with forceps, excess of alcohol drained off, and the remainder got rid of by passing through a flame. They should finally be rubbed with a clean handkerchief. Slides may be cleaned in the same manner, but it is sufficient to polish them with the finest emery paper and then to place them in absolute alcohol till required. The alcohol is best removed by wiping dry with a clean cloth.

If ordinary slides or cover-slips have to be cleaned in a hurry, glacial acetic acid, followed by water and alcohol, gives good results.

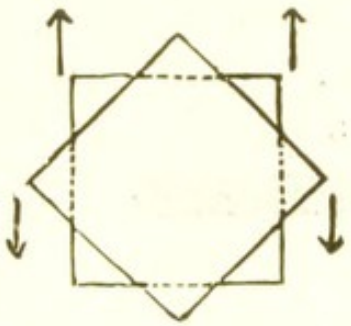


Fig. 68.—Method of making slides on cover-slips. (*After Daniels.*)

How to make films. (1) *On cover-slips.*—The surface of the cover-slips must on no account be touched by the fingers. They may be held by their corners between the thumb, middle and index fingers, but it is preferable to use forceps—a clamp forceps for the lower cover-slip and a fine-pointed forceps for the upper. Clean, dry, and prick the lobe of the ear ; wipe away the first drop of blood, and when another about the size of a large pin's head has appeared, touch its apex with the upper cover-slip and lightly drop it diagonally on to the surface of the lower. Directly the blood has spread, separate the slips with the utmost rapidity, avoiding any pressure or lifting (Fig. 68). If the separation is delayed the slips tend to stick and the films are useless ; if made too soon the resulting films are small and thick.

(2) *On slides.*—Apply one end of a slide to a drop of blood, place the slide on some firm, smooth surface,

such as a polished table without a cloth, holding it in position with the thumb and index finger of the left hand. The narrow edge of a second slide is placed in the drop and held there till the blood has spread across it ; it is then drawn slowly over the whole length of the first slide. The inclination of the second slide to the first should be at an angle of 45°, and there should be no pressure whatever between the two surfaces ; this can be ensured by holding the second slide between the thumb on one side and the index and middle fingers on the other, allowing the tips of the thumb and index

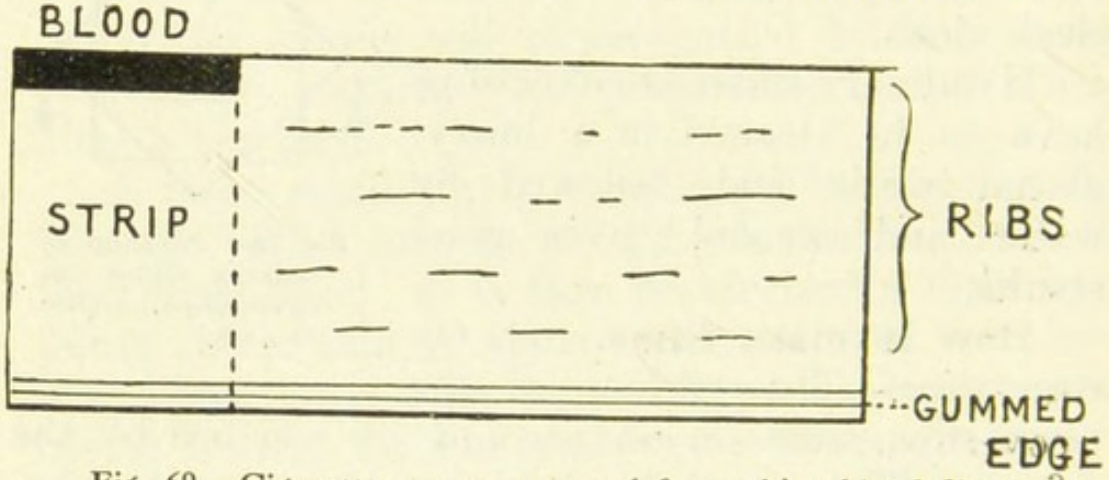


Fig. 69.—Cigarette paper prepared for making blood films.
(*After Howard and Pakes.*)

finger to rest on the table. The more slowly one slide is drawn over the other the thinner is the resulting film. Even spreading of the film is aided by warming the first slide in the flame of a spirit-lamp immediately before applying it to the drop of blood. After the blood is spread it should be dried by being waved rapidly in the air to prevent undue shrinkage of the cells.

Fair results may be obtained by the use of gutta-percha tissue or cigarette paper.

Take a piece of *smooth* gutta-percha tissue about 2 in. long and 1 in. broad. Fold it slightly along its long axis so that one side becomes somewhat convex.

Pass one end of the convex surface lightly across the top of the drop of blood, and immediately lay it flat on the slide near one end. When the drop has spread itself out, draw the gutta-percha tissue flatly along the surface of the slide. If cigarette paper * is used, cut strips across its long axis, each about ½ in. broad, and as long as the paper is wide (Fig. 69). Pass the edge of the strip across the summit of the drop of blood, lay it on the slide, and, when the drop has spread, draw it along. Use a fresh strip for each slide.

Fixation of the film.—Films may be fixed either while still wet or after drying. They may be fixed wet by exposure to formol vapour for twenty minutes; after drying, by immersing in absolute alcohol for fifteen minutes. Fixation of the films when dry is provided for by the methods of Jenner and Leishman, since the stains are dissolved in absolute alcohol.

How to stain the film.—Either of the two following methods gives excellent results:—

1. *Jenner's stain.*—The stain consists of a 0·5 per cent. solution of a specially prepared crystalline compound of methylene blue and eosin in pure methyl-alcohol. Films are made on cover-slips in the usual way. So soon as they are dry a few drops of the solution are poured on, and they are covered with watch-glasses to prevent evaporation and precipitation of the stain. Pour off in one to four minutes. Rinse in *distilled* water till pink (this takes five to ten seconds). Dry rapidly high over a flame or by waving in the air. Mount in xylol balsam. In a successful film the red corpuscles are terra-cotta-coloured; nuclei are blue, platelets mauve, the granules of polynuclear cells and myelocytes red, mast cells dark violet, bacteria, filarial and malaria parasites blue.

* The "Tarlene" or "Zig-Zag" papers are best. Ordinary glazed notepaper will also do, but cigarette paper is preferable.

Leishman's stain.—This is a simplification of the method of staining first introduced by Romanowsky. The stain consists of a compound of alkaline medicinal methylene blue and eosin, extra B.A. (Grübler), dissolved in pure methyl-alcohol in the proportion of 0·5 per cent. The dry film is well covered with the stain, which should be evenly distributed over the entire slide or cover-glass. At the end of one minute, double the quantity of distilled water is carefully added and mixed with the stain by means of a clean glass pipette. At the end of seven minutes the mixture is poured off and the film covered with distilled water only for two minutes. The water is then washed off with fresh distilled water, and the film gently blotted dry with clean blotting-paper. When dry it can be mounted in xylol balsam.

Examination of the film.—In a good film the corpuscles should be spread out evenly, no rouleaux being seen. Even with the low power the white cells can be recognized by their stained nuclei, and some idea of their relative numbers gained. For the minute examination of the white cells a high power, and preferably an immersion lens, is requisite. In many cases it is important to make a "**differential count**" in order to ascertain the relative numbers of the different varieties of leucocyte. For this purpose 200–500 cells must be counted, which, with a little practice, can be done in a quarter of an hour.

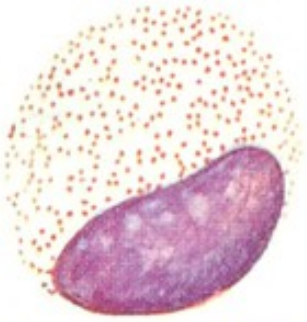
It is often necessary also to calculate the *absolute* number of each kind of white cell per c.mm. of blood, as otherwise a relative increase or diminution of one kind may be mistaken for an absolute increase or reduction. Throughout *adult* life the absolute number of polynuclears per c.mm. is about 4,000, whilst that of the lymphocytes is about 2,000.

The following are the varieties of leucocytes found

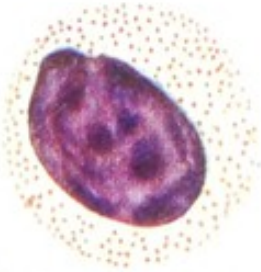
PLATE 5

BLOOD CELLS, NORMAL AND ABNORMAL.

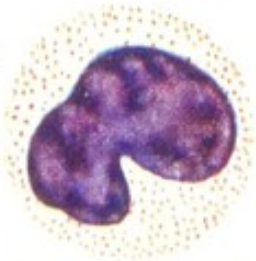
The left-hand column represents cells produced in the bone-marrow, and present in the blood in disease only. The central column represents normal and abnormal varieties of red cells. The right-hand column illustrates the normal leucocytes of the blood. (Leishman's stain.)



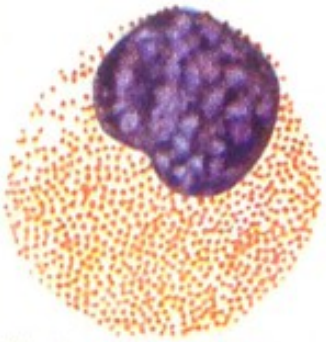
Neutrophil myelocyte
(large type).



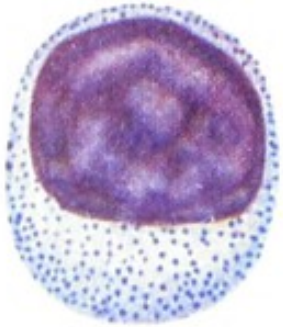
Neutrophil myelocyte
(small type).



Transitional neutrophil.



Eosinophil myelocyte.



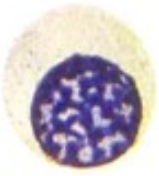
Basophil myelocyte.

Normal red cell.

Poikilocyte.

Polychromatophilia.

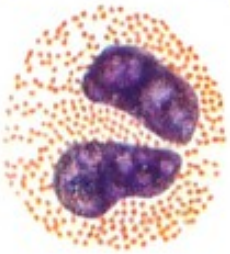
Normoblast.



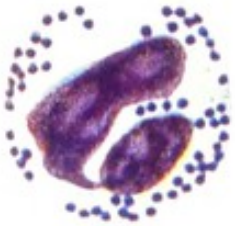
Megaloblast.

Granular
degeneration.

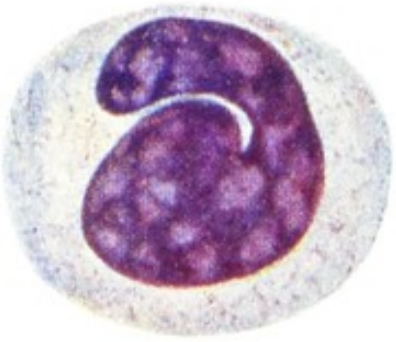
Polynuclear neutrophil.



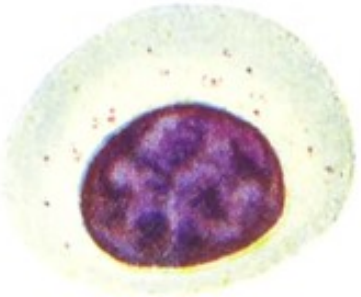
Eosinophil.



Mast cell.



Large hyaline.



Large lymphocyte.

Small lymphocyte.

PLATE 5.

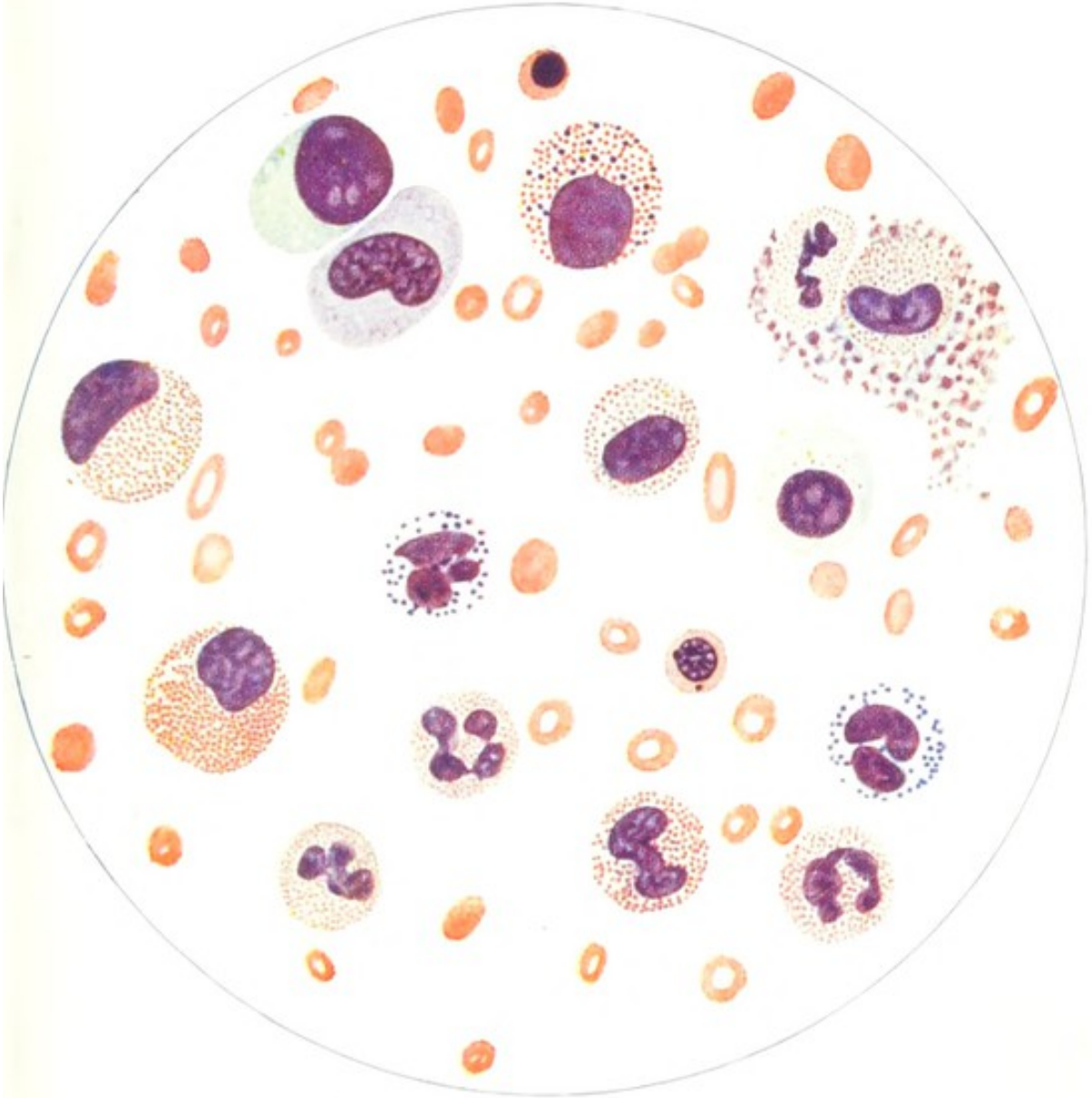


Plate 7.—THE BLOOD IN MYELOID LEUKÆMIA.
(Leishman's stain.)

in normal blood (Plate 5), with their relative proportions :—

1. *Finely granular oxyphils* (or polynuclear neutrophils). Cells with multipartite nucleus and fine neutrophil or faintly oxyphil granules : 60 to 65 per cent.

2. *Coarsely granular oxyphils* (or eosinophils). Cells with multipartite nucleus and coarse, strongly oxyphil granules : 2 to 3 per cent.

3. *Coarsely granular basophils* (or mast cells). Cells with very pale cytoplasm, a nucleus usually bilobed, and coarse basophil granules : 0·5 per cent.

4. *Large hyalines* (or large mononuclears). Cells with a characteristic notched or kidney-shaped nucleus and a slightly basophilic, faintly reticular cytoplasm : 3 to 5 per cent.

5. *Large lymphocytes* with round nucleus and clear basophilic cytoplasm : 5 to 10 per cent.

6. *Small lymphocytes* with round, deeply staining nucleus which almost fills the cell, leaving a rim of strongly basophilic cytoplasm : 20 to 25 per cent.

Some of the alterations which occur in the relative proportions of these in **leucocytosis** have already been mentioned (p. 220).

In the lymphatic form of **leukæmia** an enormous increase occurs in the number of the lymphocytes (Plate 6).

In the myeloid form of the disease the neutrophils, eosinophils, and mast cells are all increased, and in addition bone-marrow cells (" myelocytes ") appear in the blood. These are often of large size, with a single round nucleus, and contain granules which may be either neutrophilic or eosinophilic in reaction (Plate 7).

Myelocytes are also found in the blood in " splenic anæmia of infancy " and in some other conditions.

A relative diminution of the leucocytes is spoken of

as **leukopenia.** It is found in enteric fever, splenic anæmia, and some other diseases.

The **red cells** may present various alterations in disease (Plate 5). The alterations may affect—

(*a*) Their *size* and *shape.* Instead of the normal-sized erythrocytes, small cells may appear, devoid of the usual central indentation (microcytes), or unusually large forms may be met with (megalocytes), particularly in pernicious anæmia. Instead of being rounded the corpuscles may become oval, pear-shaped, etc. These changes are spoken of collectively as *poikilocytosis.*

(*b*) The *staining power* of the cells may be altered. Thus, instead of taking up eosin in the normal manner, they may stain with the basic dye, and have a violet or even bluish tinge. This is spoken of as *polychromatophilia.* It is seen in various kinds of anæmia, and is believed to indicate either a degeneration of the red cells or, more probably, an immature condition of them.

(*c*) *Nucleated forms* may appear. If these are of the same size as ordinary red corpuscles they are spoken of as *normoblasts.* They can be distinguished from lymphocytes (for which at the first glance they are apt to be mistaken) by (1) the more homogeneous and intense staining of the nucleus, (2) the presence round the nucleus of a cell body which stains red, (3) their smoother contour.

Megaloblasts are large nucleated red corpuscles. They may be even four times as large as an ordinary red cell. They have a relatively small and characteristically stippled nucleus, and a large cell-body, which always exhibits polychromatophilia. Megaloblasts are a characteristic feature of the blood in pernicious anæmia (Plate 8).

Microblasts—nucleated forms smaller than an

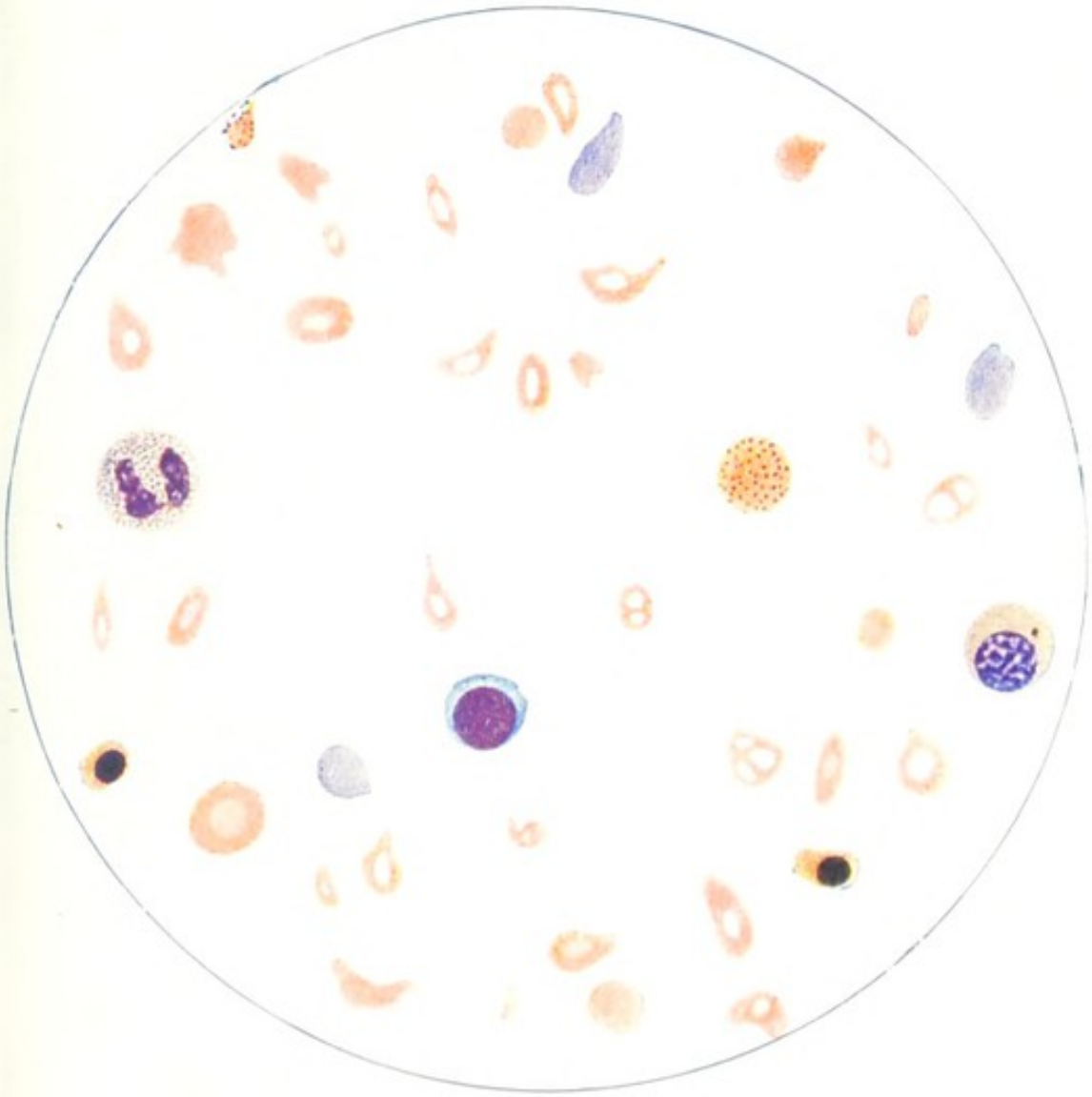


Plate 8.—THE BLOOD IN PERNICIOUS ANÆMIA.
(Leishman's stain.)

ordinary red corpuscle—are found in traumatic anæmias, and are of no great significance.

Parasites in the blood.—We have already described the way to look for **filariæ** in the blood (p. 229). Of this parasite there are several species which are the embryos of corresponding parental forms. The embryos live free in the blood (Fig. 67); the parental forms are found in the tissues and lymphatics. In one species the embryos are present in the blood during the night only (*F. nocturna*, Fig. 66), in another only by day (*F. diurna*). In yet another form (*F. perstans*) they are always present. The blood in suspected cases should therefore be examined both during the day and during the night. For the diagnosis of the different species special works must be consulted, but the chief points to attend to are (1) the time when the parasites are present in the blood; (2) the nature of their movements; (3) whether or not they possess a sheath; (4) the shape and character of their extremities.

To recognize the **parasite of malaria,** proceed as follows :—

Prepare a film of fresh blood in the manner already described (p. 227). Examine with a $\frac{1}{12}$ immersion lens and rather feeble illumination. Look in the red corpuscles for the presence of small black specks, often rod-like and showing slow movements of translation (Plate 9, Figs. 1, 2). These are surrounded by clear areas. One may also see in the centre of some of the red cells clear amœboid areas which show no pigment (Plate 9, Figs. 1, 2). Rosette forms may be visible (Plate 9, Fig. 2). These forms of the parasitesare always present in cases of malaria which have not had quinine. Other varieties are only met with in some chronic or malignant cases. Of these there are two chief forms—(1) the crescentic, which are not found except in cases of malignant infection, and (2) the

flagellated (Plate 9, Figs. 4, 5). These are easily recognized. The crescentic bodies are highly refractile, rather longer than a red blood-corpuscle, and about 2 μ in diameter. Particles of pigment may be recognized in the parasite and also in some of the ordinary leucocytes.

The examination of blood for the malarial parasite demands some care.* Manson says that one must devote half an hour to the examination of a slide before pronouncing on the absence of parasites in it. The quartan form of the parasite (Plate 9, Fig. 2) is distinguished from the tertian (1) by being smaller in size, (2) by its pigment granules being darker, (3) by its showing fewer segmenting forms.

In addition, stained preparations of the blood should invariably be made, and preferably with Leishman's stain in the manner described above (p. 234).

Trypanosomata may be looked for in fresh blood, or they may be fixed and stained in blood films. As the parasites are often few, it is important, in doubtful cases, to centrifuge the blood before examining it, when most of the trypanosomes will be found collected along with the leucocytes in a thin, pale-coloured layer. It is not easy in all cases to obtain a sufficient quantity of blood to fill an ordinary centrifuge tube; therefore one may employ a small tube with a diameter of some 5 to 8 mm., similar to Wright's pipette figured on p. 608, but with the mixing chamber considerably longer. This chamber is half filled with blood, and into the remaining space is drawn a solution made by dissolving 1 grm. of sodium citrate in 100 c.c. of normal saline solution. The bulb is thereafter sealed off in the flame of a spirit-lamp and placed in the hæmatokrit arm of the centrifuge. After revolving for ten or fifteen minutes a distinct white ring is formed. The bulb is then scratched with a file about $\frac{1}{2}$ cm. above the white ring, the clear fluid removed, and the white ring itself transferred, with the aid of a capillary pipette, to a cover-slip, and examined on an ordinary slide. The

* Parasites are most abundant in the blood about eight to twelve hours after the rigor.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4. Fig. 5.

Plate 9.—THE BLOOD IN MALARIA, SHOWING DIFFERENT FORMS AND STAGES OF DEVELOPMENT OF THE PARASITE.

(*After Thayer and Hewetson.*)

Fig. 1.—The parasite of tertian fever.
Fig. 2.—The parasite of quartan fever.
Fig. 3.—The parasite of æstivo-autumnal fever.
Fig. 4.—Crescentic and oval bodies.
Fig. 5.—Flagellated forms.

examination should be made with a $\frac{1}{6}$-in. objective and an eyepiece with a fairly wide field ; and before a negative conclusion is arrived at, not less than a quarter of an hour should be spent in searching the specimen. The parasite is more readily recognized by its movements than by its form. In doubtful cases it is important to centrifuge the blood imperfectly, so as to precipitate most of the cells, thereafter to remove the supernatant fluid with a pipette and to centrifuge this fluid a second time, when the deposit that subsides will contain nearly all the trypanosomes.

Fixed preparations can be made by staining blood films, prepared in the usual way with Leishman's stain (p. 234). For the study of finer structural details the following more elaborate method has been devised by Dutton and Todd :—

Fix the blood film by exposure, for five to fifteen seconds, to the vapour of a mixture of equal parts of glacial acetic acid and 2 per cent. solution of osmic acid. Thereafter stain with one or other modification of Romanowsky's stain (Appendix, 31) for three to six minutes, the slide to be stained being placed face downwards in the solution. When the staining is complete, wash with water, dry in the air, and mount. Preparations should be kept unmounted, as they are apt to fade quickly in balsam. In such preparations the trypanosome appears as a spindle-shaped body, along one border of which a narrow and delicate "undulating membrane" may be observed. Within the body two masses of chromatic substance are visible. Of these the larger, which is the nucleus, lies more anteriorly. The smaller, known as the micronucleus, or blepharoplast, or centrosome, is generally situated near the posterior or rounded end of the trypanosome, and in its neighbourhood a vacuole is often found. From the centrosome a fine filament emerges, and, passing along the free border of the undulating membrane, is continued anteriorly as a flagellum. On an average, the length of the human trypanosome may be reckoned at 17 to 25 μ (including the flagellum, which averages 6 or 7 μ) ; the breadth is from 1·5 to 2 μ.

Trypanosomes can also be found, in the later stages of the disease, in the cerebro-spinal fluid. The latter is easily obtained by lumbar puncture (p. 577), and is often more turbid than usual in consequence of the presence of an excess of leucocytes. The specimen is best centrifuged and examined fresh, as the organism does not stain so satisfactorily in cerebro-spinal fluid as it does in blood. In cases where there are enlarged glands these may be punctured, and a drop of fluid withdrawn and examined for trypanosomes, which may be fairly abundant.

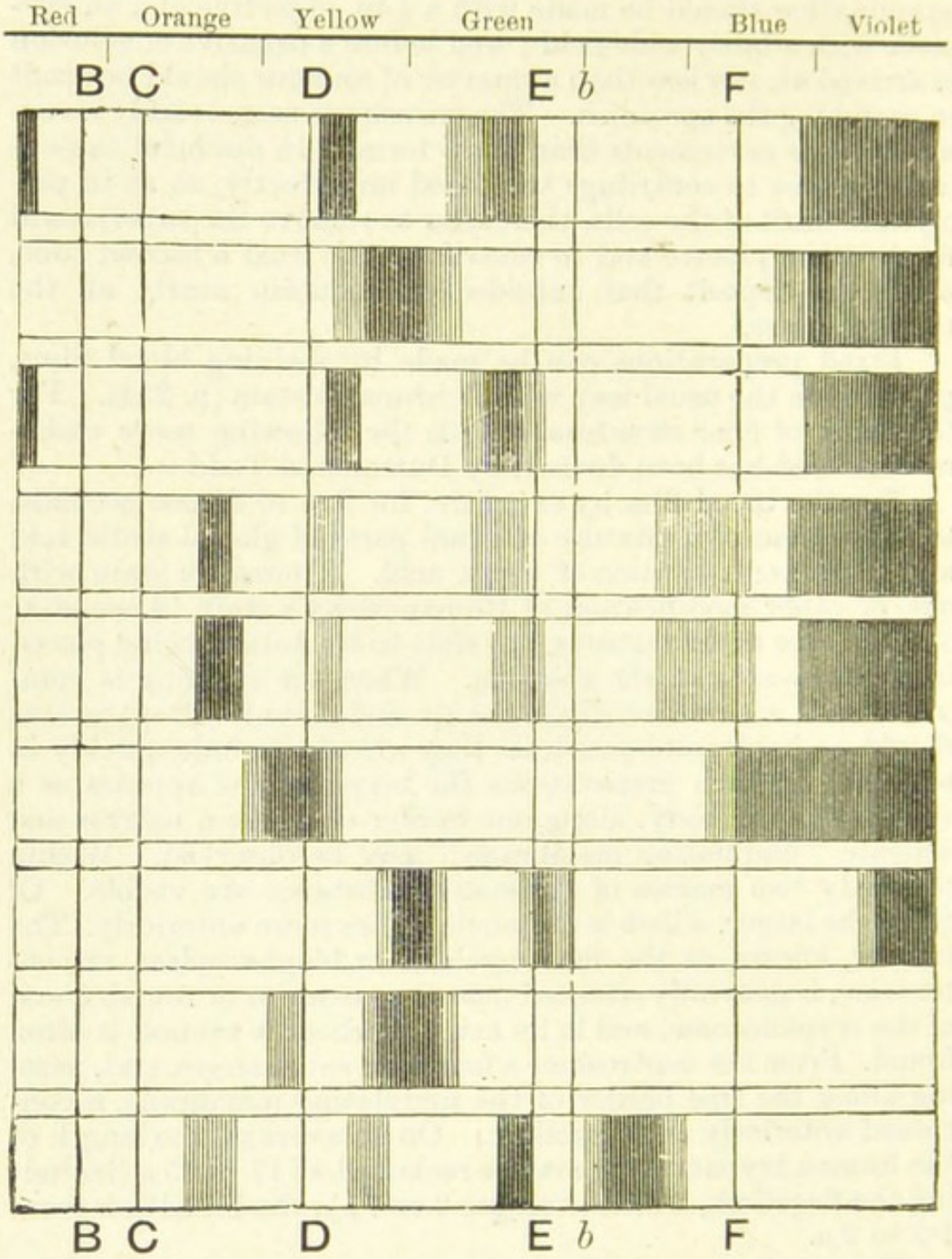


Fig. 70.—Spectra of hæmoglobin and its derivatives.

I., oxyhæmoglobin; II., reduced hæmoglobin; III., carbonic-oxide hæmoglobin; IV., methæmoglobin (in acid solution); V., acid-hæmatin; VI., alkali-hæmatin; VII., hæmochromogen; VIII., hæmatoporphyrin (in acid solution); IX., hæmatoporphyrin (in alkaline solution).

In many cases where no trypanosomes can be found in the blood they may be readily demonstrated in fluid from glands. In most cases of the disease the organisms are never numerous either in the blood or the spinal fluid, and a negative

diagnosis in such cases may only be justifiable after inoculation experiments have failed.

Leishman-Donovan bodies are small round or oval bodies with a diameter of about 3 μ, which are found in splenic blood in certain fevers. They develop, outside the body, into large flagellated forms. They can be stained by Leishman's or Romanowsky's methods, and are then found to possess two chromatic masses, a large and a small, the latter of which is often associated with a vacuole. The parasites appear to occupy the interior of endothelial cells or of leucocytes. They are excessively rare in the general blood-stream, and should be sought in blood aspirated, with rigid precautions, from the spleen. It is stated that the best results are obtained when the aspirated blood contains some traces of splenic pulp. Puncture of the spleen is occasionally attended with grave risks of hæmorrhage in cases of kala-azar; the procedure should therefore be undertaken only where it is obviously necessary and after repeated efforts to detect the parasite in the general circulation have proved unavailing. Sufficient material for examination can be obtained by means of a small syringe armed with a fine hypodermic needle, with the minimum of risk to the patient.

The examination of the blood for **bacteria** is considered in Chap. XIV.

Carbonic oxide in the blood. — In cases of suspected carbonic-oxide poisoning, the presence of the gas may be detected by spectroscopic examination. Some blood is obtained by pricking the thumb and squeezing two or three drops into several c.c. of distilled water. The solution has a cherry-red colour. Place some of it in a thin, flat glass tube, and examine with a hand spectroscope. Direct the instrument, as in all such examinations, towards a white cloud, and not towards the sun. Two bands (Fig. 70) are seen (bands of carbonic-oxide hæmoglobin) occupying very much the position of the oxyhæmoglobin bands. They are distinguished from the latter by the fact that the addition of a few drops of sulphide of ammonium produces no alteration in them.

Specific gravity of the blood. — For the

clinical estimation of the specific gravity of the blood various methods have been proposed. The simplest is the following :—

A mixture is made of chloroform and benzol in such proportions that the specific gravity of the fluid, as taken with a sensitive urinometer, is 1060. Some of this is placed in a tall glass vessel, and a drop of blood added to it from a hæmocytometer pipette. If the drop remains suspended without either rising to the surface or sinking to the bottom, the specific gravity of the blood is the same as that of the mixture. If the drop floats, add benzol out of a burette, stirring well with a glass rod, until the drop remains suspended. If it sinks, add chloroform till a similar result is attained. Then take the specific gravity of the mixture with a small hydrometer, and the result will give the specific gravity of the blood. Provided the same hydrometer be used in every case, the results are uniform and reliable.

The normal specific gravity of blood is about 1060. Variations are not of much clinical value. The specific gravity is always in proportion to the amount of hæmoglobin—a low specific gravity means little hæmoglobin. The ratio is so constant that one can tell the percentage of hæmoglobin by taking the specific gravity.

The **molecular concentration** of the blood, i.e. the proportion of molecules in solution in a given quantity of serum, can be determined by cryoscopy. The details of the method will be found in the chapter on the Urinary System (p. 315).

The clinical estimation of the **alkalinity of the blood** can only be accomplished by the use of rather elaborate methods, the results yielded by which are not of sufficient importance to justify their description in a work such as this.

The **coagulability of the blood** can be estimated with a fair degree of accuracy by means of Wright's coagulometer.* The instrument consists of a series of fine tubes of equal calibre which are kept immersed in water at body temperature. Blood is drawn into each of the tubes at definite intervals, and, after the lapse of varying periods of time, one blows down the tubes in succession. If the blood can no longer be blown out, coagulation has occurred. The interval between the filling of the tube and the occurrence of coagulation is known as the "*coagulation time.*" At a temperature of 37° C. the coagulation time of a healthy individual is about four minutes.

* The instrument is supplied, along with full directions for use, by Hawksley, 357, Oxford Street, London, W.

CHAPTER VI

THE RESPIRATORY SYSTEM

I. ANATOMY

(Plates 1, 2, 3, 4)

THE following anatomical facts must be borne in mind when the lungs are examined :—

1. **Borders of the lungs.** **Right lung.**—The anterior border passes forwards, downwards, and towards the middle line from the apex, which, situated at the level of the neck of the 1st rib, corresponds posteriorly with the 7th cervical spine. Behind the sternum, at the level of the 2nd rib, it has nearly reached the middle line, and passes directly downwards to the level of the junction of the 6th costal cartilage with the sternum, where it turns rather abruptly to the right to pass outwards as the lower border. The lower border meets the right parasternal line at the level of the upper border of the 6th rib, the mammary line also at the level of the 6th rib, the axillary lines at the 7th and 8th ribs, the scapular line at the 10th rib, and at the side of the vertebral column reaches as far as the 10th interspace or 11th rib.

Left lung.—From the apex to the level of the 4th costal cartilage the anterior border passes in a direction which corresponds with that of the right lung. At this point it bends rather suddenly outwards, thereby leaving part of the anterior surface of the heart exposed, and passes in an arched line outwards and downwards to reach the 6th rib a little externally to the parasternal line. From this

point the lower border passes backwards along a line corresponding to, but a little lower than, that of the lower border of the right lung. The lower borders of both lungs are convex towards the abdomen. In forced respiration they may vary in level to the extent of 2 or even 3 in., according to the phase of the respiratory cycle. In quiet respiration the difference between the extremes is only about 1 cm.

2. **Lobes of the lungs.**—It is often important to know the limits of the individual lobes of the lungs. This may be done by drawing a line from the 2nd dorsal spine to the 6th rib in the mammary line; this corresponds to the upper border of the lower lobe. A second line, drawn forwards on the right side from the centre of this line to meet the sternum at the level of the 4th costal cartilage, will mark the boundary between the upper and middle lobes.

Obviously, therefore, the greater part of each lung, as seen from behind, is composed of the lower lobe, only the apex belonging to the upper lobe; while the middle and upper lobes on the right side, and the upper lobe on the left, occupy most of the area in front. In the axillary regions, parts of all the lobes are accessible.

The **bifurcation of the trachea** corresponds in front with the lower part of the manubrium sterni; behind, with the disc between the 4th and 5th dorsal vertebræ.

The **reflected pleural sacs** reach decidedly lower than the inferior borders of the lungs, whose limits they overstep for about 2 in. in the mammary, nearly 4 in. in the midaxillary, and $1\frac{1}{2}$ in. in the scapular lines. The sinus thus formed lies on the left side above the resonant stomach cavity, and therefore, should it become distended with fluid, as

in cases of hydrothorax, a dull area will be discovered at a part where the healthy percussion note is tympanitic. The anterior reflection of the left pleura below the 4th rib is considerably nearer the middle line than the anterior border of the left lung; hence in emphysema, when the lung presses forward into this available space, the area of absolute cardiac dullness is greatly encroached upon.

With reference to the correspondence of points in front and at the back, Quain gives the following relations as existing during expiration :—

"The upper margin of the sternum is on a level with the disc between the 2nd and 3rd dorsal vertebræ; the junction of the manubrium and body is opposite the 5th dorsal vertebra; and the xiphi-sternal articulation generally corresponds to the lower part of the 9th dorsal vertebra."

The **scapula** is a useful landmark posteriorly. Its upper angle, when the arms hang by the side, is generally on a level with the disc between the 1st and 2nd dorsal vertebræ, the root of the spine with that between the 3rd and 4th dorsal vertebræ, and its lower angle with the body of the 8th dorsal vertebra.

In reference to the ribs, the upper angle of the scapula just covers the 2nd rib; the lower angle reaches as low as the 7th interspace or 8th rib.

The 12th rib cannot always be felt. It is not safe, therefore, to count the ribs from below upwards.

For convenience in description, the thorax is mapped out into regions, as follows :—

1. **Three central regions anteriorly.**

Suprasternal, from the cricoid to the upper border of the manubrium.

Superior sternal, from the upper border of the manubrium to the level of the 3rd chondrosternal articulation.

Inferior sternal, from the 3rd chondrosternal articulation to the lower end of the sternum.

These three regions are bounded laterally by the lateral sternal lines and their upward continuations.*

2. **Five antero-lateral regions** on each side.

Supraclavicular, bounded above by an oblique line from the side of the cricoid to the outer end of the clavicle, below by the clavicle.

Clavicular, composed of the area occupied by the clavicle.

Infraclavicular, bounded above by the clavicle, below by a horizontal line at the level of the 3rd chondrosternal articulation.

Mammary, from the lower edge of the infraclavicular area to the level of the 6th chondrosternal junction.

Inframammary, below that level.

These regions extend outwards to the anterior axillary line.

3. **Two lateral areas on either side.**

Axillary, } meeting each other at the
Infra-axillary, } level of the 6th rib.

4. **Four regions at the back on either side of the spine.**

Suprascapular.

Scapular, subdivided into supra- and infra-spinous.

Infrascapular, and

Interscapular.—The position of the dorsal regions is sufficiently defined by their names.

* Sometimes the sternal regions are classified as "episternal" and "xiphisternal."

II. INSPECTION

Inspection determines—

A. Form of chest.

1. Healthy.
2. Symmetrical chests with features indicating proclivity to disease.
   - The alar chest.
   - The flat chest.
3. Symmetrical chests with features indicating past disease.
   - The rachitic chest.
   - The pigeon breast.
   - Harrison's sulcus.
4. Symmetrical chests with features indicating present disease.
   - The barrel-shaped chest.
   - Bilateral retraction.
5. Unilateral changes.
   - Enlargement.
   - Diminution.
6. Local changes.
   - Bulging.
   - Retraction.
   - Funnel-shaped depression.

B. Movements of chest.

1. Respiratory.
   - (1) Rate.
   - (2) Rhythm.
   - (3) Type.
   - Character (*see also* Chap. II.).
     - Amount of expansion.
     - Unilateral fixation.
     - Local lagging.
     - Local indrawing and bulging.
2. Non-respiratory. Pulsations (Chap. IV.).

A. Form of the Chest

This depends partly on the curvature and obliquity of the ribs, partly on the curves of the spinal column. The curvature of the sternum results from the relations of these factors.

When the ribs are normally curved, the more horizontally they lie the more nearly does a cross section of the chest approach the form of a circle, the wider are the intercostal spaces, and the more obtuse does the subcostal angle become; whilst, on the contrary, increasing obliquity of the ribs leads to

narrowing of the intercostal spaces, to increasing ellipticity of the cross section of the chest, the major axis lying transversely and the minor axis in an antero-posterior direction, and at the same time the subcostal angle becomes more acute. In a healthy male the angle is about 70°, in a female about 75°. The variations may amount to 10° above or below these averages. When there is lateral curvature of the spine, the chest is rendered asymmetrical; when the spine is unduly concave forward, other changes are produced, which will be dealt with subsequently.

1. The **ideal healthy chest** will conform to the following description: It is bilaterally symmetrical, its contours are smooth, it has no deep hollows, and at most shows only a slight recession below the clavicles. In cross section it is an ellipse, broader from side to side than from front to back in the proportion of about 7 to 5; its general shape is ellipsoidal, with the longest axis vertical. In children the cross section is more nearly circular.

The sternum, which is convex from above downwards when viewed from the front, lies at the bottom of a shallow groove known as the sternal furrow, formed by the pectoral muscles of each side. The junction of the manubrium with the body of the sternum exhibits a slight angular projection (angulus Ludovici), sometimes visible, almost always palpable. The sternal furrow ends below, at the level of the 7th costal cartilage, in the infrasternal depression (or scrobiculus cordis). A slight hollow below the clavicle marks the separation between the divisions of the pectoralis major; it should not be deep, and ought only to be distinct when the muscle is made to contract. A second hollow, which is much more distinct, separates the pectoralis from the deltoid. This fossa lies farther from the middle line, and is

known as the infraclavicular (or Morenheim's) fossa. It becomes very marked in many cases of phthisis.

The shape in the mammary regions depends greatly on the degree of development of the mammary gland and on the amount of subcutaneous fat. In the adult male the nipple is usually about 4 in. from the middle line, in the 4th intercostal space.

In actual practice it is very rare to find a chest which is perfectly symmetrical. Generally the right side is rather more capacious than the left, the right clavicle is tilted more than the left, and the spinal column almost always has a slight degree of lateral curvature. In inspection of the chest the examiner should first look at it from the front, then from the side, thereafter from the back, and, finally, he should look over the shoulders from behind and above, so as to see the profile of a horizontal section of the thorax. The last method is very useful in detecting lack of symmetry or unequal expansion on the two sides. The neck, especially as regards the manner in which it is set on the chest, and the epigastrium should be inspected at the same time as the thorax. In examining the chest from behind, it is important to note whether the vertebral borders of the scapulæ are unduly prominent, whether they are equidistant from the middle line, and whether their lower angles lie at the same level on either side.

Deviation from the normal form may affect either the whole of the thorax or localized parts of it. The **abnormal shape** of the chest as a whole may be grouped in three classes, according as it indicates merely a proclivity to lung disease, a history of former disease, or the existence of present disease. The first class contains the alar and flat chests; the second the rickety chest, the pigeon breast, and the chest with Harrison's sulcus; the third the barrel-

shaped chest and the hollow or retracted chest. In these groups the changes affect both sides of the thorax, and so the symmetry remains undisturbed. In other instances the morbid conditions at work may lead to unilateral changes in the shape of the chest, one side having its volume either increased or diminished, and being otherwise deformed. Lastly, the chest may exhibit local deviations from the normal form, due generally to local disease.

2. **Symmetrical chests with features indicating proclivity to lung diseases ("phthinoid" chests).**—The two forms which belong to this class are the alar and the flat chest.

i. The **alar chest** is one where the vertebral borders of the scapulæ project unduly, and the shoulders droop. The cause of this appearance is to be found in the obliquity of the ribs, which makes the projection of their curves and angles in the horizontal plane more sharp, leads to a long and rather shallow thorax, and is associated with a long neck and prominent throat.

ii. The **flat chest** is due to a loss of the forward convexity of the costal cartilages, which become more or less straight. As a result, the sternum is less distant from the vertebral column than usual. The flat chest is often, but not always, associated with the alar form.

3. **Symmetrical chests with features indicating past diseases** (and not seldom predisposing to pulmonary disease).

This group contains a number of forms, but only a few need be considered here.

i. **The rachitic chest.**—In rachitis the bones are less rigid than usual, and so are more readily deformed by any applied force. From the nature of the disease the part that yields most readily is that where the bone

and cartilage meet, and therefore, when any cause prevents the free access of air to the lungs during inspiration, this part bends inwards before the pressure of the external air. A vertical groove is thus formed in this region, and persists even after the cause which first led to its production has disappeared. The section of a rachitic chest is shown in the accompanying figure, where the depressions situated at a little distance from either side of the sternum are easily recognized. When the rachitic condition is severe the line of least resistance becomes so weak that no unusual obstruction to inspiration is necessary in order to produce the grooves; the slightly lower air-pressure within the thorax, which is necessarily present during inspiration, being sufficient to lead to its formation (Fig. 71).

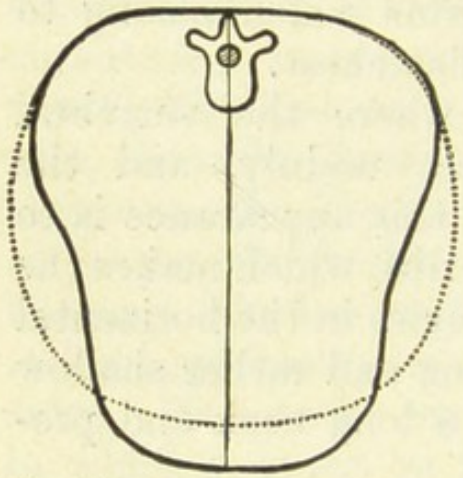


Fig. 71.—Cross section of rachitic chest. (*Gee.*) The dotted line represents the normal outline for the same age.

ii. **The pigeon breast.**—Here, in consequence of some obstruction (often quite trivial) to inspiration at a time of life when the ribs are soft, they become straightened in front of their angles, where, owing to their smaller degree of curvature, they are most readily deformed by external pressure. The result is that the sternum becomes unduly prominent and projects beyond the plane of the front of the abdomen, so that there is a sharp angle at its lower end. At the same time the cross section of the chest ceases to be elliptical, and approaches a triangular form, the angles being situated at the sternum in front, and at the costal angles behind (Fig. 72).

iii. **Harrison's sulcus.**—This is a transverse con-

striction which, beginning at the level of the xiphisternum, passes outwards and slightly downwards. It seldom reaches as far as the midaxillary line. This deformity is due to the same cause as the last, but either the obstruction has been slighter, or the bones have been more fully hardened.

The depression is therefore limited to the most yielding part of the chest, and this corresponds to the region where the cavity is widest. Lower down than the sulcus, the liver and other abdominal viscera had supported the chest wall and so prevented it from being drawn inwards; whilst, higher up, the greater curvature of the ribs had enabled them to withstand the external pressure.

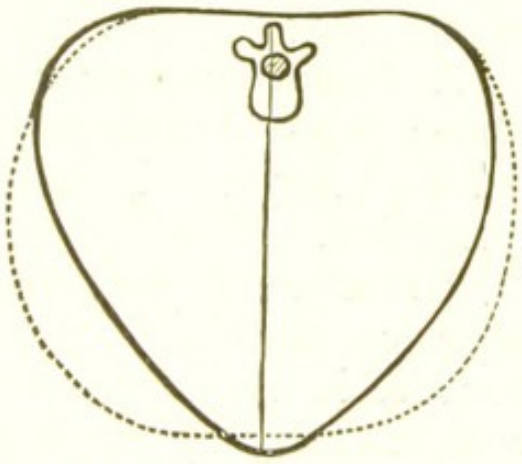


Fig. 72.—Cross section of pigeon breast. (*Gee.*) The dotted line represents the normal outline.

The three deformities just described are frequently found together in one individual, and cases of pigeon breast almost invariably exhibit a well-marked Harrison's sulcus.

4. **Symmetrical chests with features indicating present disease.**

i. In **emphysema** the increased volume of the lungs demands increased space for their accommodation. To provide this, the ribs are less obliquely set than usual, while the spine becomes unduly concave forwards, and the sternum is much more arched than under normal conditions, while the angle of Louis becomes extremely prominent.

Since this increase of volume is accomplished by the mechanism which is provided for the expansion of

the chest in inspiration, the latter can only be effected by movements of the chest as a whole, and so the accessory muscles of respiration have to take the place of the intercostals, thereby becoming abnormally conspicuous, whilst the diaphragm has a considerable excess of work imposed upon it. The chest of emphysema is described as "*barrel-shaped.*"

Kyphosis may produce a form of chest which closely simulates that of emphysema.

ii. **Bilateral hollowing** is an extreme case of the flat chest already described, and is caused by the existence of phthisis.

5. **Unilateral changes in the shape of the chest which affect the whole of one side.**—These may result either in unusual bulging or in retraction.

Unilateral enlargement is due either to the presence of fluid* or of gas in the pleura, or to increase in volume of one lung due to a tumour, or compensatory hypertrophy.

Diminution of volume results from shrinkage of the lung. This may be caused by phthisis, or it may be the result of adhesions formed during an attack of pleurisy with effusion. Collapse of a lung from obstruction of the bronchi may produce a similar result.

Before connecting these changes with disease of the lungs or pleura, the observer must ascertain that no *scoliosis* exists; for the rotation of the vertebræ in this condition leads to a deformity which, when

* The presence of fluid does not always cause enlargement of the affected side of the chest, although it often does so. Sometimes, indeed, that side is smaller. The explanation is not very obvious, but it must be recollected that the fluid is primarily accommodated by displacement of the lung, and only later, after the elasticity of the lung has been exhausted, will the contour of the thorax be altered.

inspected only from the front, it is almost impossible to discriminate from those just described.* Abdominal disease must also be excluded.

6. **Local changes** affecting only part of either side.

i. **Bulging.**—In emphysema the apices may produce an unusual fullness above the clavicles, and in pleural effusion, especially when of a purulent nature, the interspaces which lie in the area of effusion often bulge considerably; these phenomena may accompany a general enlargement, or occur without it.

Tumours of various kinds, and disease of the heart, may be the cause of localized bulging.

ii. **Shrinking.**—In phthisis one or both apices are often contracted, and thereby a hollowing is produced above the clavicles. A similar condition occurs also in the infraclavicular regions, which may exhibit marked hollowing. This is particularly noticeable in Morenheim's fossa.

To detect either bulging or flattening (as well as diminished expansion) it is important to look tangentially along the chest either from above the shoulders or upwards from below. Both in phthisis and in other wasting diseases the interspaces are very sunken, and the ribs prominent, in consequence of the malnutrition of the muscles and subcutaneous tissues.

Not infrequently a local shrinking is due to adhesions of the pleura resulting from a former attack of pleurisy.

iii. A **funnel-shaped depression** is sometimes found in the lower part of the middle line of the thorax in front. Sometimes it is congenital, or it may be developed in infancy with or without any obstruction

* Scoliosis may, however, be induced by the retraction of a lung in a young subject.

to respiration being present. It extends in some cases as high as to the 3rd rib. A similar depression—though seldom of such magnitude—is found as a trade deformity in shoemakers.

B. Movements of the Chest

The movements of the chest during respiration also demand attention, and the rate of movement, its rhythm, its type, and its amount, must be noted.

1. The **rate** for an adult in health is about 18 or 20 respirations per minute, but there is a wide margin on either side of these figures. Increased rapidity may result from exertion, nervous excitement, fever, or defective aeration of the blood, whether this be due primarily to cardiac, pulmonary, bronchial, or laryngeal causes, or to some alteration in its oxygen-carrying power. It may also arise from the association of pain with all attempts at respiration, as in pleurisy and peritonitis, when the breathing becomes shallow, and must therefore be more frequent to make up for the slighter expansion.

The **ratio between respiration and the pulse** is important. In health it is about 1 to 4 ; in pneumonia respiration may occur almost as frequently as the pulse ; in certain cases of narcotic poisoning the ratio may become 1 to 6 or 7.

2. The **rhythm** varies very considerably even in health, and if the act be performed consciously it may become very irregular. Hence it is important to study it when the patient is off his guard, as only then can accurate observations be made. Either inspiration or expiration may be unduly prolonged ; the former being commonly associated with laryngeal or tracheal, the latter with bronchial or pulmonary diseases. A peculiar type, where successive respirations gradually get deeper and deeper till a maximum is attained, and

then fall off again until a pause of complete apnœa occurs, to be followed by another wave of gradually deepening and then diminishing respiration, is known as **Cheyne-Stokes breathing.** The pause may last for fully half a minute, though it is often shorter, and the whole cycle is usually completed in less than two minutes. It is very conspicuous when the patient who exhibits it is asleep, or is unconscious ; but is apt to be overlooked if the patient is awake, and particularly if he is talking. Apart from completely typical Cheyne-Stokes respiration, various modifications, more or less nearly approaching it, occur.

There is another form of respiration which is often mistaken for Cheyne-Stokes breathing, but which is really different. In this form, instead of a gradual increase in the depth of respiration from the apnœic pause to the middle of the cycle, the deep breathing begins suddenly, and gradually diminishes, until the apnœic pause is reached, thereafter to recommence once more with full vigour. It is often observed in cases of meningitis.

3. **Type.**—Breathing may be more evidently performed by the upper part of the thorax ; this is known as the **thoracic type of respiration.** It is found to a certain degree in women, but in its full development is either associated with paralysis of the diaphragm, or else is a result of its fixation from inflammatory causes or from increased abdominal pressure.

In men and young children the **diaphragm** and **abdominal muscles** play the most important part in respiration ; and in cases where the intercostal muscles are paralysed, or where some inflammatory and painful condition, such as pleurisy or pleurodynia, exists in the thorax, the breathing may be wholly abdominal in type.

In health the male type of respiration may be described as **abdomino-thoracic,** and the female as **thoracico-abdominal,** or almost purely thoracic.

The presence of **pain** or **dyspnœa** should always be inquired for, and its exact nature noted. (*See* Chap. II., p. 37.)

4. Regarding "**movement**" during respiration, the points to be noted are its amount, whether it is expansive in character, and whether it is similar, or different, on the two sides and over corresponding areas.

Amount of movement and expansion are by no means interchangeable terms; in emphysema the chest may move considerably, but there is little expansion.

In comparing the two sides it will often be found that deficient or absent movement betokens pleurisy with effusion, or non-expansion of the lung from consolidation or rigidity of its structure.

Local deficency in expansion is frequently a very important indication of phthisis, or it may be due to lobar pneumonia, the former especially at the apices, the latter at the apex or base according to the situation of the disease.

Sometimes one part of the chest wall lags behind the rest during inspiration. Any such lagging is important as suggestive of disease. The existence of any **indrawing** of the chest wall or of the interspaces during inspiration, or of any **bulging** during expiration, must be noted. Both may occur physiologically, in which case the conditions are present over the whole chest, and are not very conspicuous, or they may result from pathological conditions, when they sometimes affect the whole thorax, at other times one side, and yet at others only appear locally. Examples of inspiratory indrawing are found in obstruction of the

larynx (general), or in blocking of some of the smaller bronchi (local). One of the best instances of localized expiratory bulging is seen at the apices of the lungs in advanced emphysema.

Under the name of the **diaphragm sign**, Litten has described the occurrence of an indrawing of the lower intercostal spaces during normal inspiration. The phenomenon has long been observed, but Litten has added considerably to our knowledge of its significance. The lower intercostal spaces, passing obliquely downwards and forwards, are crossed by the more horizontally disposed inferior borders of the lungs. When a breath is drawn a depression can be observed in each interspace just below the point to which the border of the lung has reached, and it is obviously caused by the separation of the contracting diaphragm from the inner surface of the chest wall. As the inspiratory act is continued, this depression travels downwards, immediately preceding the advance of the expanding lung.

In order that the phenomenon may be seen to the best advantage the patient should lie on his back, the head but slightly elevated. The room should be lighted from a single window, towards which the patient's feet are turned, and the observer should place himself with his back to the window at a distance of three or four paces from the patient, and at an angle of 45° to the line of the entering light. On instructing the patient to breathe rather deeply a moving shadow will be seen to mark the upward and downward passage of the depression. The shadow is only visible in the interspaces, as the ribs are too unyielding, and its range will be found to extend, in a vertical line, over three or four interspaces during deep respiration, and over one or two when the patient takes ordinary breaths. In health the shadow can generally be seen with equal distinctness on both sides. It is readily visible on the anterior and lateral aspects of the chest, whilst in thin people it can be seen on the back also when they assume the knee-elbow position.

Absence of the shadow on one side usually indicates some morbid condition of the lower part of the corresponding lung or pleural cavity, or paralysis of the phrenic nerve of that side. Irregular movements of the shadow are suggestive of local adhesions. Alterations in the level of the shadow indicate an abnormal position of the diaphragm resulting from changes in the thoracic or abdominal viscera.

III. PALPATION

Palpation determines—

A. **Form of chest** (confirms or modifies the results of inspection, q.v.).

B. **Movements of chest.**
   1. Respiratory (*see also* Inspection).
   2. Pulsations (Chap. IV.).

C. **Vibrations**
   - Palpable pleuritic friction.
   - Palpable râles.
   - Vocal fremitus
     - Increased.
     - Diminished.
     - Absent.

D. **Tenderness.**

E. **Fluctuation.**

F. **Resistance of chest walls to compression.**

Palpation takes note, first, of the form and movements of the thorax ; second, of vibrations or tremors which are communicated to the hand ; and third, of the behaviour under pressure of any pain of which the patient complains. Under the first head inspection is supplemented; under the second, one learns something of the accompaniments—e.g. friction, or rhonchi—which interrupt the smoothness of the respiratory movements, and also of vocal fremitus, which serves to indicate the condition of the conducting media. The third enables one to detect the cause of many thoracic pains.

Before making a systematic examination, it is well to lay the hand on any part of the chest which presents an obvious swelling, or where the patient complains of pain. In doing so the observer should remember to look at the patient's face rather than at the part under examination, as he thus most quickly learns whether he is causing any avoidable suffering. Pain may be due to inflammatory conditions in the chest wall ; to intercostal neuralgia, where, as a rule, specially painful spots can be discovered corresponding to the points

where the branches of the affected nerves escape through the fascia ; to intercostal myalgia, where the pain is aggravated by pinching the affected muscles ; or to pleurisy. In the case of pleurisy, pressure may considerably increase the pain by bringing the opposed surfaces of the inflamed pleura more firmly into contact. At the same time the nature of any swelling should be investigated. The hand will also supplement the information derived from inspection with regard to prominence of the intercostal spaces, and may occasionally detect fluctuation in them when there is pleuritic effusion. Fluctuation also occurs, and is much more distinct, when an abscess has formed in the chest wall. Such an abscess may be due to disease of the bones or soft parts forming the parietes of the thorax, or to pus which has broken through from the pleural cavity (*empyema necessitatis*). In the latter case the pus may often be driven back by gentle pressure, to reappear when the patient coughs.

When these preliminary observations have been completed, the observer should direct his attention to the **form of the thorax.** Here the hand is best aided by mechanical appliances, such as the cyrtometer, and by simple measurements. Tracings and measurements should be taken at the periods of full expiration and inspiration. In a well-formed adult male the girth of the chest at the level of the nipples should be 34 in. at the end of expiration, and should measure at least 2 in. more when a deep inspiration has been taken. Height, age, and build of course greatly modify these measurements, and insurance returns indicate that different races vary very considerably in chest girth. It is generally far more important to ascertain the increase of girth between expiration and inspiration, both full and ordinary, than to determine the exact circumference of the chest at either phase.

If the shape of a cross section of the chest is required, a tolerably efficient cyrtometer can be improvised by connecting two pieces of flattened composition gas-pipe, each about 2 ft. long, by a hinge of elastic tube. The hinge should be placed over the spine, and the metal pipe moulded to the surface of the chest. It can then be opened at the hinge and closed again over a piece of paper, to which the outline should be transferred if a permanent record is desired. To prevent any risk of subsequent confusion, the back and right side of the tracing should at once be marked as such, and a line should be drawn from the position of the hinge to the point in front which corresponds with the middle of the sternum. The length of this line may be checked by a pair of callipers, as a precaution against accidental bending of the cyrtometer during its transference from the patient to the sheet of paper. For convenience in preservation, a reduced copy of the tracing may be made by means of a pantograph; if this is done a note of the exact proportion between the original and the copy should be recorded on the latter.* There is no difficulty in applying the same simple instrument so as to obtain the outline of the chest in other planes than the horizontal. Thus, by placing the hinge above the shoulder, the two pieces of pipe may be carried down the parasternal line in front and in a corresponding line behind, whilst by sharply bending their lower ends outwards they may be made to cross each other in the axillary line and the point of intersection marked. The instrument is then opened at the hinge and readjusted over the paper so as to yield the desired tracing.

The **nature of the respiratory movements** must next be studied. It is important to make certain

* Various more elaborate instruments have been devised, but they are cumbrous, and consequently not of great clinical use. The best of them is Dr. Graham Brown's perigraph.

that the two sides of the chest move to approximately the same extent. This is done by fixing the finger-tips of either hand at the patient's sides, and making the radial borders of the thumbs meet in the middle line in front of the chest. The hands being kept rigid, the patient is directed to take a full inspiration, when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest.

Sometimes one half of the thorax lags behind the other; this is readily detected by the hands no longer moving synchronously.

The movements at the apices may be similarly observed. In this case the physician stands behind the patient, and, fixing his thumbs on the vertebræ, lets his fingers lie over the right and left lung apices reaching towards the clavicles whilst the patient breathes deeply. Thereafter one hand should be placed on the front of the chest, and the other on the epigastrium. In health, as the chest expands, the epigastrium is also raised to a greater or less degree. If the epigastrium fall in with each expansion of the chest, there is reason to suspect paralysis or flaccidity of the diaphragm. Fixation of the diaphragm with immobility of the epigastrium during respiration is generally due to abdominal disease (*see* p. 57).

Vibrations may be detected by palpation. *For this purpose the palm of the hand should be applied flat on the chest*, and, since the sensitiveness of the two hands is often unequal, *the same one should be employed on both sides*. In addition to the vibrations already referred to in Chap. IV. (p. 124), fremitus may be due to pleural friction, to catarrhal changes in the mucosa of the bronchi, leading to local constrictions, or to fluid in the bronchi or in pulmonary cavities. After the presence or absence of these forms of fremitus

has been determined, the observer should study the **vocal fremitus,** or vibrations which the voice communicates to the chest wall. These are conducted from the larynx by the trachea and bronchi to the smaller tubes within the lungs, and thence through the lung tissue to the surface. Anything which affects the conducting power of the air-passages or lung tissue, or the interposition of additional materials through which the vibration must pass to reach the palpating hand, will obviously affect the intensity of the fremitus. To test the vocal fremitus, the patient is told to repeat "one, one, one," or "ninety-nine," in a clear voice. The hand placed on the thorax detects distinct vibration whilst this is done, and it must be determined whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however, forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished—and also whether they correspond to what former experience has led the observer to recognize as normal for the region under examination, for a similar chest, and like pitch and loudness of voice. **Vocal fremitus is increased** when the voice is of a deep pitch, when the chest wall is rigid, and often when it is thin, as also when the lung is consolidated, or contains a cavity near its surface. Since the right bronchus is wider and shorter than the left, whilst the septum separating the two bronchi occupies a position to the left of the centre of the trachea, the laryngeal sounds pass more freely along the right than they do along the left bronchus, and therefore the vocal fremitus is normally somewhat greater over the right lung than over the left. **Vocal fremitus is diminished** when the pitch of the voice is high, when the chest wall is thick, and especially when there is much thickening of the pleura. It is greatly diminished, or totally

absent, when the lung is separated from the chest wall by pleuritic effusion. The cause in this case is not that fluid is a bad conductor of sound or of vibration —the reverse is the case—but that the relaxed lung itself fails to convey the vocal fremitus, and so the vibrations never reach the fluid. In young persons and in female subjects the vocal resonance is different both in character and intensity from that which occurs in male adults. The differences are due to the different conformation and degree of rigidity of the thorax, and to the distinctive pitch and quality of the voice in each instance. The resistance of the chest to compression is best estimated by placing the hand over the sternum whilst the patient is lying down, and attempting to press it backwards towards the vertebral column. The rigidity increases with advancing age, and also in certain diseases (e.g. in phthisis and in emphysema). Where this is so the prognosis is less favourable, as free expansion of the lung is hindered.

IV. PERCUSSION

Percussion determines—

A. **The boundaries of the lungs** (topographical percussion).

B. **The resonance of the lungs.**

(*a*) Normal variations in different parts.

(*b*) Abnormal alterations.

1. Quantitative { *Increase* (hyper-resonance). *Diminution*, in varying degrees, from slight impairment to absolute dullness.

2. Qualitative: Tympanitic { High-pitched. Medium-pitched. Low-pitched.
Skodaic.
Boxy.
Cracked-pot.
Bell sound (coin percussion).
Amphoric.

But little need be added here to what has been already stated in Chap. IV. (p. 128) regarding the theory of percussion. It must be recollected that it is a most difficult task to give even a partial explanation of the phenomena observed, from the standpoint of physics, and in practice it is rarely necessary to appeal to theory, as a long experience has enabled physicians to attach certain meanings, more or less empirically, to various percussion phenomena.

It may, however, help the student to appreciate the various sounds when he hears them, if a few of the main factors in their causation are recapitulated.

First, we have to consider the materials which produce the sound. These are the pleximeter, the chest wall beneath it, and the subjacent viscus so far as it comes within the range of action of the percussion stroke. The pleximeter sound, by the choice of a suitable material, may either be rendered insignificant, or, in consequence of its special qualities, immaterial in its effect on the resonance. The chest wall yields a sound varying with the part struck, and depending for its quality on whether sternum, clavicles, ribs, or soft parts underlie the pleximeter. The sound due to the wall is, however, subordinate to that of the organ beneath when this contains air and the percussion stroke is firm enough.

The **character of the sound produced** varies quantitatively and qualitatively, the quantitative variations depending on the force of the blow delivered, and on the capacity of the part struck to resound to the blow. The **quality of the sound** depends on the particular vibrations which are elicited, and on the selective reinforcement of some of them by the resonance of the organs involved.

When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not

modified by excessive tension of the containing walls of the space, the sound heard has a tympanitic character; but when the cavity is subdivided into a number of small loculi by numerous septa, more or less tense, a characteristic resonance, no longer tympanitic, is produced. Such conditions prevail in the healthy lung, and the observer must learn by assiduous practice to recognize its distinctive quality. In general terms, this pulmonary resonance may be said to be low in pitch and clear in character.

In percussion over the lung we endeavour to ascertain three sets of facts: first, the position of the apices and lower border of the lungs, and also of that portion of the anterior border of the left lung which lies over the heart; second, the state of the lungs in regard to the quantity of air contained in their various parts, and the tension of their elastic framework; and, third, whether they are unusually remote from the surface of the chest, the separation being due to thickened parietes, or fluid or gas in the pleural cavity.

The apices and borders.—Resonance can usually be observed in health for $1\frac{1}{2}$ to 2 in. above the level of the clavicle. The **apices** are either equally high above the clavicles, or the right may reach a shade higher than the left; if the right is a little lower than the left, or the left decidedly lower than the right, there is a probability of past or present disease in the lung whose apex fails to attain the normal limits. Should both apices be very low in level, there may be disease of both lungs. In emphysema both apices are generally found considerably higher than in health. When the examination is made the patient must look straight before him, not turning the head to the side away from the examiner, as this alters the tension of the muscles over the lung. The percussion stroke should be moderately strong, and care should be taken that it

is delivered quite perpendicularly to the surface. Whenever there is any doubt of the apices of the lungs being normal the whole course of their upper borders should be determined. Commencing posteriorly at the level of the spine of the vertebra prominens, the limit of lung resonance in health passes outwards along a line which curves gradually upwards to reach the anterior border of the trapezius, about 1½ in. above the level of the clavicle. Thence it passes obliquely downwards and forwards until it approaches the outer border of the sterno-mastoid, when it inclines more directly downwards towards the clavicle. Sometimes it hardly reaches so far forwards as the sterno-mastoid, at other times the line runs along the surface of the muscle. In cases where the tracheal resonance interferes with the precise delimitation of the lung, the difficulty may be avoided by making the patient open his mouth, thus altering the pitch of the tracheal percussion note.

If disease of the upper lobe of the lung is suspected, it is advisable to percuss along the top of the shoulder from the acromio-clavicular articulation inwards, noting the points at which the pulmonary resonance begins and ends. The distance between these points can then be accurately measured on each side and any difference recorded. The record is valuable for subsequent reference.

The **lower border of the right lung** lies over the liver, and is thin ; therefore its exact situation is best made out by light percussion. Posteriorly, however, the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly. When the patient is obese, very heavy percussion with several fingers may be necessary in order to penetrate the parietes, and bring the lung tissue within the sphere of influence of the blow. In quiet respiration the lower border is found to lie in the mammary line at the 6th rib, in the midaxillary line at the 8th rib,

in the scapular line at the 10th rib, and, nearer the vertebral column, as low as the 10th space.

On the left side the lower border overlaps the stomach, and so the transition is not from lung resonance to dullness, but to tympanitic stomach resonance. Posteriorly, however, the splenic dullness and the dullness of the various solid structures which lie below the lung near the spine are interposed, so that the conditions resemble those found on the right.

The position of the lower border corresponds pretty closely with that on the right side ; it may, however, be found a trifle farther down. In old people the lower borders of both lungs extend beyond these limits ; in children they do not reach them by about a rib's distance lower and higher respectively.

The **anterior border of the left lung** emerges from behind the sternum at the level of the 4th costal cartilage, and forms the upper and left limits of the area of superficial cardiac dullness.

The limits described are exceeded in very deep inspiration, and in diseases such as emphysema, where the volume of the air-containing lung is increased. In pneumothorax the lower border of resonance is often considerably below the limits assigned, and the character of the sound is different (p. 274).

The limits are not attained when the lungs are shrunken or consolidated, when increased abdominal pressure interferes with the normal level of the diaphragm, or when there is effusion in the cavity of the pleura. In this case, should the effusion be left-sided, instead of passing in the anterior axillary line from lung resonance to tympanitic stomach resonance, a band of dullness will be found between the two resonant areas ; and since the lower limit of the pleural sac reaches nearly 4 in. lower at this point than the inferior border of the lung, the dullness will pass down-

wards to a lower level than the normal lung resonance does, and Traube's area (p. 67) will be encroached upon (Fig. 73). In consolidation of the lung, on the contrary, this area will not be diminished.

Since, in health, the borders of the lungs have a considerable range of movement during deep respiration, whilst in the presence of disease the range is often

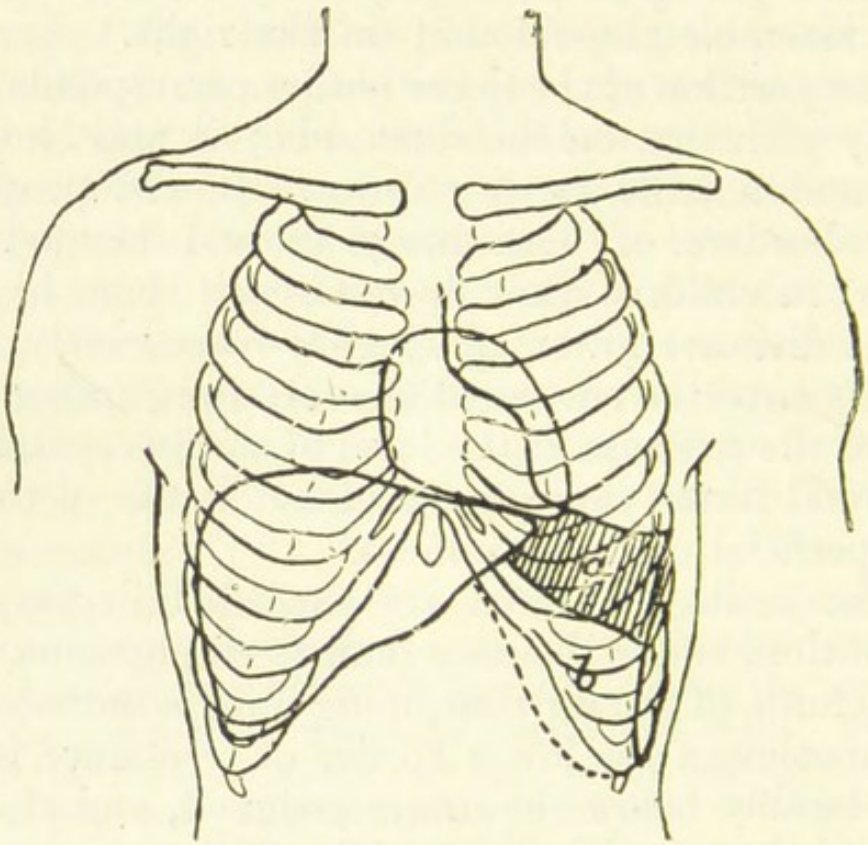


Fig. 73.—Traube's area in pleuritic effusion.
a, portion rendered dull; *b*, portion remaining resonant.

much restricted, it is important to percuss the apices and lower borders of the lungs during both expiration and full inspiration. A unilateral diminution of the range of movement, and still more a unilateral absence of movement either at the apex or base, is an important sign of early infiltration of the lung. Where the range of excursion is deficient at both apices the defect may be due either to bilateral disease, or to imperfect use of the lungs such as is often observed in sedentary

persons. To this procedure Dr. R. W. Philip has applied the name "*tidal percussion.*"

Having outlined the lungs, the **character of the percussion sound over the various parts** must be studied. Beginning in front, the examiner should tap lightly on the most prominent point of each clavicle—being careful to ascertain that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly determine whether under like conditions of percussion the effects on the two sides are identical. Thereafter the other corresponding areas on either side should be carefully compared, many points being systematically percussed in each area, and especially in the supraclavicular triangles. The presence of the heart will obviously interfere, in certain parts of the left side, with the development of a sound resembling that on the corresponding point on the right.

When the front has been fully examined, the observer should percuss in both axillary and infra-axillary regions—the patient meanwhile holding his hands joined above his head; lastly, the various areas posteriorly should be worked out; the patient, if able to sit up, being instructed to fold his arms and bend slightly forwards.

It is most essential at all parts of this examination that the patient's attitude be a comfortable one, and that his arms and shoulders be placed symmetrically. The head must not be inclined to either side.

If any of the regions are unusually hollowed, so that the finger cannot be readily adapted to them, a small cork will make a good pillar pleximeter.

Should the patient's chest be unsymmetrical, equal resonance on the two sides is not to be expected.

In a healthy individual the resonance in the various regions will exhibit certain characteristics:—

Apices.—Clear, not very intense, as the vibrating mass is small, and tending to have a slight tympanitic quality added as the trachea is approached. The right apex is usually rather less resonant than the left.

Clavicular regions. *Sternal end.*—Clear, moderately intense, with tympanitic element due to trachea. *Centre.*—Clear, more intense than in supraclavicular or outer clavicular regions. Devoid of tympanicity. *Outer end.*—As centre, but less intense.

Infraclavicular regions.—Clear and intense. Slightly tympanitic near sternum.

Mammary regions.—Here there is naturally a difference between the two sides: on the right, the lung is encroached on in the lower part of this area by the liver; on the left, the heart occupies a good deal of the space, and the stomach note is elicited through the thin lung at the lower part. In general, however, the pulmonary resonance is clear and fairly intense, except where the neighbouring organs come within the range of vibration. The chest wall here is thicker from the presence both of the pectoral muscles and the mammary gland, and the sounds elicited are consequently more muffled.

In the **inframammary regions** the sounds are greatly influenced by the neighbourhood of the liver, the colon, and the stomach. The lung sound, however, is clear, though not intense, the thin layer of lung becoming rapidly emptier of resonance as its lower border is approached.

In the **axillary regions** the sound is more intense and clearer than elsewhere, diminishing, however, in intensity at the lower part of each lateral area.

Posteriorly, the great masses of muscle which clothe the back muffle the resonance and make it feebler; and therefore firmer percussion, often with several fingers, is required. The scapular region is

most muffled, the infrascapular least so. The interscapular and suprascapular regions are intermediate in quality.

In disease the resonance may be affected (1) quantitatively and (2) qualitatively.

1. **Quantitative.**— **Resonance is increased** in emphysema (slightly), but at the same time the pitch is raised by the greater tension of the chest wall, and this in some cases not only prevents the increased

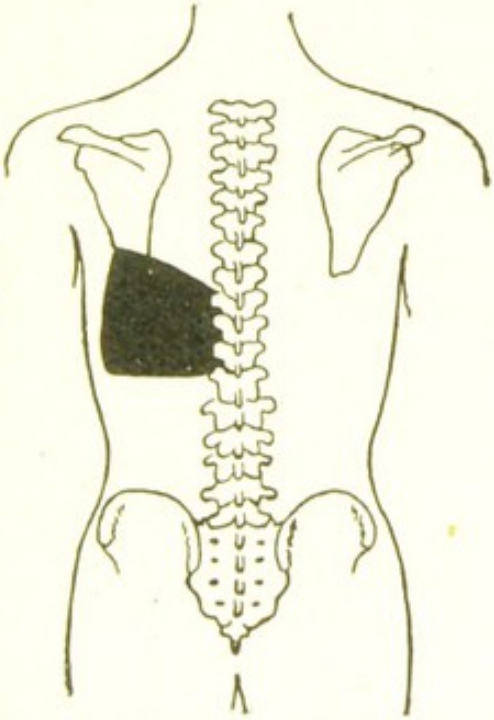


Fig. 74.—Pleurisy, with effusion, seen from behind. (Case 1.)

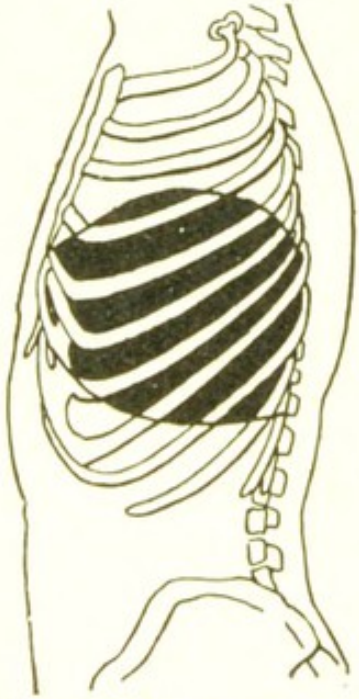


Fig. 75.—Pleurisy, with effusion, seen from the side. (Case 2.)

resonance from being observed, but almost suggests dullness.

When the lung tissue is relaxed, but still contains air, the effect of the septa which subdivide the air columns is for the most part abolished, and the sound becomes distinctly tympanitic. At the same time the resonance is increased in intensity. This is sometimes called **skodaic resonance,** and occurs above the level of a pleural effusion, or in the upper portion of a lung whose lower lobe is affected by pneumonic

consolidation. When air has found its way into the pleural cavity the sound is, as a rule, intensely tympanitic, unless the air be under considerable pressure. A characteristic form of high-pitched tympanitic resonance may be heard, in pneumothorax, by percussion over the front of the chest with a couple of coins—one being used as a plessor and the other as a plexímeter—whilst the observer listens at the back of the patient. In very marked cases the sound is soft and musical, and has been compared to the chiming of a distant church bell; in cases that are less pronounced it approximates rather to the stroke of a hammer on an anvil when heard a long way off.

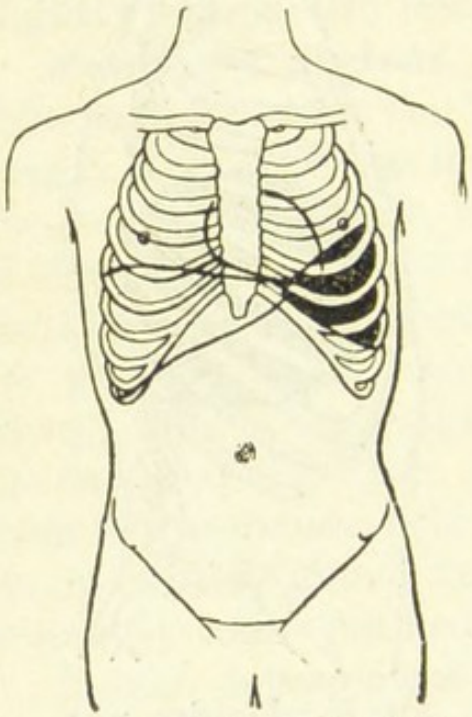


Fig. 76.—Fleurisy, with effusion, seen from the front. (Case 1.)

Instead of making use of coins, the chest wall may be flicked by the finger and thumb whilst the physician auscultates. It will be found that the "flick," which is heard through the stethoscope over the *normal* chest as a dull thud, is converted into a ringing or chiming sound whenever the area of pneumothorax is reached. The alteration in note is more striking than when coins are used, for it is no longer simply a difference in intensity that is observed, but an entire change in the character of the sound.

Cavities in the lung, or the presence of large or medium bronchi within range of the percussion stroke, likewise cause the sound to become tympanitic. A tympanitic sound, which may closely resemble that caused by the presence of a vomica, is heard when the portion of lung which lies between the trachea or primary bronchi and the surface becomes consolid-

ated or retracted. This sound is sometimes called "Williams's tracheal resonance," and is most frequently discovered in the 1st or 2nd intercostal spaces near the sternum.

Resonance is diminished in cases where the pleura is thickened, or where there is consolidation of the lung—either of a whole lobe, as occurs in pneumonia, or of small patches, as in early phthisis. In the latter instance a particular strength of percussion stroke will in each case be found to develop the dullness to the best advantage, according to the size of the solid patch and its distance from the surface. When fluid is present, as in hydrothorax or pleurisy with effusion, the *dullness is absolute*, and an unusual sense of resistance is experienced by the pleximeter finger. In pleurisy with effusion the upper limit of the fluid generally follows a curved line, as is shown in the accompanying figures (Figs. 74, 75, 76).

In the cases of patches of solid lung substance the airless portion is often surrounded by a shell of tissue in which the septa are relaxed. The result of percussion over this composite arrangement is to produce a sound whose resonance is less intense and emptier than the healthy lung would yield, whilst what is left of it assumes a subtympanitic quality. The effect is described as a **boxy or wooden sound.**

2. **Qualitative.**—Several peculiar sounds which are produced by percussion in pathological conditions remain to be noted.

Cracked-pot sound.—This is due to a sudden expulsion of air through a constricted orifice. It occurs in cases where percussion is practised over a cavity which communicates with a bronchus of moderate size, and is most distinct when the mouth is opened. It has a hissing character, combined with a chinking sound like that produced by shaking coins together.

It is also heard in certain cases of thoracic fistula, and occasionally in pneumothorax, as well as in the relaxed lung above the level of fluid in pleurisy, and near the consolidated area in penumonia. If healthy children are percussed while they are crying, a cracked-pot sound is often produced.

Amphoric resonance.—This phenomenon is due to the selective reinforcement of certain vibrations by a large cavity; by this means the overtones are accentuated and die out more slowly.

The following alterations in percussion sounds may be observed under certain pathological conditions; their explanation is for the most part simple from a physical standpoint:—

(*a*) Tympanitic resonance, when due to the presence of a pulmonary cavity which communicates with a bronchus, is raised in pitch when the patient opens his mouth.

(*b*) The pitch of the percussion sound over a cavity varies with the position of the patient. The most obvious explanation is that, if the cavity be partly filled with fluid, this varies in position, and so alters the shape of the cavity when the patient changes his attitude. Other factors, however, often enter into the interpretation of this change.

(*c*) The resonance over a cavity becomes higher in pitch during inspiration, and lower during expiration. The phenomenon depends on the tension of the wall of the cavity.

(*d*) In pneumothorax the metallic resonance is higher in pitch when the patient is lying down than when he sits up.

In certain conditions of malnutrition the muscles on the front of the thorax are unduly irritable. In these circumstances a light tap over the sternum produces fibrillary contractions, at some distance off, in the pectoral muscles. This phenomenon often occurs in phthisis, and is known as *myotatic irritability* or *myoidema*.

Attempts have been made, with some measure of success, to map out not only the limits of the lungs but their several lobes, by placing a vibrating tuning-fork, whose stem ends in a small plate, upon an interspace in front of the chest, and at the same time auscultating posteriorly with an ordinary stetho-

scope. A marked difference can sometimes be observed in the note which is heard when the stethoscope passes from one lobe to another, the tone being clearer and louder when the lobe against which the tuning-fork is placed is the one to which the stethoscope is applied.

V. AUSCULTATION

Auscultation determines—

A. Character of respiratory sounds.

1. Vesicular breathing (rustling in character)
   - Normal.
   - Puerile.
   - Harsh.
   - Jerky or cog-wheel.
   - Feeble or absent.
   - With prolonged expiratory murmur.
2. Bronchial breathing (guttural [*ch*] or aspirate [*ha*] in character)
   - I. Ordinary
     - Low-pitched (cavernous).
     - Medium-pitched.
     - High-pitched (tubular).
   - II. Amphoric (with an echoing quality added)
     - Low-pitched.
     - Medium-pitched.
     - High-pitched.
3. Indeterminate or broncho-vesicular breathing.

B. Vocal resonance.

1. Quantitative changes
   - *Increase*
     - Slight.
     - Marked, Bronchophony.
     - Extreme, Pectoriloquy.
   - *Decrease*
     - Slight.
     - Marked.
     - Entire absence.
2. Qualitative changes
   - Ægophony.
   - Amphoric resonance.

C. Accompaniments.

1. Râles
   - Dry (Rhonchi)
     - Sibilant or high-pitched.
     - Medium-pitched.
     - Sonorous or low-pitched.
   - Moist (Crepitations)
     - *Non-resonant* or toneless
       - Fine.
       - Medium.
       - Coarse.
     - *Resonant* (metallic) or consonant
       - Medium.
       - Coarse.
2. Friction sounds.—Fine, medium, coarse.
3. Splashing sounds.—Hippocratic succussion.

In auscultation three observations must be made at each point examined: First, the character of the breath sounds; second, the character of the vocal resonance; and third, the presence or absence of other sounds.

In order to make these observations with facility, the examiner should attend to the attitude of the patient, which must be as symmetrical and as unconstrained as circumstances will permit. This is easily attained when the patient can sit up; but if he is unable to do this he should be rolled round first to one side and then to the other, in order that the back, and especially the bases of the lungs, may be thoroughly examined. The student must remember that in serious cases great injury may be done to a patient by too prolonged an examination. Care must be taken, especially when an ordinary single stethoscope is used, that the chest-piece is accurately applied, and that no undue pressure is exerted. The patient must be directed to breathe through the nose, regularly and fairly deeply, but not noisily.

A. Character of Respiratory Sounds

There are two typical varieties of breath sound, both of which are audible in health at certain parts of the chest, and these must be carefully studied. The first is known as vesicular breathing, the second as bronchial. The former is heard over healthy lung tissue, the latter over the trachea and main bronchi.

In **vesicular breathing**, which can be heard typically in the axillary and infrascapular regions of a healthy individual, the following facts will be noted:—

The inspiratory sound is fairly intense, and is audible during the whole of the act. The pitch is

low, and the quality is characteristic, being somewhat rustling. It is this quality which is especially described as vesicular.

The expiratory sound follows that of inspiration without a distinct pause—unless, as not infrequently happens, the patient holds his breath for a second at the end of inspiration; it is less intense than the inspiratory sound, is lower in pitch, and lacks the vesicular quality, being more of a simple blowing sound. It only remains audible during the earlier part of the expiratory phase, and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory.

To learn to recognize **bronchial breathing**, the student should listen over the trachea, though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung.

The inspiration sound is moderately intense. It becomes inaudible shortly before the end of inspiration. Its pitch is much higher than that of vesicular breathing, and the quality is blowing or hollow, with a guttural or aspirate intonation.

The expiration sound is generally more intense than the inspiratory; the pitch is often higher; the duration extends through the greater part of expiration, being as long as, or even longer than, the inspiratory sound, from which it is divided by the silent period that marks the end of inspiration. In quality it exactly resembles the inspiratory sound, being aspirate or guttural in character. This quality is sometimes described as "tubular," but the same name is also applied to one of the *varieties* of bronchial breathing, and so is better avoided.

1. The principal variations which can be detected in **vesicular breathing** are as follows:—

i. **Puerile.**—The sounds are harsher than in the adult, but have a similar duration.

ii. **Harsh,** with prolongation of expiration, the character, however, remaining vesicular. This frequently indicates loss of elasticity of lung tissue; hence it often occurs in early phthisis, but may occur in bronchitis.

iii. **Jerky, interrupted, or "cog-wheel" inspiration.**—Here the sound is not continuous, but occurs in waves or sharp jerks. This indicates irregular expansion of the alveoli, due to unequal elasticity in various parts of the lobules, and is therefore not infrequently present in early phthisis. It may also result, however, simply from nervousness, and to carry any weight as a physical sign it must be well marked even on deep inspiration. Even then, however, it may mean little or nothing, and should only take a very secondary place.

iv. **The respiratory murmur may be feeble,** or even inaudible. In quiet breathing the expiratory sound is often quite absent. By making the patient breathe more deeply the murmur may be rendered audible. When marked, this condition may indicate defective expansion.

Total disappearance of the breath sounds usually occurs below the level of fluid in pleuritic exudation, because the relaxed lung does not conduct sounds well, and hence they are not conveyed to the fluid, which is itself a comparatively good conductor (*see* p. 292). If, however, there be only a small quantity of fluid present, the sounds may be faintly heard as the relaxation of the lung tissue is less pronounced. Occasionally it happens that when a considerable quantity of fluid has accumulated, the breath sounds, instead of disappearing, become loud, and possess a marked bronchial character. In such cases the vocal

resonance is also loud, but is usually more or less ægophonic. This exceptional state of matters is most commonly observed posteriorly over the lower lobe of the lung, and may be due to collapse of part of the lung enabling the stronger vibrations which are present in a bronchus to be transmitted to the fluid with less loss of intensity than if they had first required to pass through air-containing lung.

With regard to *prolongation* of the expiratory sound, it must be recollected that in certain diseases, such as emphysema and asthma, the expiratory act is performed much more slowly than in health. In consequence of this, the respiratory sound may also be prolonged; hence in these diseases prolongation of expiration conveys a meaning different from the usual one.

Some patients are habitually shallow breathers, whilst others naturally breathe deeply. The ear can detect these variations, partly by the duration of the respiratory sounds, and partly by their intensity. The depth of breathing as estimated by auscultation is sometimes known as the "*respiratory excursion.*"

2. **Bronchial breathing** may be subdivided into three varieties, according as the laryngeal respiratory sound is conveyed to the ear through consolidated lung from the larger, medium, or smaller air-passages, each of which, by reinforcing certain elements of that sound, gives it a distinctive character.

In the first case we have **low-pitched** bronchial breathing, the more capacious tubes responding best to the deeper-toned elements of the laryngeal murmur; in the second case the pitch is **medium**; in the last it is **high.** Low-pitched bronchial breathing is heard pathologically over moderately large cavities in the lungs, and is hence sometimes called *cavernous;* high-pitched bronchial breathing is heard when

consolidation has occurred round the smaller tubes, as in pneumonia, where the most perfect examples of bronchial breathing may often be found. Here the character is aspirate rather than guttural. This variety is often known as *tubular breathing*.

A special variety of bronchial breathing exists under diseased conditions, and is known as **amphoric respiration.** It resembles the sound produced by blowing across the mouth of a bottle or the muzzle of a gun. The sound, when analysed, is found to consist of one or more low-pitched fundamental tones and a number of high-pitched overtones. It is characteristic of a direct communication between a bronchus and either a considerable cavity with fairly smooth walls or a pneumothorax. The latter condition yields the best examples.

In cases where the resonance of a bronchus is within earshot of the observer, but where at the same time air-containing lung intervenes between the bronchus and the chest wall, the sound of the breathing combines both vesicular and bronchial elements, one or other type predominating according to the exact relations in each case. This variety of breath sound is known as **broncho-vesicular or indeterminate.** In such cases it is usually the expiratory sound which has more or less of a bronchial character. It occurs in health in certain regions where anatomical causes favour its production, especially near the roots of the lungs behind, and in the upper portions near the middle line in front. The resonance of bronchi which lie so deeply in the chest as to be completely muffled by the thick layer of lung tissue that separates them from the ear may become audible when the tissue around them becomes solidified, and thus conducts the sounds more effectively. If the consolidation reach to the surface of the lung the breathing will be

bronchial, but if it fail to extend so far, bronchial breathing will be heard through the vesicular breathing which is still being produced between it and the surface.

The breath sounds must be auscultated in the various regions that have already been examined by percussion, their character in each noted, and similar regions on the two sides of the chest compared; care being taken that the points examined correspond accurately to one another.

B. Vocal Resonance

The second series of observations is directed to **the intensity and character of the vocal resonance.** It varies in intensity even in health on the two sides and over different areas of the lung, being louder on the right side, and more intense the nearer the stethoscope is to the larger bronchi. When the patient repeats the words "one, one, one," or "ninety-nine," the ear receives from the chest no distinct impression of the syllables pronounced, but only a buzzing sound, whose intensity depends on the loudness and depth of the patient's voice and on the conductivity of his lungs. Other words or sentences may be used instead of the above, but on the whole these are well adapted to produce satisfactory and uniform vibration of the chest, and are therefore suitable for the purpose of comparing different points with one another.

An easy way of keeping a standard of intensity in the mind when examining is to conceive of the sound taking rise at different distances from the observing ear.* In some cases the sound is very distant. This

* The same method is to some extent applicable to the examination of breath sounds. It is rather arbitrary, as it does not take account of the differences which are normally found in different areas of the chest; still, it is serviceable, especially for beginners.

is equivalent to "marked decrease" in vocal resonance. Sometimes the sound appears to be produced at a little distance from the chest-piece of the stethoscope. In this case the resonance is slightly decreased, and, to make certain of this, a comparison should at once be made with the corresponding point over the other lung.

In fact, as in percussion and palpation, so in estimating resonance—each point examined on one side of the chest should be at once compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of a single stethoscope. If it seems to be nearer the ear than this, the resonance is increased. When it is near the ear-piece of the stethoscope the increase is "marked," and the condition is often described as **bronchophony.**

If the words become articulate and seem to be spoken right into the auscultator's ear, it will generally be found that even whispered words are clearly heard. This condition is called **pectoriloquy.** Increased resonance occurs when, through any cause, the lung substance conducts the sound-waves set up by the voice more clearly than usual from the bronchi. Consolidation is the commonest cause of increased lung conductivity. Bronchophony occurs when a moderately large bronchus is surrounded by a layer of solid lung reaching to the chest wall. Pectoriloquy is fairly characteristic of a cavity of some size communicating with a bronchus. In some cases, however, a certain degree of pectoriloquy is heard over the front of the upper lobe of the lung when the lower lobe is compressed, as, for instance, by pleuritic effusion. Care must be taken that the articulate sounds do not reach the observer either through the other ear or by

the patient's lips being directed towards the stem or ear-piece of the stethoscope.*

For reasons already explained, vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest wall (*see* p. 280). It is also diminished in cases of thickened pleura, and of emphysema.

In certain conditions the **quality** of the vocal resonance undergoes modification. Pectoriloquy has already offered us an example of such a modification, but a noticeable change also occurs in pneumothorax, when an **amphoric** or metallic **echoing resonance** is imparted to the voice, as well as to the breath and heart sounds. Another alteration in the quality of the vocal resonance is observed in some cases of pleurisy. When the quantity of effusion is rather scanty, so that the lung is only separated from the chest wall by a thin layer of fluid, a nasal or bleating character may be imparted to the voice. This bleating tone is observed much more frequently at the back, near the lower angle of the scapula, or between that point and the axillary line, than it is over other regions of the thorax. It is known as **ægophony,** and is probably due to collapse of the bronchial tubes.†

* Laënnec ("Traité de l'Auscultation Médiate," 2nd ed., vol. i., p. 60, *et seq.*) formally defines pectoriloquy as "la résonance de la voix qui se fait dans une excavation formée accidentellement au milieu du tissu pulmonaire," and this definition was endorsed by the Committee of the International Medical Congress held in London in 1881. There is no doubt, however, as has been indicated by Flint and others, that in certain cases pectoriloquy can be heard over solidified lung in the absence of cavity formation. Probably in most of these cases one is dealing with a somewhat thin layer of solid lung overlying a bronchus. In Germany many authorities use the term as equivalent to well-marked bronchophony, but Laënnec distinguished clearly between them.

† Dr. Stone (quoted in Fagge's "Textbook of Medicine," 4th ed., vol. i., p. 1038) considers that the peculiar quality of the voice is due to the fundamental tone being intercepted by the effusion to a much greater degree than the overtones.

C. Accompaniments

The last series of observations is directed to the detection and recognition of various **adventitious sounds.**

These may arise either in the lung or in the pleura, and it must never be forgotten that sounds by no means very dissimilar may be produced by the friction of the stethoscope on a hairy chest wall; but the latter can usually be suppressed by moistening the skin. The accompaniments arising in the *lung and bronchi* themselves first demand attention.

Such accompaniments are collectively known as **râles,** and are subdivided into dry râles and moist râles. **Dry sounds,** known also as **rhonchi,** are produced in the air-passages, and are due to partial obstruction of their lumen either by swelling of the mucosa or by the presence of tough secretion. The mechanism of their production is thus comparable with that to which cardiac murmurs owe their existence.

They vary in pitch, the variations being in a great measure due to the size of the tubes where they take origin. The smaller tubes are the seat of high-pitched or **sibilant rhonchi,** and these are most abundant during the latter part of inspiration; the medium-sized tubes yield medium-pitched rhonchi, and the larger bronchi produce the deep-toned or **sonorous rhonchi,** which are heard early in inspiration, and may be almost continuous. Dry sounds are characteristic of bronchitis, but are also found quite apart from any definite bronchitis in certain other diseases of the respiratory system, such as cases of phthisis when the bronchial tubes get plugged.

Moist râles, also called **crepitations,** are discontinuous sounds, and are produced either in the alveoli or in the bronchioles and bronchi. They produce on

the ear a noise like the bursting of smaller or larger air-bubbles, and indicate the presence of fluid secretions in the air-cells or tubes. They are classified as fine, medium, and coarse or bubbling.*

Fine crepitations are caused by the opening up of collapsed alveoli whose walls have been agglutinated by the exudation of a little fluid secretion. This at first causes them to adhere, but, as the air-pressure gradually increases during the movement of inspiration, the adhesion at last gives way suddenly, and allows air to enter. The separation of the walls is accompanied by a cracking sound, which can be imitated by separating the moistened forefinger and thumb near the ear. When this condition occurs in a number of alveoli, the combined effect is to produce a sound of fine crepitation. It occurs only † near the end of inspiration, as is to be anticipated from its mode of production, and indicates the presence of exudation in the alveoli of the affected part of the lung. Fine crepitations are very characteristically present during the first stage of pneumonia, and in acute congestion from any cause; they are also met with in early miliary tuberculosis. After atelectasis they are occasionally heard, and in œdema of the lung they are found in association with bubbling râles caused by the simultaneous presence of fluid in the bronchi.

Medium crepitations occur chiefly in the smaller bronchi, and are audible at the end of inspiration and the beginning of expiration. They are caused by the air bubbling through fluid secretion which has been poured out into the lumen of the bronchi.

Coarse bubbling crepitations occur in the larger divisions of the bronchi, and may be heard at almost

* The term "crepitation" is sometimes restricted to the first variety, the others being called fine and coarse bubbling râles.

† Rarely, a few sounds closely resembling fine crepitations are heard during expiration.

any phase of respiration; they may be quite continuous in their occurrence. Coarse crepitations may also originate in pulmonary cavities.

Sometimes the râles are **non-resonant or toneless.** In this case they occur, as a rule, in spongy lung-tissue; but in other cases they are quite **resonant,** and convey an impression to the ear of being all possessed of a definite pitch. There are only two conditions in which resonant râles are present—either consolidation exists, or there is a cavity of sufficient size to act as a resonator for râles which are produced either in itself or in a neighbouring bronchus.

The highest degrees of resonance are known as **metallic** and **tinkling consonances.** Here the râles have a very distinct high pitch, and give the impression of a shower of drops falling into a metallic vessel, which reverberates the sound of their fall. This is associated with amphoric breathing, and, like it, suggests either a large cavity or pneumothorax.

The position where râles are heard greatly influences the importance to be attached to their presence. If heard at the apex, they at once suggest phthisis; whilst medium and coarse crepitation at the bases may be due merely to a transient exudation which will rapidly disappear. When the patient has been breathing quietly for some hours, and especially if he has been lying in bed, a few crepitations, even if heard at the apex, may be due to temporary causes, though they should always be regarded with a degree of suspicion.

The commonest accompaniment arising in the *pleural cavity* is a **friction sound** characteristic of pleurisy at the stage where exudation is not abundant enough to separate the inflamed and roughened surfaces. It possesses a creaking or rubbing character, often quite characteristic; but sometimes, when less well marked, rather hard to distinguish from a râle.

The friction sound may be fine, medium, or coarse. In some instances it is palpable, but, since coarse râles may be so too, this does not serve to distinguish them.

The chief features of difference are that friction sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other, to reappear at a corresponding period of expiration. They are, moreover, unchanged after the patient has coughed, whilst râles may alter under these conditions because of changes in the disposition of the secretion which causes them. The fact that friction is sometimes more localized than crepitation may also be of service. Sometimes friction is markedly intensified by increasing the pressure with which the stethoscope is applied. This acts by causing the roughened surfaces to rub against each other more firmly. Pressure does not affect the intensity of râles. The situation of the doubtful sound, or the presence of pain, or some point in the history of the case, may assist the observer in arriving at the diagnosis.

It must never be forgotten that the presence of one form of accompaniment does not exclude the others. Any two or all three may be found coexisting in one case. When pleuritic friction is developed along the anterior edge of the left lung, and especially when that part of it which is in relation to the apical segment of the heart is affected, the friction sounds often assume the rhythm of the heart beat rather than that of the respiratory movements. Hence the sound is liable to be mistaken for pericardial friction. To distinguish between this so-called *pleuro-pericardial friction* and that of true pericarditis will rarely be very difficult if it is recollected that the former, depending as it does on the apposition of two roughened patches of pleura, is only heard during those phases of respiration when the patches are in contact. Hence a deep

inspiration, by removing one of them from the other, may prevent the production of the sound, whilst in other cases holding the breath, or emptying the lungs as completely as possible, may lead to a like result. In short, pleuro-pericardial friction is much more dependent than true pericardial friction on the movements of respiration.

Hippocratic succussion is the name given to a splashing sound which can be heard when a patient who has both gas and fluid (usually pus) in the pleural cavity is shaken or moves suddenly.

Post-tussive suction is the term applied to a sucking noise, resembling that produced by an india-rubber ball that has been compressed and is springing open again, which is sometimes heard immediately after a cough. It occurs over a cavity in the lung when its walls are not too rigid, and is caused by the re-entry of the air. When distinctly heard it is of considerable diagnostic value, as it can only occur when a cavity is present.

VI. PHYSICAL SIGNS OF THE PRINCIPAL PULMONARY DISEASES

1. **Acute bronchitis.**—The patient is somewhat breathless, and coughs. The sputum is at first mucous and scanty, but subsequently becomes muco-purulent and abundant. On percussion the resonance is normal; on auscultation the breath sounds are vesicular, and accompanied by sonorous and sibilant rhonchi, the latter being especially prominent when the smaller tubes are implicated. The vocal resonance is unaltered.

2. **Chronic bronchitis.**—The signs resemble those of acute bronchitis, but pain is less, and dyspnœa is more marked. The sputum is abundant and muco-purulent. Coarse crepitations are usually abundant.

3. **Emphysema.** — The patient suffers from breathlessness, and is often somewhat cyanosed. He generally has a good deal of cough and some expectoration. The chest is barrel-shaped, and its expansion during inspiration is insufficient, whilst the expiratory phase of respiration is prolonged. On percussion there is a hyper-resonance, sometimes a trace of tympanicity. The borders of the lungs encroach on surrounding organs, and the area of superficial cardiac dullness may be greatly lessened. Auscultation reveals weak breath-sounds and diminution of vocal resonance. This disease is often complicated by chronic bronchitis, and then the breath sounds become harsher than normal, and there is considerable prolongation of the expiratory murmur.

4. **Phthisis.**—The earliest signs of the disease are often loss of weight and appetite, cough, and tendency to sweating during the night. At a later stage one finds severe cough, especially in the morning, sometimes hæmoptysis, increased rate of respiration, diarrhœa, hectic fever, and the other symptoms of an acute inflammatory disease. Inspection reveals in many cases a phthinoid chest with local retraction and defective movement. By palpation one detects increased vocal fremitus; by percussion, localized dullness, especially above or below the clavicle, and sometimes the physical signs of a cavity are apparent. By auscultation one finds that expiration is prolonged, or that the breathing is bronchial in character. The breath sounds are accompanied by crepitations, most of which are of medium size. The phenomena of percussion and auscultation over the apex of the right lung are frequently equivocal: one is only justified in diagnos ing phthisis there when the physical signs are very well marked. The sputum contains tubercle bacilli, and often also elastic tissue.

5. **Lobar pneumonia** is recognized by its sudden onset, with rigors, cough, and pain in the side, associated with fever, which remains continuously high. The face is flushed, the breathing rapid, and the sputum, which is not copious, is rust-coloured and excessively tenacious. The microscope reveals the presence of pneumococci. The physical signs vary with the stage of the disease. *First stage:* Percussion sound rather tympanitic but slightly dull, fine crepitations present. *Second stage:* Absolute dullness on percussion, high-pitched bronchial breathing, increased vocal resonance and fremitus. *Third stage:* Diminution of dullness, disappearance of bronchial breathing, presence of medium and some fine crepitations; vocal resonance and fremitus return to normal.

6. **Chronic interstitial pneumonia.**—The patient complains of some breathlessness on exertion, and of cough with rather copious muco-purulent expectoration. The sputum may be fetid, or, when the disease is a pneumoconiosis, may contain characteristic elements. The physical signs are a gradually developed flattening over the affected region, where also expansion is absent, or defective and delayed; the shoulder of the diseased side droops. On percussion there is a dull area surrounded by one where the resonance is boxy, and the heart is drawn over by the contracted lung. Auscultation reveals feeble or bronchial breathing, and a few crepitations and rhonchi. The vocal resonance and fremitus are exaggerated.

7. **Pleurisy** is characterized by the presence of fever, pain in the side, restrained but rapid breathing, and suppressed dry cough. In the earliest stage fine friction is audible before any other abnormal signs can be detected. Subsequently, as fluid gathers, the affected area becomes dull, whilst the lung above yields a tympanitic resonance. As the dullness increases,

the breath sounds, vocal resonance, and fremitus diminish in intensity, and at last they wholly disappear. Above the level of the fluid, during the height of the disease, the breathing may be somewhat bronchial and accompanied by fine crepitations. As the fluid is reabsorbed in process of recovery, the dullness again decreases, whilst the breath sounds and vocal resonance gradually return, and when the fluid has almost disappeared friction is once more heard, but now of a much coarser character than at first. In certain cases, for some time during the advance and again during the recession of the disease, ægophony occurs. In massive effusions the neighbouring organs are displaced, and there may be bulging of the intercostal spaces.

In cases of pleurisy with effusion an area of paravertebral dullness is often found on the opposite side from that where the disease exists. This area takes the form of a right-angled triangle whose apex lies about the level of the upper border of the effusion, and whose base, corresponding in level to the lower border of the lung, extends to a distance of 2½ or 3 in. from the spine. It varies somewhat in extent with the attitude of the patient, and is frequently observed to diminish when the patient lies on the affected side. It is usually known as **Grocco's triangle.**

8. **Pneumothorax.**—The patient complains of sudden pain and breathlessness. The affected side is distended and immobile, or lags behind the other. On percussion there is a loud, deep resonance more or less tympanitic, and by coin-percussion a characteristic ringing sound is elicited. The breath sounds and vocal resonance are absent, or, if a bronchus communicates with the pneumothorax, are replaced by amphoric breathing and resonance, whilst if fluid is present one may hear metallic tinkling and elicit Hippocratic succussion. The surrounding viscera are displaced.

9. **Hæmorrhagic infarction** of the lung

occurs in the course of valvular heart disease, and is characterized by the sudden onset of pain associated with blood-stained expectoration. If the infarct is near an accessible portion of the surface of the lung, one can discover a patch of dullness, with altered breath sounds and crepitations.

10. **Asthma** may usually be regarded as a symptom rather than a disease. Cardiac asthma has already been described, and asthmatic conditions may likewise arise from polypi or other sources of reflex nasal irritation, or from disease of the stomach or kidneys. The form known as bronchial or "spasmodic" asthma results mainly from spasm of the muscles of the smaller bronchi. In it the patient is found sitting up or leaning forward with the hands fixed on some object in order to give additional purchase to the accessory muscles of respiration. The face is flushed and the vessels are turgid. Expiration is prolonged and laboured, and the lungs are too full of air. Percussion yields a somewhat hyper-resonant sound. Auscultation reveals at first musical râles and wheezing sounds, with marked prolongation of expiration. After secretion has been established, deeper rhonchi become audible. The sputum, which is scanty, contains small lumps, in which Curschmann's spirals and Charcot-Leyden crystals are found.

11. **Signs of pressure on a main bronchus.**—The signs in this condition are not uniform, but the following are fairly typical: There is no marked dyspnœa until an advanced stage of the disease, except after a severe paroxysm of cough, but the rate of respiration is usually somewhat increased. The patient often suffers from spasmodic cough, caused by the difficulty of forcing tough sputum through the stricture. On examining the thorax one finds that the *sound side* is expanded, and shows a large respiratory

excursion and low position of the diaphragm, whilst the breath sounds are vesicular, but harsher than usual. On the *affected side* there is usually some general retraction. Mobility is limited or wholly absent. Inspiratory retraction of the intercostal spaces often occurs. A peculiar respiratory vibration is sometimes felt near the constricted part of the bronchus, and vocal fremitus is diminished over the whole side of the chest. On percussion the resonance is normal in the earlier stages, but as the access of air becomes more obstructed the note rises in pitch. Eventually dull areas due to tumour or pleurisy may appear. On auscultation the breath sounds are at first harsh and blowing, especially in the upper interscapular region; subsequently they generally grow weak, but a rough respiratory murmur is audible near the point of stenosis. Finally, the breath sounds are wholly extinguished, and a deep, sonorous rhonchus, best heard in the interscapular region, alone remains. Vocal resonance is diminished, and may be almost ægophonic. The changes in the lung may affect neighbouring viscera, and the heart may be drawn towards the affected side by the retraction of the airless lung, or thrust from it by the growth of an aneurysm or malignant tumour. Pain is less common than a sense of localized oppression.

VII. THE SPUTUM

The characters of the cough have already been treated of in a previous chapter (Chap. II., p. 38). It remains to add a few notes on the appearance and examination of the sputum in different diseases.

Naked-Eye Inspection of Sputum

The following are the principal points to be observed with the naked eye :—

1. Quantity.
2. Consistency.
3. Whether homogeneous or in layers of different appearance.
4. Whether frothy or airless.
5. Colour and transparency.
6. Odour.

The above qualities depend on the character of the material which is coughed up. The main varieties are mucous sputum, serous sputum, fibrinous sputum, purulent sputum, and blood. In many instances transition types between them are observed.

Mucous sputum is characteristically present in early bronchitis. It is clear, tough, and sticky. As a rule, the amount is not great. At a later stage of bronchitis the mucus is mixed with pus cells. The sputum is then less tough, more copious, and has a greenish-yellow colour.

Muco-purulent sputum occurs in many diseases of the lung. In phthisis with cavity formation one often finds small ragged lumps of muco-pus, surrounded by mucus, which are heavier than the other constituents since they are airless. They therefore sink to the bottom and become more or less flat and button-like. This constitutes the "**nummular**" sputum of phthisis. If there be a fair amount of serous or watery fluid mixed with such sputum it gradually settles into three layers, the lowest being purulent, the next serous, and the uppermost composed of frothy mucus.

Sputum composed of pus alone usually proceeds from an abscess which has ruptured into the lung or air-passages.

Serous sputum occurs apart from mucous expectoration as a thin, watery fluid, generally blood-stained. It indicates œdema of the lung. Pulmonary

œdema without extravasation of blood yields a white frothy sputum like soapy water.

Blood may be coughed up alone, or the sputum may be more or less bloodstained. It must be distinguished from blood brought into the mouth from epistaxis, gastric hæmorrhage, or bleeding from varicose veins in the walls of the œsophagus. Its brighter colour and its frothy appearance often make the discrimination perfectly simple. When it comes from the lungs its presence may result either from pulmonary or cardiac disease, or from aneurysm.

Several diseases cause a **characteristic coloration of the sputum.** Thus, in pneumonia it is **rusty,** and so viscid that it often will not fall out of an inverted spittoon ; it is **bright-yellow or green** when a liver abscess has ruptured into the lung, and the latter colour also appears in some cases of pneumonia. Sometimes, when an amœbic hepatic abscess has discharged by the lung, the sputum has the appearance of **anchovy sauce. Black sputum** is common with coal miners, whilst red-streaked sputum is suggestive of phthisis. **Prune-juice sputum** occurs when blood lingers in a lung which has become œdematous. Thus it is found in cases of chronic pneumonia that are going on to disintegration of the lung tissue. **Red-currant-jelly sputum** is said to be characteristic of malignant disease in the lung. It has also been found in hysteria.

The **quantity** of sputum coughed up in twenty-four hours is important ; and still more so whether large quantities are rapidly got rid of at considerable intervals, or whether it comes away in small amounts and frequently.

Occasionally small **casts of bronchi** are to be found in the sputum, but the examination for formed elements is best conducted with the aid of a microscope.

The **odour of the sputum** is seldom very characteristic. Ordinarily it has a "stale" smell, but in cases of gangrene of the lungs, of fetid bronchitis, and of bronchiectasis it may develop an exceedingly penetrating putrid odour. An unpleasant odour may also be acquired during its transit through the mouth.

Microscopical Examination of Sputum

Generally it is well first to examine an unstained and fresh specimen, and thereafter to use special methods for the recognition of bacteria. To select a suitable piece, place the sputum in a flat glass vessel, which can be laid on either a white or a black background as is found convenient. Mixed with the amorphous mucous exudation which forms the basis of the sputum may be seen various organized and crystalline substances, of which the following are the principal groups :—

1. **Cellular structures.**—i. **Pus corpuscles** in various stages of granular degeneration and with several nuclei.

ii. **Epithelium** from the mouth, air-passages, and alveoli. The latter may contain pigment which has reached them from the air, or they may exhibit a very characteristic iron-containing pigment, which is unusually abundant in cases of heart disease with pulmonary congestion, and indicates brown induration of the lung. This pigment yields the hæmosiderin reaction on the addition of hydrochloric acid and potassium ferrocyanide.

iii. **Salivary corpuscles** are picked up by the sputum in its passage through the mouth.

iv. **Red blood-corpuscles.**—A few are of no importance. Large numbers occur in hæmoptysis.

v. **Eosinophil cells** occur in asthma, and are

often associated with Charcot-Leyden crystals (Fig. 80). They are large, and contain numerous fine granules which stain with eosin.

2. **Elastic fibres** indicate destruction of lung tissue, whether from phthisis, gangrene, or abscess. In gangrene only a few fibres escape the destructive process. They are found in the small tough lumps of the sputum, and are best demonstrated by a rapid heating with an equal quantity of 10 per cent. solution of caustic soda, which liquefies the other

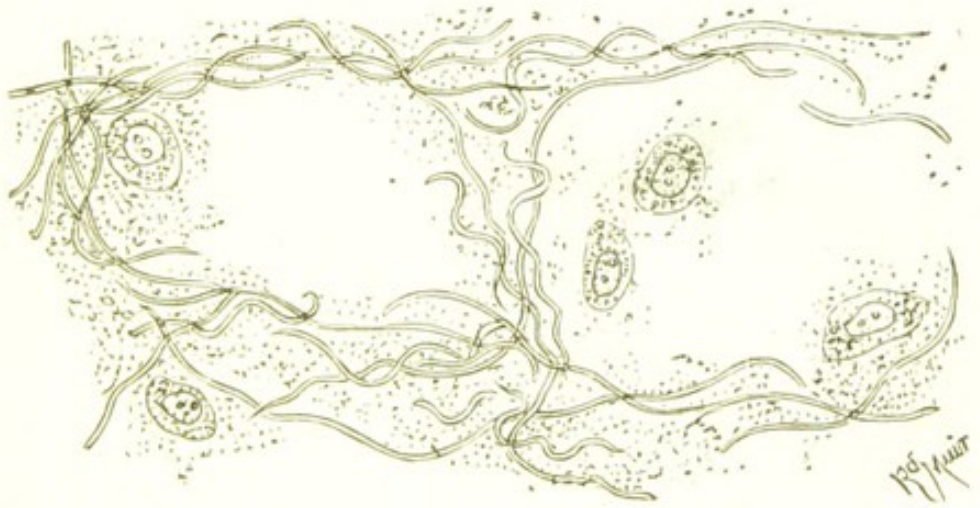


Fig. 77.—Elastic tissue from lung in sputum of a case of phthisis. × 300.

elements more quickly than these fibres. After boiling, a gelatinous mass is left, to which a considerable quantity of water should be added and the mixture left in a conical glass till the elastic fibres settle to the bottom. Thus they may be isolated, and in well-marked cases exhibit the alveolar arrangement of the lung tissue. Too prolonged an exposure to the caustic will lead to the solution of the elastic fibres as well as of the other constituents. (Fig. 77.)

3. **Fibrin casts,** often large enough to attract the unaided eye, are still more frequently visible under a low power of the microscope. (Fig. 78.)

4. **Curschmann's spirals** are found in the

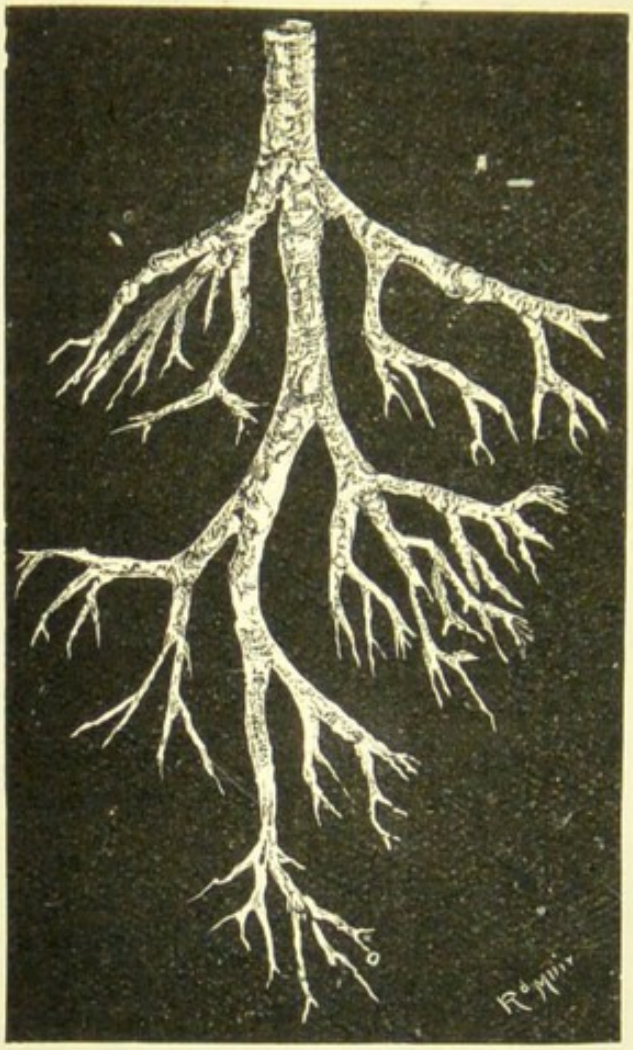


Fig. 78.—Bronchial cast from a case of plastic bronchitis. Natural size.

sputum of asthmatic patients. Some of the sputum should be spread out on a piece of glass on a black surface. The spirals look like little sago grains. When unrolled they appear as convoluted threads which may be quite an inch in length. Under the microscope they show a central core, round which a sheath of tough mucus, with a large number of small round cellular elements in it, is coiled (Fig. 79).

5. **Crystals.** — i. In asthma, fine colourless crystals with sharp extremities are often found. They are frequently associated with the spirals already described, and are known as **Charcot-Leyden crystals.** They are probably phosphates of an organic base. (Fig. 80.)

ii. **Fatty acid crystals** are needle-shaped, and generally occur in clusters.

iii. **Cholesterin** occurs in rhomboidal plates, which generally have a small notch in one corner. They are met with in old purulent sputum from pulmonary cavities, but their presence is uncommon.

iv. **Hæmatoidin crystals** occur where there has been an old hæmorrhage in cases of abscess and empyema. They have a characteristic brown-yellow colour, and appear as needles, rhombi, and plates.

v. **Leucin** and **tyrosin** may be found on rare occasions in pus from old perforated empyemata.

6. **Parasites.**—These belong both to the animal and vegetable kingdoms. Of animal parasites **echinococci** are the most important. The presence of hooklets, and still oftener of fragments of the laminated ectocyst, is the usual indication of their existence (*see* p. 106). In China and other parts of Eastern Asia, **Distōma pulmonale** is frequently found. Its presence causes sharp attacks of hæmoptysis, which may be mistaken for the hæmoptysis of phthisis. Microscopic scrutiny of the sputum, however, generally reveals characteristic ova (Fig. 81).

Fig. 79.—Curschmann's spirals in sputum. × 200 and natural size.

Fig. 80.—Charcot-Leyden crystals. ×350

The vegetable parasites are fairly numerous. Besides bacteria, which are considered in a separate

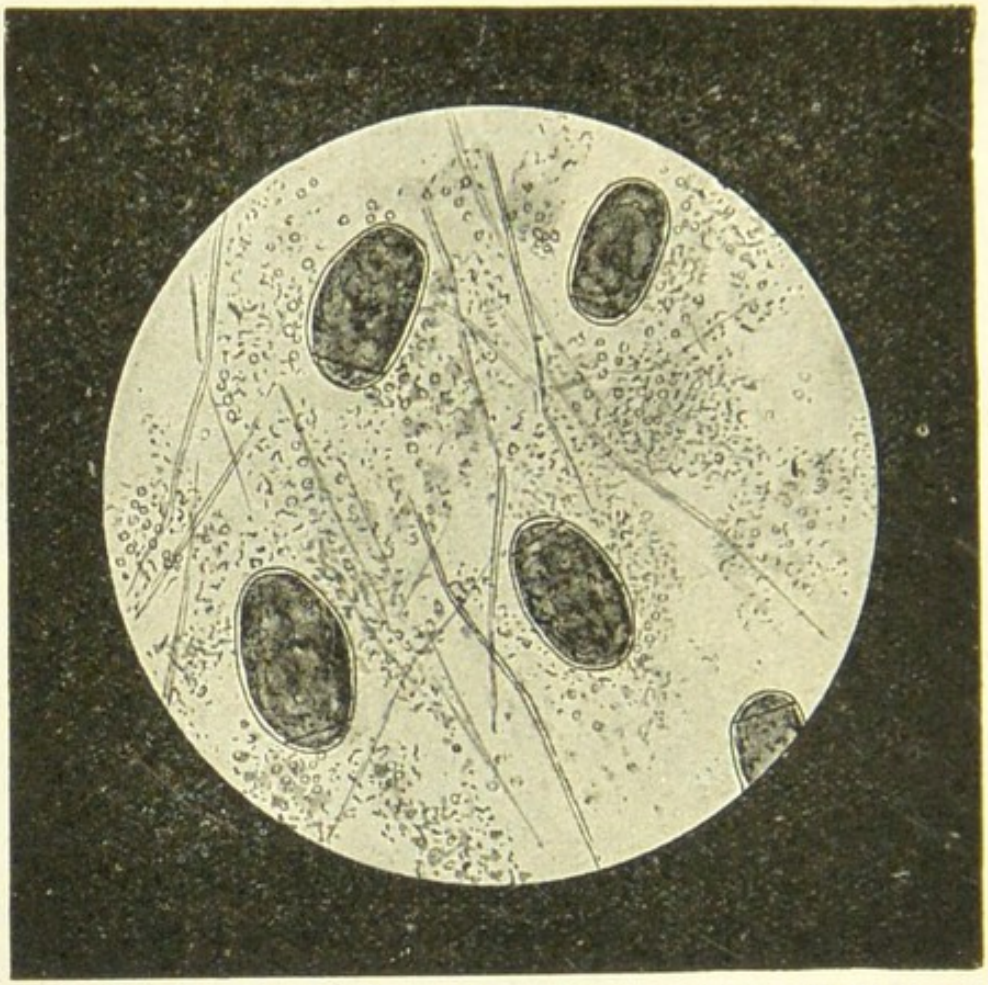


Fig. 81.—Ova of Distōma pulmonale in sputum. High power.

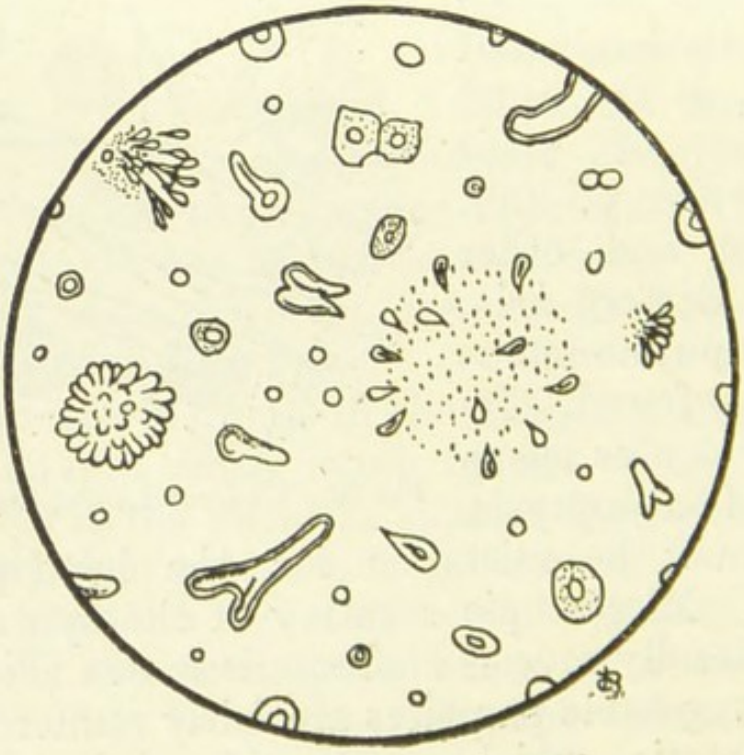


Fig. 82.—Actinomyces in sputum.

chapter (p. 587), and amongst which the most important are tubercle bacilli, pneumococci, and Pfeiffer's bacillus, some of the higher fungi are also found, the most important being **actinomyces** and **aspergillus fumigatus** (Figs. 82, 83).

Fig. 83.—Aspergillus fumigatus. × 300.

APPENDIX TO CHAPTER VI

On Graphic Methods of recording the Conditions observed in the Heart and Lungs

1. **Full-size outlines of the heart.**—The position of the nipples, and the outlines of the clavicles, ribs, and sternum, should be carefully traced on the chest with a dermatograph pencil, and the same should be done for the outlines of relative and absolute dullness of the heart and liver. These tracings should then be gone over rapidly with a small paint brush dipped in sweet almond oil, and a sheet of tissue-paper pressed down upon the patient's chest. The oil will leave a mark on the paper, which can be more strongly traced with a pencil after the paper has been removed.

An alternative method is to **photograph the chest** with the lines drawn in. This has the convenience of preserving the record in a less bulky form. A scale of inches should in this case be laid across the patient's epigastrium to permit of absolute measurements being taken from the photograph.

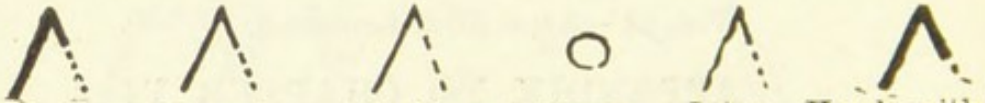
2. **The use of symbols on outline charts.**

i. **The heart.**—The presence and position of murmurs are easily indicated by shading. Their intensity is roughly represented by the heaviness of the shading. Examples of this method are seen in Figs. 35, 36, and 41. When two synchronous murmurs are present, and one wishes to show where the first, after becoming fainter, gives place to the second, which grows increasingly loud as the stethoscope is carried along the line joining their areas of maximum intensity, one makes use of the musical signs of diminuendo and crescendo, > and <. The situation of pericardial friction is indicated by a zig-zag line, ∿∿∿. When the apex beat does not reach the edge of the deep cardiac dullness, its situation is shown by a small cross, **X**.

ii. **The lungs.**—The position of any dull area is indicated by shading. If, following the suggestion of Prof. Sahli, one represents superficial dullness in blue chalk and deep in red, one can not only make the record clearer, but can also superpose on the same chart auscultatory phenomena in black symbols without clogging it. The auscultatory phenomena are well represented by the following symbols * :—

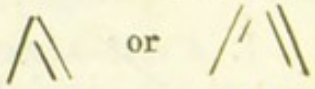
1. Types of breathing :—

(*a*) Vesicular.



Puerile. Normal Adult. Feeble. None. Interrupted. Harsh, with expiration prolonged.

(*b*) Transition type. } "Broncho-vesicular" or indeterminate.



or

(*c*) Bronchial.

Tubular or High-pitched. Medium-pitched. Low-pitched.

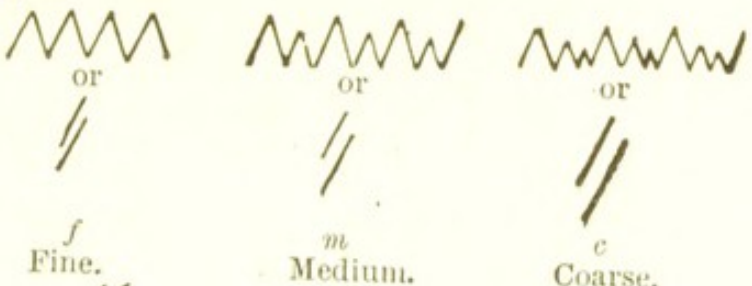
(*d*) Amphoric.

High-pitched. Medium-pitched. Low-pitched.

* Mostly after Professor Wyllie.

2. Accompaniments :—

(*a*) Friction.



(*b*) Dry râles.

(*c*) Moist râles :—

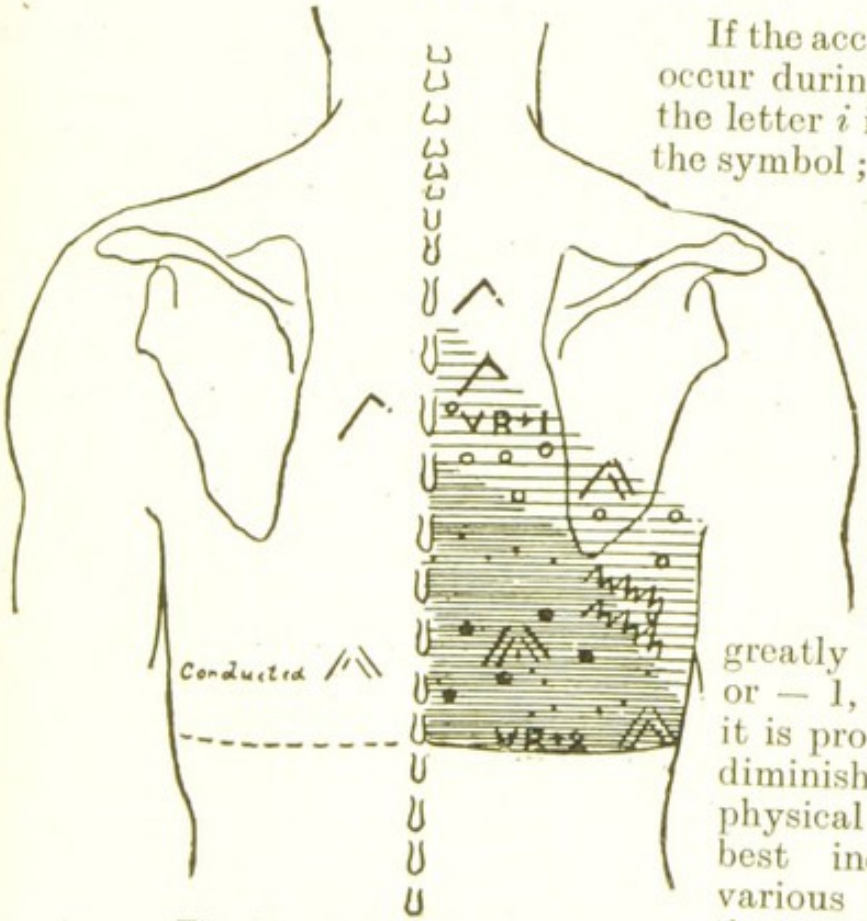


Fig. 84.—Record of pneumonia.

If the accompaniments occur during inspiration, the letter *i* is prefixed to the symbol ; if during expiration, *e*.

Vocal resonance is indicated by the letters V R, followed by + 1, + 2, + 3, if it is slightly, moderately, or greatly increased ; or — 1, — 2, — 3, if it is proportionately diminished. Other physical signs are best indicated by various letters of the alphabet. The accompanying diagram (Fig. 84) illustrates the application of these symbols to a case of pneumonia.

CHAPTER VII

THE URINE

The method of interrogating a patient whose symptoms point to an affection of the urinary system has already been described (p. 9), and the physical examination of the kidneys has been considered along with that of the other abdominal organs (p. 77).

In this chapter we propose to take up the examination of the renal secretion.

Collection of samples.—Owing to the variations in the composition of the urine at different times of the day, the sample examined should, if possible, be taken from the total urine of the twenty-four hours. If only one sample can be obtained, it should be that which is passed about three hours after taking a meal, as abnormal ingredients are then more likely to be present. The sample should be poured into a tall conical glass, covered, and allowed to stand for some hours in a cool place. If it be desired to preserve the urine for some time, two or three drops of formalin or some powdered thymol should be added.*

Any suspended matters soon settle to the bottom of the glass, and the examination of the sample may then be proceeded with. This should be conducted (1) physically, (2) chemically, (3) microscopically.

* The preservation of urine is beset with some difficulties. Formalin reduces Fehling's solution, and may therefore lead to an erroneous conclusion as to the presence of sugar; thymol may give with Heller's test a ring closely resembling that given by albumin. Of the two, thymol is preferable; it has no reducing power and may be used for diabetic urine. A little of the solid should be placed in the receptacle in which the twenty-four-hour sample is to be collected.

I. PHYSICAL EXAMINATION

Attention should be paid to the following points, viz.: (1) Quantity, (2) colour and transparency, (3) consistence, (4) odour, (5) density, (6) cryoscopy, (7) naked-eye characters of the deposit.

1. **Quantity.**—The amount of urine passed during the day should be measured separately from that passed during the night. The sum of the two gives the total for twenty-four hours. The bladder should be emptied at a fixed hour—say 8.30 a.m.—and the product discarded. All the urine passed during the day is carefully collected, and the bladder emptied again at 8.30 p.m., the product being added to the day's secretion. This is the amount of the "*day urine.*"

The bladder is again emptied at 8.30 next morning, and the product added to that which has been passed during the night. The total quantity is the "*night urine.*" This added to the day urine gives the total for twenty-four hours.

It is often difficult to collect all the urine that is passed, some being lost with the motions. This is especially the case with children, female patients, and those who pass their evacuations involuntarily. Where great accuracy is required, recourse must be had to the catheter.

A healthy adult male passes on an average 50 oz. (1,450 c.c.) of urine in twenty-four hours; women, a few ounces less.

The following table represents the amount of urine passed daily by children of different ages (Holt):—

First twenty-four hours	0 to 2 oz.
Second twenty-four hours	$\frac{1}{3}$ „ 3 „
Three to six days	3 „ 8 „
One week to two months	5 „ 13 „
Two to six months	7 „ 16 „

Six months to two years . . .	8 to 20 oz.
Two to five years	16 ,, 26 ,,
Five to eight years	29 ,, 40 ,,
Eight to fourteen years . . .	32 ,, 48 ,,

Churchill, in a paper read before the American Pediatric Society in 1898, asserts, as the result of his own observations, that the amount of urine passed by children is less than is usually supposed.* He gives the following averages of quantity and specific gravity at different ages:—

Age	*Quantity*	*Sp. gr.*
3 years . .	358 c.c. . . .	1024
4 ,, . .	299 ,, . . .	1027
5 ,, . .	392 ,, . . .	1024
6 ,, . .	405 ,, . . .	1023
7 ,, . .	564 ,, . . .	1018
8 ,, . .	628 ,, . . .	1021
9 ,, . .	731 ,, . . .	1020
10 ,, . .	768 ,, . . .	1023
11 ,, . .	716 ,, . . .	1018
12 ,, . .	829 ,, . . .	1021

Above the age of 15 the quantity passed is about up to the adult standard.

It will be observed that, relatively to their weight, children pass more urine than adults. This is to be attributed to the relatively greater activity of the metabolic processes in children and to the more fluid nature of their diet. The above quantities, however, are only roughly approximate, and in many cases one will find that the amount of urine excreted by a child of given age is smaller than the quantity tabulated.

Normally, very much more urine is secreted during the day than during the night. The normal proportion of day urine to night urine is 100 : 25–60. Approximation of the night quantity to that of the day is always abnormal, and is especially apt to occur in chronic renal disease, of which, indeed, it may

* *See also* "An Essay on Clinical Urology in Infancy and Childhood," by Fernandes Figueira, *Lancet*, Sept. 12th, 1896.

constitute one of the earliest signs. Thus the proportion of day to night urine may become 100 : 100 or even 200. The solids are increased in proportion to the water.

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink, and after exposure to cold. Conversely, one finds the *secretion diminished* when little food or drink has been taken, and after exposure to heat—especially if followed by sweating.

A **pathological increase** in the urine occurs in diseases associated with an increased arterial pressure —e.g. granular kidney ; also in both forms of diabetes, during the absorption of exudates, and in some neurotic conditions—e.g. hysteria. **Abnormal diminution** of urine is found where the arterial pressure is lowered or the intravenous pressure in the kidney increased—e.g. in acute nephritis and in advanced mitral disease ; also in all fevers, in diarrhœa and vomiting, and in cerebral irritation—e.g. concussion.

2. **Colour and transparency.**—Normal urine is said to have the colour of amber or pale sherry. The exact tint fluctuates widely even in health, depending upon the degree of dilution and upon the reaction. An acid urine is always darker than one which is alkaline, even when they are equally concentrated. The colour of normal urine is mainly due to a yellow pigment, to which the name of urochrome has been given. The pigments uroerythrin and urobilin only occur in very small quantity in the urine under normal conditions. In febrile and some other diseases, however, a large quantity of urobilin may appear in the urine. The latter has then a warm orange colour, and usually shows a dull pink tint at the apex of a conical glass. Urobilin is not present in freshly voided normal urine, its place being taken by its precursor urobilinogen.

On standing, this becomes converted into urobilin, giving the urine a darker colour. The change takes place in a few minutes if hydrochloric acid be added to the urine, being particularly noticeable when excess of urobilinogen is present. Excess of urobilinogen may be tested for with the following reagent: Dimethylparaminobenzaldehyde 2 grm., acid. hydrochlor. 5 per cent. 100 c.c. Ordinary urine gives a yellow colour, going red on heating; with excess of urobilinogen the red colour appears *in the cold.*

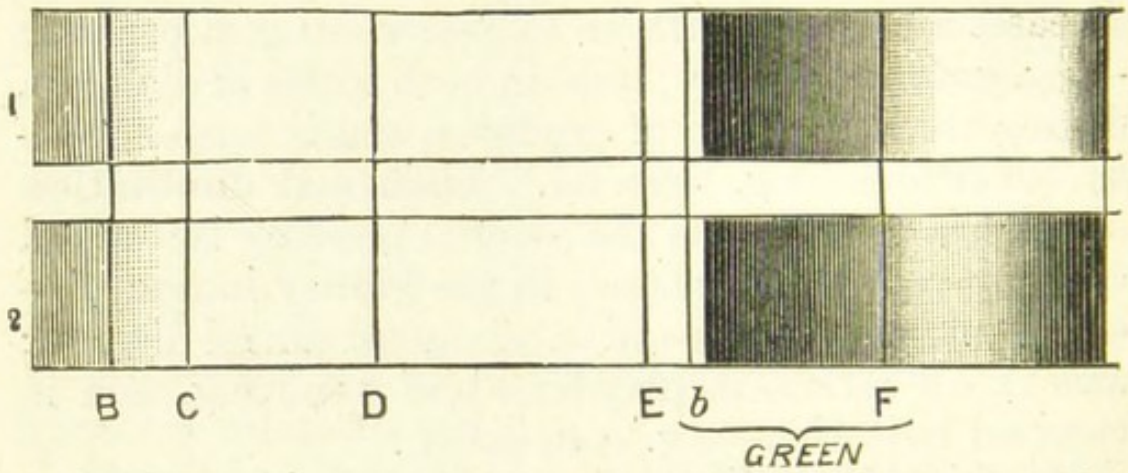


Fig. 85.—1. Spectrum of urobilin; 2. Spectrum of urobilin masked by other pigments. (*See* text.)

If urobilin urine be examined spectroscopically * in a thin layer the urobilin band will be seen in the green between *b* and F (Fig. 85). Such a urine is often dichroic—looking red by transmitted and green by

* The following directions for the spectroscopic examination of urine are given by A. E. Garrod :—

1. Use a small direct-vision spectroscope.
2. Examine the urine in a 6-oz. conical glass. This permits of the inspection of layers of different thickness.
3. Hold the slit 1 in. from the glass, and move it up and down the entire length of the cone.
4. Either daylight or artificial light may be employed.
5. If in doubt as to the bands, shake up 400 c.c. of the urine with 50 or 60 c.c. pure amylic alcohol and a few drops of acetic acid. Collect the layer which floats. Clear it, if need be, by the addition of a little ethyl alcohol, filter, and examine.

reflected light. The presence of excess of urobilin may be confirmed by rendering the urine strongly alkaline with ammonia, filtering, and adding to the filtrate a few drops of a 10 per cent. solution of chloride of zinc. If excess of urobilin be present, the solution becomes fluorescent.

The table on p. 312 shows the chief varieties of alteration in colour of the urine, with their causes.

Normally, when freshly passed, urine is quite transparent, but it may be **opalescent** from the presence of various substances in suspension. If the opalescence persists after filtration, it is due to the presence of bacteria.

A slight opalescence which causes the urine to look smoky is produced by the presence of small quantities of blood (*see* p. 347).

Alkaptonuria.—This is a condition in which the urine is natural-looking when passed, but, when exposed to the air, becomes gradually darker from the surface downwards; ultimately it may be dark brown or black. It is due to the presence in the urine of dihydroxyphenyl acetic (homogentisinic) acid.

The addition of an alkali causes the urine to become dark at once. Such urine reduces alkaline solution of cupric oxide, but the bismuth test for sugar is negative. With Millon's reagent it gives a yellow precipitate, and the addition of dilute ferric chloride drop by drop causes a passing deep-blue colour. Such urine, moreover, will not ferment with yeast nor turn the plane of polarized light.

The condition is a rare anomaly, attended by no symptoms beyond sometimes a slight dysuria and frequency at night. It is due to an inborn error affecting the breaking down of the tyrosin linkage in the processes of protein metabolism.

Urines containing melanin also become darker on

Alterations in Colour of Urine

Colour	*Cause*	*Condition or Remarks*
Nearly colourless.	i. Large amount of urine excreted.	Much drinking. Nervous conditions, diabetes insipidus, etc.
	ii. Diminution of pigment.	
Orange-coloured.	i. Small amount of concentrated urine.	Hard muscular work. Fevers.
	ii. Increased pigment.	Fevers.
	iii. Occasionally bile pigment.	Jaundice.
Orange-coloured. / Reddish-brown.	Administration of rhubarb, senna, chrysophanic acid.	(These turn yellow with acid, red with alkali; a normal colour going red with alkali = phenolphthalein.)
Dark-brown.	Methæmoglobin.	(P. 349.)
Red.	i. Blood.	(P. 347.)
	ii. Aniline dyes in sweets, etc.	
Port wine.	Hæmatoporphyrin.	(P. 349.)
Brownish-black.	i. Melanin.	Melanotic sarcoma. (Darkens on standing, does not reduce Fehling's solution, gives greenish-brown precipitate with ferric chloride.)
	ii. Much hæmoglobin.	(Yields chocolate deposit.)
	iii. Alkaptonuria.	(P. 311.)
Greenish-black.	i. Hydroquinone, carbolic acid, salol, guaiacol, resorcin, naphthalin, etc.	
	ii. Bile.	In old-standing cases of jaundice.
Yellowish-green. / Green.	i. Bile.	Jaundice (p. 366.)
	ii. Santonin.	(Turns red with alkali.)
Yellowish and milky.	i. Pus.	(P. 368.)
	ii. Fat as (*a*) emulsion, (*b*) droplets.	Chyluria.
		Lipuria in advanced renal disease.
Greenish-blue. / Blue.	i. Excess of indigo-forming bodies.	Typhus.
	ii. Administration of methylene blue.	(Violet with alkali; cuts off red and yellow in spectrum.)

exposure to the air, owing to oxidation of the pigment, but they do not reduce cupric oxide.

Carbolic-acid urine also becomes darker on exposure to air, owing to the oxidation of the hydroquinone which it contains into pigments similar to those in alkaptonuria.

In alkaline urines an **iridescent pellicle** frequently appears on the surface. When the urine has cooled this can be skimmed off like a thin brittle film. It is composed of phosphate of lime. The idea formerly entertained that such a pellicle occurs especially in the urine of pregnancy is groundless.

3. **Consistence of urine.**—In health the urine is quite watery in consistence. If much sugar or bile be present it is less mobile, and in the presence of bile or of much albumin the froth which forms on shaking is more persistent than is usual. Alkaline urine containing pus may be quite ropy. A special alteration in consistence occurs in the condition known as **fibrinuria.** When this is present the urine is reddish-yellow when passed, but soon sets into a jelly, which contracts somewhat on standing. If only little fibrin be present the whole urine may not coagulate, but a sticky sediment forms at the bottom of the vessel. Fibrinuria is due to the entrance of blood plasma into the urinary tract. It occurs as a very rare symptom of villous growth in the bladder, and sometimes also after the administration of cantharides. The fibrin may be recognized as such by washing it in 5 or 10 per cent. salt solution, with the addition of a little thymol, and then placing in 1 per cent. HCl. It swells up, but is not dissolved unless pepsin be also added.

4. **Odour.**—Normal urine has a characteristic "aromatic" odour. When the urine has stood for some time the odour becomes ammoniacal. In cases

where there is an abnormal communication between some part of the urinary tract and the intestine the odour may become fæcal. In acetonuria the odour is fruity. After the administration of turpentine the urine has an odour like violets. Cubebs, santonin, and some other drugs also impart to it their peculiar smells. In diabetes the odour has been compared to that of new-mown hay.

5. **Density.**—Clinically the specific gravity of urine is always taken with the instrument known as a **urinometer.** An ordinary urinometer is graduated for a temperature of 15° C., and will record variations in specific gravity from 1000 up to 1060.

How to use the urinometer.—The urine should be allowed to cool, and should be placed in a tall jar, wide enough to allow the urinometer to float freely without touching the sides. All bubbles must be removed from the surface by means of bibulous paper. The urinometer should be wiped clean and placed floating in the centre of the jar. The eye is then placed level with the surface of the urine, and the division of the scale to which the latter reaches read off. Care must be taken to read the level of the true surface of the urine, not the edge of the rim which heaps itself up around the shaft of the urinometer.

If only a small specimen of the urine be obtainable it may be necessary either to use "specific gravity beads," or else to add water to it in order to get enough fluid to float the urinometer. The specific gravity found is then multiplied by the necessary figure according to the degree of dilution.

Normal urine has a specific gravity varying from 1015 to 1025. If very concentrated, the specific gravity may rise to 1035 even in health. During the first month of life the specific gravity varies between 1001 and 1005, but by the second year it has reached 1026 or 1030, and in older children the urine tends to be rather more concentrated than that of adults.

The gravity is greatly increased by cooling. If, for example, it be 1020 when passed, it will rise to about 1025 when the urine has cooled to the tempera-

ture of the room. This should be specially borne in mind in insurance work.

In normal urine the specific gravity is in direct proportion to the amount of urea present. An abundant urine of *low* specific gravity is suggestive either of diabetes insipidus or of chronic renal disease. An abundant urine of *high* specific gravity is characteristic of diabetes mellitus. In the latter condition the specific gravity may reach 1075; in most cases, however, it is between 1040 and 1045. In diabetes insipidus, on the other hand, the specific gravity may fall to nearly that of distilled water.

The presence of albumin in the urine does not materially affect its specific gravity. It should also be borne in mind that the urinometer is entirely unaffected by the presence of merely suspended substances.

Estimation of the amount of solids.—This may be done roughly by multiplying the last two figures of the specific gravity taken at 15° C. by 2·33. The result is the number of grammes of solids in 1 litre of the urine, or the number of grains of solids in 1,000 fluid grains of urine; e.g. if the specific gravity of a urine is 1020, it contains $20 \times 2{\cdot}33 = 46{\cdot}6$ grm. of solids in every litre, or 4·6 per cent. This multiplied by 4·375 gives gr. per ounce—in this case 20·1. The average daily output of solids in the urine is about 60 to 70 grm. (2–2½ oz.). The above mode of calculation is not applicable to urine containing abnormal ingredients, e.g. sugar or albumin.

6. **Cryoscopy.**—The object of this procedure is to determine the molecular concentration of the urine by observing the depression of its freezing-point below that of distilled water. The physical laws on which cryoscopy depends may be summarized as follows: (1) Every substance which is soluble in a fluid that can be frozen, when so dissolved lowers its freezing-point, and does so proportionately to the degree of

concentration of the solution, provided always that the substance remains simply in solution and neither combines with the fluid nor is altered in constitution by it. (2) Different substances dissolved in a given fluid in quantities proportional to their molecular weights all lower the freezing-point to the same extent. Thus, 58 grm. of NaCl, with an approximate molecular weight of 58, lower the freezing-point of a given quantity of water to the same extent as 101 grm. of KNO_3, whose molecular weight is 101. (3) When several substances are successively dissolved in the same solution, the lowering of the freezing-point corresponds to the sum of the amounts of depression due to each of the substances taken by itself. (4) In the case of aqueous solutions, which alone are found in the fluids of the body, experiment has determined that one mol* of any substance dissolved in 100 grm. of water lowers the freezing-point of the solution by 18·6° C.

The only serious complication results from the fact that substances which are capable of electrolysis are partly found in solution not in their original form, but with their molecules broken up into two portions, known as *ions* in consequence of their conduct when a current of electricity is passed through the solution. The proportion of molecules so divided becomes greater the more the solution is diluted. Each of these two fragments of the original molecule behaves, so far as the lowering of the freezing-point is concerned, as if it were a complete molecule, and hence in the case of such substances the lowering of the freezing-point is relatively greater than in the case of those whose molecule is not split into ions by the process of solution. It is possible by means of electrical methods to determine the proportion of ions which are present, but the details of the process are beyond the scope of this manual.

Several forms of apparatus are employed for determining the reduction of the freezing-point. Those most applicable to clinical purposes are Beckmann's, Claude and Balthazard's, and Zikel's. The first, which is most commonly used, consists of the following parts : An outer cylindrical glass jar contains the freezing mixture. This jar is covered by a lid with a large central aperture, and two smaller openings permit of a stirrer and a thermometer being introduced into the freezing mixture. The central aperture admits of a wide tube closed below and with a ring of cork in its open upper end, into which is fitted the

* The word "mol" has been suggested by Ostwald as a short equivalent for gramme-molecule, i.e. one gramme multiplied by the molecular weight of the substance. Thus one mol of NaCl = 58 grm., and one mol of KNO_3 = 101 grm.

freezing vessel, provided with a side tube, and closed above by a cork, through which pass a stirrer of platinum wire and a very delicate thermometer, graduated to hundredths of a degree. As this thermometer has not a fixed zero, one must first determine the reading which it gives for the freezing-point of pure water in the following manner: Into the freezing tube so much distilled water is introduced as will rather more than submerge the bulb of the thermometer. The outer cylinder is then filled with a freezing mixture, which is so composed that it is not greatly colder than the freezing-point to be determined—(for clinical urine estimations one aims at a temperature not lower than $-5°$ to $-7°$, but if very accurate results are required, a temperature not more than a few tenths of a degree lower than the freezing-point of the solution which is under examination) —and in which by regular stirring a uniform temperature is maintained. The freezing tube is at first placed directly in the freezing mixture, and when the temperature of its contents has nearly reached its freezing-point, it is placed in the wide tube already described as fixed in the cover of the outer cylinder, and, protected by the air-mantle which now separates it from the freezing mixture, it slowly cools till it reaches a temperature slightly below the true freezing-point. During this process the platinum stirrer must be carefully used to maintain a uniform temperature throughout the water. When the freezing-point has been slightly overstepped, the water suddenly freezes; this generally takes place spontaneously, but if unduly delayed can be promoted by dropping through the side tube a tiny fragment of ice. The process of freezing leads to the liberation of latent heat, with the result that the thermometer rises to the true freezing-point, and remains there for several minutes whilst the process of freezing is completed. This temperature is noted as that at which pure water freezes; the water is then replaced by an equal quantity of urine to be examined, and the process is repeated. By subtracting the temperature of the freezing-point of the urine from that of the pure water, one obtains the depression of freezing-point due to substances dissolved in the urine, and can thus calculate the molecular concentration of the fluid, for if $\triangle$ represent the depression observed, then, since the depression due to 1 mol dissolved in 100 grm. of water is 18·6° C., we know that the specimen under investigation contains $\frac{\triangle}{18{\cdot}6}$ mols in the same volume.

If urine be collected separately from each ureter, one is able to determine the relative concentration of the fluid excreted by each kidney—a most valuable piece of information.

The molecular concentration of healthy urine fluctuates within rather wide limits, the freezing-point varying from about $-1{\cdot}3°$ to $-2{\cdot}2°$ C., and in some cases even more widely.

Blood is examined in exactly the same manner as urine, but more care must be taken in obtaining a very accurate reading of the thermometer, as the difference in freezing-point between normal and pathological states is much smaller. It is also important to secure a sufficient quantity of blood, as the bulb of the thermometer must be wholly immersed. Several instruments have been constructed so that the capacity of the freezing tube is smaller, but the accuracy of the readings has been found to suffer.

The normal depression of the freezing-point of blood is $0{\cdot}56°$; a depression greater than $0{\cdot}58°$ may usually be held to justify the opinion that the kidneys are failing to perform their function adequately, and that *both* of them are affected.

Since these temperatures are not much below that of the freezing-point of pure water, and since ordinary mixtures of salt and ice produce much lower temperatures, it is obvious that if one desires very accurate readings some other freezing mixture is to be preferred. The most suitable are those known as cryohydrates, where a finely powdered mixture of ice and a salt are employed, an excess of the salt being present. Cohen supplies a table of the most useful, from which the following are taken :—

Glauber's salt gives a constant temperature of				$-0{\cdot}7$
Potassium bichromate	,,	,,	,,	$-1{\cdot}0$
Potassium sulphate	,,	,,	,,	$-1{\cdot}5$
Potassium nitrate	,,	,,	,,	$-3{\cdot}0$
Zinc sulphate	,,	,,	,,	$-5{\cdot}0$
Barium chloride	,,	,,	,,	$-7{\cdot}0$

The method of using the other forms of apparatus which have been mentioned is very similar, but in Claude and Balthazard's instrument,* instead of a freezing mixture, the temperature of the outer chamber is reduced by the evaporation of ether or bisulphide of carbon. Zikel's pektoscope † resembles Beckmann's instrument more closely, but is in some ways more convenient for clinical work. The thermometer possesses a smaller range, but is more readily adjusted, and the tube which surrounds the freezing tube is made open below, so that the mixture in the outer jar may rise round the freezing tube until a temperature near the freezing-point is reached, when

* Made by Berlemont, Paris.

† Made by Gebr. Muencke, Karlstr. 18A, Berlin.

by means of a small indiarubber bulb it may be expelled, and the process of freezing completed with an air-mantle.

7. **Naked-eye characters of the deposit.**—When voided, normal urine is perfectly clear and transparent. After it has stood for some time there appears in it a deposit of "**mucus.**" This forms a woolly-looking cloud which usually settles to the bottom of the glass, but, if the urine be of high specific gravity, may be in the middle of the glass or even at the top. Opinion differs as to whether this "mucus" is a gluco-protein (mucin) or a nucleo-protein. It is possibly both, the latter being in excess.

If traces of blood be present in the urine the cloud of "mucus" has often a brownish tint.

The normal urinary ingredients, which may separate out in the form of a deposit visible to the naked eye, are—earthy phosphates, urates, and free uric acid.

Phosphates.—The phosphates of calcium and magnesium separate out if the urine be neutral or alkaline. They form a colourless deposit. It can be recognized by the fact that if a little of it be transferred by a pipette to a test tube, and some dilute acetic acid added, the deposit dissolves. A deposit of *pus* is apt to be mistaken for one of phosphates. They may be distinguished by moving the glass gently from side to side. It will then be found that a deposit of phosphates is more flocculent and less compact than one of pus, and the surface layers of it are easily detached on shaking, and float up; this does not happen in the case of pus. Acetic acid, also, does not dissolve pus, while the addition of caustic alkali renders it ropy. If the urine be acid, however, and its reaction has not been tested, a deposit of pus cannot be distinguished with the naked eye from a deposit of phosphates. It should also be borne in mind that deposits of phosphates and pus often occur together.

Urates.—The urates of sodium, potassium, and ammonium may form a deposit if the urine be concentrated or highly acid. They may appear, even in health, when the urine cools. Owing to their affinity for the urinary pigments the deposit is usually coloured, being commonly red, or like terra-cotta, forming what is known as the "*brick-dust*" *deposit.* If the urinary pigment be scanty, however, the deposit may be merely yellowish, or even colourless. Deposits of urates can always be recognized by the fact that they disappear rapidly on heating the urine. The heating ought to be accomplished gradually, because the urine may also contain albumin, which, if the urine be rapidly heated, may be coagulated before the deposit of urates has all had time to clear up, and thus confusion may arise. Acetic acid does not dissolve a deposit of urates. On the other hand, strong mineral acids, such as nitric acid, dissolve the deposit at once, with the production of effervescence.

Acid urate of soda is a rare deposit. It occurs in acid urines. It forms a yellowish, granular, sandy-looking sediment. It does not dissolve readily on heating.

Acid urate of ammonia forms a very similar deposit, but it occurs in ammoniacal urines, and is therefore mixed up with a deposit of phosphates.

Uric acid.—This may form a scanty deposit visible to the naked eye. The deposit occurs in the form of crystalline grains of a darkish-brown colour, and is therefore known as the "*cayenne-pepper*" *deposit.* When in doubt use the microscope.

The sulphates practically never form urinary deposits. **Oxalates** do, but the deposit is generally scanty, mixed up with the cloud of mucus, and not easy to recognize with the naked eye. Sometimes, however, the crystals form a glistening layer above

the mucous deposit. We have already spoken of the occurrence of fibrin, and the other abnormal ingredients which may be deposited will be described in the section on the microscopical examination of the urine.

We would warn the reader against the common mistake of supposing that a substance is necessarily being excreted in excess when it appears in the urine in the form of a deposit. This, of course, is not necessarily the case at all. Thus the occurrence of a "cayenne-pepper" deposit does not necessarily mean that the patient is excreting an excess of uric acid. It may merely be due to the fact that the conditions which normally cause the uric acid to be in solution have become modified. The urine may be abnormally acid, for example, or it may be deficient in colouring matter or in salts, all of which conditions tend to lessen the solubility of uric acid, and to favour its deposition in the form of crystals. Similarly in the case of a deposit of phosphates. That does not mean that more phosphoric acid is being eliminated; it merely indicates that the urine has become alkaline.

II. CHEMICAL EXAMINATION OF THE URINE

1. Reaction

This is taken with litmus paper. The urine is usually acid in reaction. This is not due to the presence of free acid, but to acid salts—chiefly the acid phosphate of sodium (NaH_2PO_4). Sometimes the reaction is blue to red litmus paper, and red to blue litmus paper. This amphoteric reaction is due to the presence of large quantities of the disodic phosphate (Na_2HPO_4) in addition to the acid salt. It has no clinical significance. The urine may be normally alkaline after meals. This is sometimes known as the "*alkaline tide.*" It reaches its acme three hours after the taking of a meal. It is chiefly due to the disodic

phosphate replacing the acid salt. Alkalinity of the urine may be due to ammonia. This can be detected by its smell, also by the fact that if the red litmus paper which has been turned blue be heated, the red colour is restored, owing to the ammonia being driven off.

For clinical purposes **the total acidity and the "ammonia"** in the urine may be determined as follows :—

Place in a flask about 15 grm. of powdered potassium oxalate (neutral to phenol-phthalein). (This is to precipitate the calcium in the urine, otherwise the formation of calcium phosphate interferes with the end point of the reaction.) Add 25 c.c. of urine, an equal quantity of distilled water, and 10 drops of 1 per cent. alcoholic phenol-phthalein. Mix well. After one minute run in $\frac{N}{10}$NaHO from a burette until a faint pink colour results. Read the burette ; this gives the total acidity in terms of $\frac{N}{10}$NaHO. Now to 5 c.c. of formalin in a beaker add 5 c.c. of water and a few drops of phenol-phthalein ; then run in $\frac{N}{10}$NaHO till a faint pink colour again appears. Add this mixture to the neutralized urine in the flask. The pink colour disappears. Run in $\frac{N}{10}$NaHO until it returns, and read burette. This reading gives the amount of ammonia in terms of $\frac{N}{10}$NaHO. By the addition of the neutral formalin the ammonia in the urine is combined with it to form a neutral compound, urotropine. The previously neutralized acid is thus liberated and determined by the second fixation. The amount (in grammes) of N present as ammonia is determined by multiplying this reading by 0·0014. This reading is in reality a trifle high, but is sufficiently accurate for clinical purposes. The error is due to the fact that by this means the amount of the amino-acids present in the urine is also determined. Except in cases such as cystinuria, the amount of these acids is so small as to be negligible. For very accurate work, Folin's method may be employed ; an advanced physiological-chemistry manual should be consulted.

2. Examination of the Urine for its Normal Non-nitrogenous Constituents

(1) **Chlorides.**—Chloride of sodium is the chief inorganic constituent of normal urine. Small quantities of the potassium salt also occur.

Qualitative test for their presence.—Filter the urine if not already clear. If albumin is present, remove it by boiling. Add to ½ in. of the urine in a test tube a few drops of nitric acid (be sure that the acid used is quite pure and free from HCl), and then as much of a 3 per cent. solution of nitrate of silver as there is of urine. If the normal amount of chlorides be present, an abundant curdy precipitate appears at once. If the chlorides be diminished, the solution merely becomes milky. If a mere trace of them be present, the solution is opalescent; and if they be altogether absent, it remains quite clear.

The use of the nitric acid is to prevent the precipitation of phosphate of silver.

Quantitative estimation.—For ordinary clinical use *Mohr's method* may be employed. One proceeds as follows: Place 10 c.c. of the urine, freed if necessary from albumin, in a beaker, and mixed with 50 c.c. of distilled water. Add three drops of a solution of neutral chromate of potassium (1 in 20) and a pinch of calcium carbonate. The use of the latter is to neutralize any free acid that may be present. Fill a burette with standard solution of nitrate of silver (Appendix, 9). Run the silver solution into the beaker, stirring all the time. A precipitate of chloride of silver falls out. Whenever the least trace of a pink colour appears, stop. This can be best appreciated by allowing the precipitate to settle, which it very quickly does. If the sediment be in the least flesh-coloured, enough silver solution has been added. The appearance of the pink colour indicates that the silver has united with all the chlorides present and has begun to form chromate of silver with the potassium chromate.

Calculation.—One c.c. should be deducted from the total number of c.c. of silver nitrate used. The reason for this is that there exist in urine, besides chlorides, other substances with which the silver unites more readily than it does with the chromate. Roughly, the deduction of 1 c.c. is sufficient to allow for these. Every remaining c.c. of the solution used is equivalent to 10 mg. of sodium chloride. Suppose 11 c.c. to have been used in all, deducting 1 c.c. there is left 10 c.c. This is equivalent

to 100 mg. of sodium chloride, which will be the quantity of chlorides in the amount (10 c.c.) of urine used. If 1,500 c.c. was the amount of urine, in twenty-four hours it will contain 15 grm. of sodium chloride.

For the accurate estimation of *small quantities* of chlorides in the urine the method of *Volhard* should be employed, but for a description of it special works must be consulted.

About 12 grm. represents the average daily excretion of chlorides in health. The chief cause of physiological variation is the nature of the diet. Pathologically, chlorides are found to be diminished in all febrile affections with the exception of malaria. In the latter disease the chlorides are increased during the febrile period, diminished in the apyrexial intervals. In acute croupous pneumonia the chlorides are more markedly diminished than in any other fever; they may indeed disappear entirely. We regard their behaviour as of great diagnostic value. In no other disease, except perhaps typhus and rheumatic fevers, does such a notable diminution ever occur. In the diagnosis of pneumonia from empyema and pleurisy the test is of special help. The chlorides are increased after the crisis in pneumonia—the increase, however, not usually manifesting itself till the third day after fever has ceased—and also in cases where the rapid absorption of a large exudation is taking place.

(2) **Phosphates.**—Phosphoric acid occurs in the urine in two chief forms of combination. Combined with potassium, sodium, and ammonium, it constitutes the alkaline phosphates; with calcium and magnesium, the earthy phosphates. Three-fourths of the total phosphoric acid is combined with the alkalis, and only one-fourth with the earths. The alkaline phosphates, being readily soluble, never form a deposit. The earthy phosphates are insoluble in an alkaline medium, hence they are precipitated when the urine loses its

acid reaction. This precipitation is aided by the action of heat. The heat probably acts by driving off carbonic acid. Hence if a urine, the reaction of which is not acid, be heated, a cloud of earthy phosphates may appear. This is distinguished from albumin by its ready disappearance on adding a few drops of acetic acid.

Qualitative tests for phosphoric acid in urine.—(i.) Add ammonia. A white crystalline precipitate, increasing on standing, shows the presence of the phosphates of the alkaline earths (Ca, Mg), the so-called *earthy* phosphates. The *alkaline* phosphates of Na and K still remain in solution.

(ii.) To 10 c.c. of urine add half its volume of nitric acid, a few drops of ammonium molybdate, and boil. A yellow precipitate is given by both forms of phosphates.

Quantitative estimation.—Fill a burette with standard solution of uranium nitrate (Appendix, 10). Measure 50 c.c. of the urine to be examined into a porcelain dish or a medium-sized beaker. (If the urine be very concentrated, 20 c.c. of it will be sufficient.) Add to the urine 5 c.c. of an acetic acid solution of acetate of soda (Appendix, 11). If only 20 c.c. urine were taken, add 2 c.c. of the solution. Add also a few drops of tincture of cochineal to serve as an indicator. Heat the urine on a water-bath. If the latter be not obtainable, use a tripod covered with wire gauze. The urine should be heated to a temperature *just short* of boiling. When heated, run in the uranium solution, stirring all the while. As a rule, 16 c.c. may be run in right away. A precipitate of uranium phosphate falls down. Continue cautiously to add the uranium solution until the precipitate has *a slight but persistent greenish tint*. This is a sign that the uranium has united with all the phosphoric acid present and is beginning to react with the cochineal. The presence of albumin or of sugar does not affect the reaction. The use of the acetate of soda in the above process is to unite with the nitric acid liberated by the union of the uranium with the phosphoric acid.

Calculation.—Suppose 20 c.c. uranium solution have been required. The solution was made of such a strength that each c.c. = 5 mg. of phosphoric anhydride; 20 c.c. are therefore equivalent to 0·1 grm. of P_2O_5, and that is the amount in 50 c.c. of urine. If the patient is passing 1,500 c.c. of urine in twenty-four hours, his daily excretion of P_2O_5 will be 3 grm.

Normally, 2–3 grm. of phosphoric anhydride are excreted daily. Physiological variations depend chiefly upon the food. The phosphates are often considerably diminished in renal disease, but not, apparently, out of proportion to the other solids of the urine. They are said to be increased in wasting diseases of the nervous system. Their behaviour in fever is inconstant.

(3) **Sulphates.**—Sulphuric acid occurs in the urine in combination with sodium and potassium (inorganic sulphates), and with cresol, phenol, indol, skatol, pyrocatechin, etc. (organic sulphates). The former are ten to twenty times more abundant than the latter.

Sulphur is also present as "neutral sulphur," that is, the sulphur present in organic bodies of which it is an integral part of the molecule, e.g. cystin.

Test for inorganic sulphates.—Add to 10 c.c. of urine a few drops of hydrochloric acid and one-third of its volume of 10 per cent. barium chloride solution. If the normal amount of sulphates be present, an opaque milkiness will be found to develop. If the precipitate be thick and creamy, the sulphates are in excess; if a mere opalescence appear, they are diminished.

About $2\frac{1}{2}$–$3\frac{1}{2}$ grm. of sulphuric acid (SO_3) are excreted daily. The exact determination of the total sulphates, and of the proportion of inorganic to organic, is a gravimetric process unsuited for ordinary clinical work.

An approximate notion, however, of the proportion of **organic sulphates** present may be obtained by the following procedure :—

Add to the urine an equal volume of alkaline barium chloride solution (2 parts of baryta water to 1 part of barium chloride solution). This precipitates the *inorganic* sulphates along with phosphates. Filter. Render the filtrate pretty strongly acid with hydrochloric acid, and heat almost to boiling. The *organic* sulphates are thus decomposed and

thrown down in the inorganic form. Normally they should form merely a white cloud. If the precipitate be at all dense the proportion of organic sulphate is in excess.

The total sulphates are increased by an increase in the diet, and in fever. The amount of sulphuric acid excreted in organic combination is increased when a larger quantity than usual of the aromatic substances with which it is combined enters the circulation. This occurs when phenol and allied substances are given as drugs, or when the production of such substances in the body is increased, as it is whenever putrefactive processes are going on. Thus the amount of organic sulphates is increased in cases where putrid abscesses have formed, or where there is retention of the intestinal contents. But the ethereal sulphates of the urine are not, as has been supposed, a direct measure of such putrefactive processes, since the proportion of inorganic and organic sulphates and neutral sulphur varies with the amount of nitrogen in the diet.

(4) **Oxalates.**—Oxalic acid occurs in the urine, combined with calcium. The salt is usually kept in solution by the acid phosphate of sodium present in the urine. It is found as a precipitate, however, in about one urine out of every three. This is due to the absence of a sufficient amount of the phosphate of soda to keep it in solution. It does not necessarily mean that the excretion of oxalic acid is increased, although it is true that the more oxalic acid there is present, the greater is the tendency for it to be precipitated. About 0·017 grm. is the average amount of oxalic acid excreted daily. It is mainly derived from the food. It is increased after the taking of certain vegetables, especially cabbage, spinach, and rhubarb. The so-called "oxaluria" seems to be merely a variety of acid dyspepsia.

3. Examination of the Urine for its Normal Nitrogenous Constituents

Of the total amount of nitrogen in the urine—

84–87 %	is in the form of	urea;	
2– 5 %	,,	,,	ammonia compounds;
1– 3 %	,,	,,	uric acid;
7–10 %	,,	,,	"extractives" (including purin bases).

These vary in percentage according to the intake of nitrogen in the diet; the relative percentage of urea falling markedly when the nitrogen intake is much reduced.

Estimation of total nitrogen by Kjeldahl's method (modified).—Measure out 5 c.c. of urine with a pipette and place it in a Kjeldahl's flask of about 150 c.c. capacity; add to it 15 c.c. of pure sulphuric acid and a crystal of pure sulphate of copper about the size of a split pea; heat on net till the mixture is colourless or pale green. This takes about half an hour or less; violent boiling should be avoided. Allow to cool, then dilute with 50 c.c. of distilled water, again allow to cool, transfer to a distillation flask of about 700 c.c. capacity, and add enough 23 per cent. solution of caustic soda to render the mixture almost neutral; add also a pinch of talc to prevent bumping. When cool, add more soda (till the fluid is deep blue in colour), and close at once with a stopper connected to the condensing tube of the distillation apparatus; measure into a flask 100 c.c. of a decinormal solution of oxalic acid, and let the lower end of the condensing tube just dip below the surface of this solution; then boil the blue fluid. The nitrogen passes off in the form of ammonia and is fixed by the oxalic acid. When the fluid which drops from the condenser has no longer an alkaline reaction, stop the process by removing the stopper from the flask. Care must be taken that at the end no alkali comes over from the flask containing the strongly alkaline fluid, as is rather apt to occur should there be bumping. Titrate the 100 c.c. of decinormal oxalic acid with a decinormal soda solution, using methyl orange or neutral lacmoid as indicator. Every c.c. of soda less than a hundred used represents 0·0014 grm. of nitrogen.

Example.—Suppose that on titrating the oxalic acid solution with decinormal soda the neutral point is reached when 60 c.c. of the latter have been added; the remaining 40 c.c.

of the decinormal oxalic acid taken must therefore have been neutralized by the ammonia derived from the nitrogen in the 5 c.c. of urine, therefore the 5 c.c. of urine contain 0·0014 × 40 grm. of nitrogen, or 0·056 grm. If the total amount of urine passed in twenty-four hours be 1,500 c.c., this will contain 16·8 grm. of nitrogen.

About 15–20 grm. of nitrogen are excreted daily in the urine of a healthy adult on ordinary diet. A knowledge of the quantity excreted in disease is not of much value *unless one has some idea of the amount of nitrogen in the diet.* It must be remembered also that normally 1–2 grm. of nitrogen appear in the fæces.

Urea ($CO(NH_2)_2$). **Qualitative test.**—Place a drop or two of the suspected fluid on a slide and add one drop of nitric acid; warm gently. On evaporation, rhombic or hexagonal crystals of nitrate of urea will be found if the latter body be present (Fig. 86).

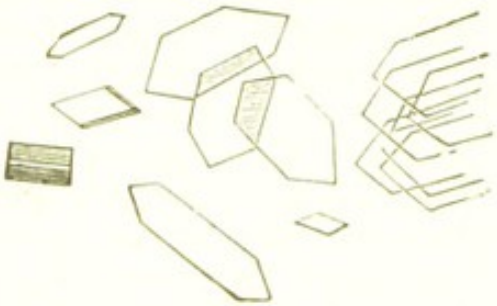


Fig. 86.—Urea nitrate.

Quantitative estimation. (*a*) *From the specific gravity.*—An approximate estimation of the amount of urea may be made by dividing the last two figures of the specific gravity by 10; e.g. if the specific gravity of a given urine be 1020, it contains (approximately) 2 per cent. of urea. This only holds good in the absence of sugar, or of much albumin, and if the patient be not very feverish.

(*b*) *From the amount of nitrogen given off on treating the urine with hypobromite of soda.*—This method, sufficiently accurate for clinical purposes, depends upon the fact that urea is decomposed by hypobromite of soda according to the following equation: $CO(NH_2)_2 + 3NaBrO = 3NaBr + N_2 + 2H_2O + CO_2$. It is found that under ordinary conditions 1 grm. of urea yields 371 c.c. of nitrogen; all that is necessary, therefore, is to remove the CO_2 by means of an alkali (caustic soda), and to measure the remaining volume of gas and to calculate from it the amount of urea which was contained in the quantity of urine taken. The various forms of apparatus employed in carrying out the process differ chiefly in the method adopted for catching and measuring the nitrogen given off.

The number of c.c. of nitrogen given off (from 5 c.c. of

urine) multiplied by 0·056 = grams of urea in 100 c.c. of urine, i.e. the percentage, and this multiplied by 4·375 = grains of urea in 1 oz. of urine.

The following are the chief forms of apparatus employed :—

i. *Dupré's.*—The apparatus consists of a cylinder filled with water, into which dips a burette open at the lower end, and connected above with a T-piece attached by one limb to a short rubber tube provided with a clip, and by the other connected with a small bottle closed with a rubber stopper; 25 c.c. of hypobromite solution are measured into the bottle, and 5 c.c. of urine into the little tube provided for it, and the latter is then lowered into the bottle and leant up against its side. The stopper is inserted in the bottle, and the level of the water, both in the cylinder and the burette, is adjusted till it is opposite the zero mark on the burette. This is done by raising or lowering the burette, whilst the clip at the top is kept open. When the water is level the clip is closed, the urine and hypobromite are mixed by inverting the bottle, and the gas is collected. After a quarter of an hour, the burette is raised till the water in it is again on a level with that in the cylinder, and the amount of gas evolved is read off. The burette is graduated in lines which show the percentage of urea, and by multiplying these by 4·375 one arrives at the number of grains per ounce.

ii. *Gerrard's ureometer* (Fig. 87).—This consists of a graduated glass cylinder closed at the upper end by a rubber stopper. Through the stopper there passes a T-tube. One limb of this tube is closed by a clip or stopcock, the other is connected to a piece of rubber tubing. The other end of the rubber tubing terminates in a piece of glass tube, which is inserted into the rubber stopper of a wide-mouthed flask of about 6 oz. capacity. From the lower end of the graduated cylinder another rubber tube passes to a short wide glass tube open at its upper end. The object of this tube is to act as a reservoir of water. It can be slipped up and down upon the cylinder by means of a metal ring.

How to use the apparatus.—Place in the glass flask 25 c.c. of hypobromite solution (Appendix, 12). An excess of hypobromite does no harm—one must merely be sure that enough is taken to decompose all the urea likely to be found in the urine. Measure 5 c.c. of urine into the small glass tube provided for the purpose. If the urine contain albumin, the latter must be first removed. This is best done by taking a definite quantity of urine—say 50 c.c.—adding to it a drop or two of acetic acid, and boiling for a couple of minutes. Filter and make up to its original volume.

The small tube containing the urine must now be lowered into the flask. This is best done by inserting the point of the little finger—not too tightly—into the mouth of the tube. The latter must then be propped up against the upper surface of the flask so as to prevent the hypobromite solution from

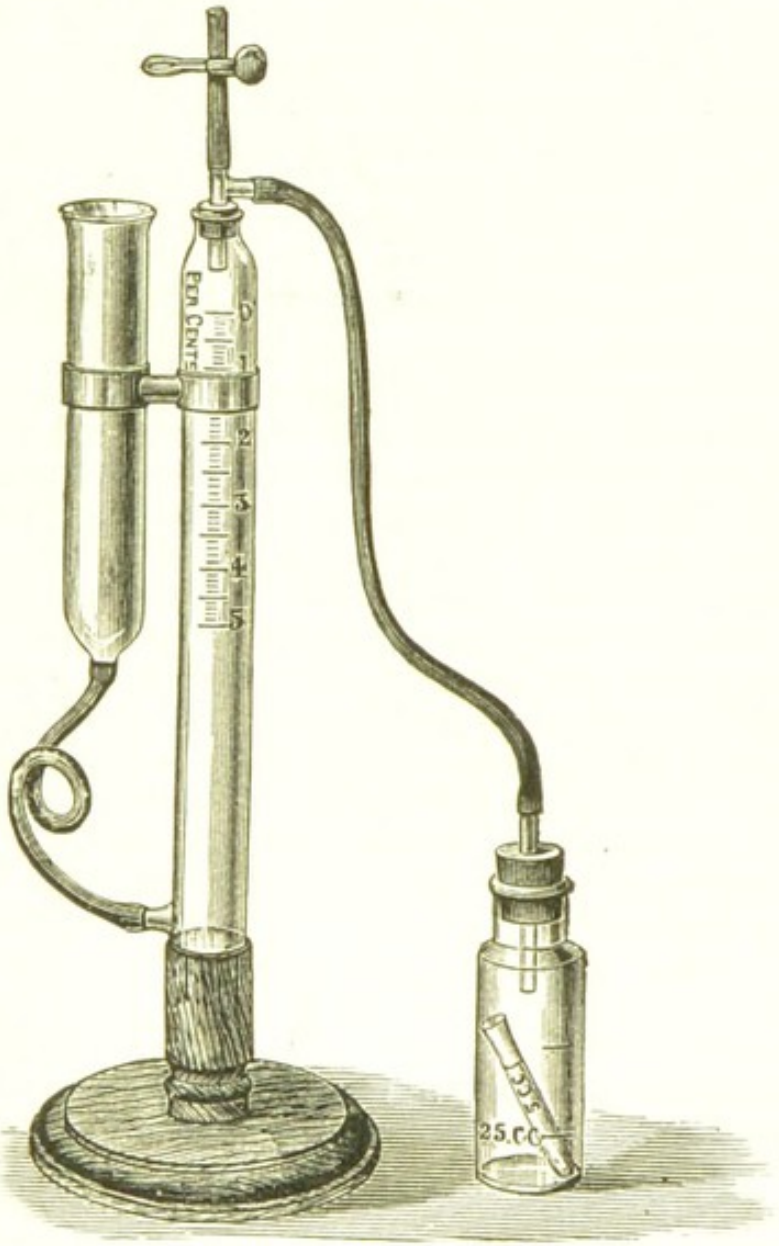


Fig. 87.—Gerrard's ureometer.

mixing with the urine. The reservoir of the graduated cylinder must now be filled with water. The stopper is then tightly inserted into the mouth of the flask. The clip or stopcock must now be opened, and the reservoir raised until the water inside the cylinder stands at the zero mark and is level with that in the reservoir. The water must also be very low down in the latter, else there will be an overflow subsequently.

Now close the stopcock and gently tilt the flask so as to allow the urine and hypobromite solution to mix. Great effervescence ensues, and the nitrogen liberated enters the cylinder and drives water out of it up into the reservoir. Wait for ten minutes to allow cooling to take place. Then lower the reservoir until the water in it and the cylinder are again level, and read off the amount of gas in the latter. The cylinder is graduated in percentage of urea. To get the number of grains per ounce, multiply this by 4·375.

In normal urine only 92 per cent. of the nitrogen of the urea is given off. If sugar be present the yield is for some reason much larger, amounting to about 99 per cent. In Gerrard's instrument the scale is constructed for normal urine. In cases of diabetes, therefore, it is necessary to correct one's result by multiplying the figure obtained by $\frac{92}{99}$, i.e. by 0·93.

iii. *Ureometer of Doremus.**—This is a very simple and cheap form of apparatus, devised by Dr. Chas. Doremus, of New York. Modifications of it are sold by Southall (Birmingham) and Cooper (London). It consists of a bent tube with a long limb closed at its upper end, and a short limb which expands into a wide bulb with an open mouth.

In using the instrument, it must first be filled with hypobromite solution. Hold the tube vertically, and pour in the solution just short of overflowing. Then gently incline the instrument so that the solution fills the long limb as far as the mark near the bend, and no air bubbles are present. A little water should be added to fill the rest of the bend and the lower part of the bulb. The instrument may now be fixed in its stand.

One c.c. of urine has to be measured out with the pipette provided. This is where the difficulty in using the instrument comes in. In order to accomplish it successfully, proceed as follows: Slip the rubber nipple high up upon the pipette. Compress the nipple, and immerse the point of the pipette just below the surface of the urine. Then allow the nipple to expand fully. Urine will be drawn up beyond the mark. Slide the nipple bodily down with the finger and thumb over the rimmed end of the pipette. This displaces the urine. Continue the displacement till the mark is reached. The pipette will now keep itself charged with exactly 1 c.c. of urine. Wipe the outer surface of the pipette, and insinuate the end of it as far as it will go into the long limb of the

* Also known as Southall's or Thursfield's ureometer. A modification of this apparatus by Hinds has many advantages over the original.

apparatus. Now compress the nipple so as to squeeze out all the urine, and withdraw the pipette, keeping up the compression until it is out of the liquid.*

In about a quarter of an hour the amount of nitrogen which has collected in the long limb may be read off. The instrument is provided with an English scale representing grains per ounce, and also with a metrical scale. The large divisions on the latter represent centigrams of urea per c.c. of urine. The scale between is subdivided into tenths. Suppose the reading to be 0·025. This means 0·025 grm. of urea in 1 c.c. of urine, and multiplying by 100 = 2·5 per cent. If more than 3 per cent. of urea be present the urine should be diluted with an equal volume of water and the result multiplied by 2. Albumin, if present, should always be removed beforehand.

About 450 gr. (25–40 grm.) of urea are excreted daily in health. This is about 2 per cent., or 9 gr. per ounce. It is increased when much food or water is taken; also in fevers, in diabetes, and in poisoning by phosphorus or arsenic. It diminishes under diminished diet, in some severe diseases of the liver (because less is formed), and in some conditions of the kidney. *No calculation of the amount of urea which a patient is passing is of much use unless the amount of nitrogen in his diet is also taken into account.*

Uric acid ($C_5H_4N_4O_3$) occurs in the urine in combination with alkalis. Being a dibasic acid, it forms two classes of salts, the normal urates ($Na_2\bar{U}$) and the acid urates ($NaH\bar{U}$). Under certain conditions uric acid becomes free in the urine, and separates out as a crystalline deposit ("cayenne-pepper" deposit). Acid urates may also separate out in a crystalline form. Both forms of separation, when occurring inside the urinary passages, lead to the disease known as "gravel" or to stone formation. The

* The pipette may be provided with a piston instead of a nipple, or one can extemporize a piston by slipping over the end of the pipette a piece of thick-walled rubber tubing in which a piece of glass rod, lubricated with glycerine, can be made to act as a piston and suck up the urine.

microscopic characters of these deposits will be described in another section. The conditions which favour the separation of uric acid and acid urates are (1) the presence of a large amount of uric acid in the urine ; (2) a high degree of acidity ; (3) the presence of little salts and of a small amount of pigment.

If present in large amounts, uric acid will reduce Fehling's solution, but not Nylander's solution (*see* Appendix, 14, 14*a*).

To demonstrate uric acid in the urine it is necessary to take about 100 c.c. of urine, add ammonia until it is alkaline, and saturate with ammonium chloride. Ammonium urate is thus precipitated. Filter off, dissolve in water, and perform the murexide test by adding one drop of nitric acid and evaporating very slowly, avoiding charring. When almost dry, add to the orange-coloured residue a small drop of ammonia. A purplish or rosy-red colour appears at the edge of the drop, often better seen after gently heating.

The action which takes place consists in the oxidation of the uric acid into alloxantine ($C_8H_6N_4O_8$)—the orange-coloured residue. On adding ammonia, purpurate of ammonia is formed ($NH_4C_8H_4N_5O_6$), and produces the purplish-red colour. The murexide test cannot be applied directly, as urine on heating with nitric acid yields a red pigment.

Quantitative estimation.—For clinical purposes this is best accomplished by a modification of Hopkins's method. It is based upon the insolubility of acid urate of ammonia in a saturated solution of ammonium sulphate. By saturating a given quantity of urine with sulphate of ammonium, all the uric acid separates out as acid urate of ammonium, which is collected ; the uric acid is then split off from it, and estimated either by weighing or by titration with permanganate of potash. Proceed as follows :—

(1) Saturate 100 c.c. of urine (preferably warmed to 45° C.) with powdered ammonium sulphate. About 30 grm. will be required. Saturation is complete so soon as a few crystals remain undissolved after vigorous stirring at short intervals. Even if these should redissolve as the temperature of the

mixture rises again after its initial depression, it does not matter.

(2) Render alkaline by adding a little ammonia.

(3) After it has stood for ½–1 hour, filter and wash the precipitate several times with saturated solution of ammonium sulphate.

(4) Wash the precipitate off the filter with a jet of hot water, add a pinch of carbonate of soda, and heat till the precipitate dissolves.

(5) Add distilled water to 100 c.c.

(6) Add 20 c.c. strong sulphuric acid.

(7) While hot, titrate with a $\frac{1}{20}$ normal solution of potass. permanganate—i.e. 1·578 grm. per litre.

(8) Stop when a pink colour lasting a few seconds has appeared. Subsequent disappearance of the colour is to be disregarded; it is sufficient that it should be visible for a second or two after stirring.

(9) Every c.c. of the permanganate used = 0·00375 grm. of uric acid.

The presence of bile pigment interferes with the titration with permanganate. In that case the uric acid must be estimated by weighing. The trouble in the above method is that the solution is so dense that it passes very slowly through the filter-paper. To obviate this difficulty, glass wool may be employed. (A little experience is necessary to enable one to pack the glass wool firmly enough to keep back the precipitate, but yet not so tightly as to render filtration very slow.)

The presence of albumin does not affect these methods. If there be a deposit of uric acid or of urates in the sample of urine, the whole should be thoroughly shaken up, the amount to be operated with measured off, and saturated as usual. Or a few drops of ammonia may be added and the urine warmed till the deposit dissolves. A deposit of phosphates may be neglected.

From 0·4 to 0·7 grm. (7–10 gr.) of uric acid is excreted daily. The amount is increased whenever a large destruction of nuclein is going on; thus in

myeloid leukæmia as much as 4 grm. may be excreted daily. It is also increased in acute fevers. It is diminished in chronic gout and after the administration of quinine.

We would again warn the reader against the common error of assuming that a deposit of urates or uric acid necessarily indicates an increased excretion of the latter.

Purin bases.—Among the chief products of the disintegration of nuclein are uric acid and some basic bodies which may be termed the "nuclein" or purin bases. These substances differ from uric acid in being basic. Hypothetical purin has the formula $C_5H_4N_4$; hypoxanthin is $C_5H_4N_4O$ (monoxypurin), xanthin (dioxypurin) is $C_5H_4N_4O_2$—i.e. one atom of oxygen less than in uric acid (trioxypurin). The other members are adenin ($C_5H_3N_4.NH_2$, aminopurin) and guanin ($C_5H_3N_4O.NH_2$, amino-oxypurin). These bases are sometimes spoken of along with uric acid under the term "**purin bodies.**" These purin bodies contain between them from 1 to 5 per cent. of the total nitrogen in the urine. The purin bases are increased, just as uric acid is, in conditions associated with increased destruction of nuclein—e.g. in leukæmia. A milk diet causes the bases to increase while the uric acid diminishes. There is no constancy in their behaviour in gout.

The purin bodies can be estimated in the urine with quite a sufficient degree of accuracy for clinical purposes by means of Walker Hall's **purinometer.***

The instrument consists of three parts—a close graduated tube; a stopcock, with a bore of the same diameter as the upper tube; a small glass reservoir of known cubical capacity. It is used in the following manner: With the stop at a right

* The instrument is supplied by Messrs. Gallenkamp, Sun Street, Finsbury, E.C.

angle to the tube, urine (first freed from albumin, if necessary, by *slight* acidification with acetic acid and boiling) is poured in up to 90 c.c. The stopcock is then turned parallel with the tube, and the lower chamber and the bore of the tap become filled with the urine; 20 c.c. of magnesia solution (Appendix, 17, No. 1) are then added, the instrument vigorously shaken, and the precipitate allowed to settle into the lower chamber. Immediately this has happened the tap is again turned at right angles. To the clear fluid remaining in the upper tube silver solution is now added (Appendix, 17, No. 2) to make the total fluid 100 c.c. The resultant precipitate consists of a mixture of silver-chloride and silver-purin. The apparatus is then inclined backwards and forwards till the precipitate is yellowish-white, which can be readily seen by comparison with the white phosphate precipitate in the lower tube. The instrument is now allowed to stand twenty-four hours, when the percentage of purins may be read off at the upper level of the precipitate. If the precipitate be not uniform the instrument must be again well shaken and the sediment allowed to settle afresh. The temperature of the room in which the estimations are made should be between 10° and 15° C., and the instrument kept away from the light during the estimation. It should be cleansed immediately after being used, and when not in use the stopper and the plug of the stopcock should be kept apart from the tube.

The only other nitrogenous constituents of normal urine which call for mention are creatinin and hippuric acid.

Creatinin ($C_4H_7N_3O$) is creatin minus water. It is not now supposed to be derived from either ingested or body-muscle creatin. Different views are held as to its seat of formation: according to one, it may be in the liver; according to another, in the muscles. Very little is really known as to the clinical significance of increased or decreased creatinin content of the urine. It is said to be increased in pneumonia, typhoid, and tetanus; decreased in leukæmia, anæmia, advanced degeneration of the kidneys and liver, muscular atrophy and paralysis. About 1 grm. of it is excreted daily. It is of importance as being

one of the constituents of normal urine, which is able to reduce cupric oxide.

Tests.—(i.) To 5 c.c. of urine add a few drops of saturated picric acid and make alkaline with a few drops of 10 per cent. caustic potash. A red colour results, which does not become opaque on heating (*see* p. 355).

(ii.) To 5 c.c. urine add 4–5 drops of dilute sodium nitroprusside, freshly prepared. Add, drop by drop, caustic potash; a red colour results, changing to yellow. On adding acetic acid and boiling, a greenish-blue colour results, depositing, after a time, a blue sediment. (Acetone gives a similar colour, but it does not change to yellow on standing, and with acetic acid it yields a purple colour.)

Hippuric acid ($C_9H_9NO_3$) occurs in the urine as hippurate of sodium. About $\frac{1}{2}$ grm. of it is excreted daily. This amount is increased by the taking of benzoic acid as a drug, or of fruits—e.g. mulberries and cranberries—which contain aromatic acids.

4. Abnormal Chemical Constituents of Urine

(1) proteins

Any or all of the proteins of blood plasma—serum albumin, serum globulin, and fibrinogen—may occur in the urine. In addition, one meets with the compound proteins—mucin and nucleo-protein—and with proteoses, both primary and secondary. It is doubtful whether true peptone ever occurs in the urine. It is true that two of these—mucin and nucleo-protein—are to be regarded as normal urinary constituents, being added to the renal secretion as it passes along the urinary passages, but in health they are present in such small amount that they may be neglected. It is also true that albumin occurs in the urine in perfectly healthy persons; the urine of healthy infants, for instance, often contains traces of it to the ordinary tests; generally, however, the amount present is so small as not to be detected by the tests

used for clinical purposes. Into the possible causes of this, and into the distinction between "functional" and "organic" albuminurias, we do not propose to enter. Chemical examination of the urine can merely show the presence in the urine of a protein; it cannot tell us to what its presence is due.

i. **Serum albumin and serum globulin in the urine ("albuminuria").**—These proteins may be found in the urine either together or separately. The former condition is the usual one, and constitutes what is ordinarily spoken of as "albuminuria." The relative proportion of each protein varies greatly in different cases, but usually serum albumin is present in larger amount than serum globulin. The variations in their relative amounts have no clinical significance. In what follows, the term "albumin" will be held to include also globulin, unless stated otherwise.

To separate albumin and globulin.—If one wishes to do this, the best plan is to render the urine slightly alkaline with ammonia, filter off phosphates if necessary, and add to 100 c.c. of the filtrate its own volume of a saturated neutral solution of ammonium sulphate. By the end of an hour, globulin, mixed perhaps with some mucin bodies, has separated out. Collect it on a weighed, dry, ash-free filter, wash with half-saturated ammonium sulphate solution, dry, and weigh. Then incinerate, and deduct the weight of the ash. The difference gives the amount of globulin present. If the total proteins have been estimated by *complete* saturation of another sample of urine with ammonium sulphate and treatment of the precipitate in the above way, the difference between the total proteins and globulin may be reckoned as albumin.

A simpler, but for clinical purposes sufficiently exact, method is that of Brandberg. It is thus described by Carstairs Douglas*: "Brandberg's method is based on the fact that in the cold nitric acid test the ring of albumin appears the quicker the more protein there is present. It has been found experimentally that if 0·0033 per cent. of albumin is present the ring appears in two to three minutes. By making a series of dilutions

* "Chemical and Microscopical Aids to Clinical Diagnosis" (Maclehose & Sons), 1899, p. 70.

of definite strength of an unknown urine an estimate may be made of the amount of albumin present in it. For example, if the urine shows the ring of coagulation when diluted 10 times, it contains 0·0033 × 10 = 0·033 per cent. of albumin; if when diluted 30-fold it shows the same, it must contain 0·0033 × 30 = 0·1 per cent., and so on. In estimating the amount present in an unknown specimen, it is necessary first of all to make certain dilutions of it, and Tappeiner recommends that, to begin with, dilutions of 10-fold, 30-fold, and say 150-fold, should be made. If now, after trying these specimens with the acid, it is found that the ring appears in less than two or three minutes in the former two, and in more than three minutes in the 150-fold dilution, the proper point will be reached at a dilution somewhere between 30-fold and 150-fold. We may find it, let us say, at a dilution of 80-fold; the percentage of albumin will then be 0·0033 × 80 = 0·26. In any case the amount of albumin per cent. is obtained by multiplying 0·0033 by the degree of dilution."

In carrying out the above procedure certain precautions must be observed :—

(*a*) The dilution of the urine must be made very exactly by the use of a burette.

(*b*) About $\frac{3}{4}$ in. of nitric acid should be used in each test, and the diluted urine run on to its surface very cautiously.

(*c*) Holding a watch in one's hand, one must know the exact time required for the appearance of a faint *but distinct* ring, and this time must fall between two or three minutes. The test tube should be held meantime against a dark surface.

Having estimated the total protein present by this method, one must saturate some of the fresh urine with magnesium sulphate, filter off the precipitated globulin, and make a fresh estimate by the same method in the filtrate. This gives the albumin alone, and the difference between this and the previous result indicates the amount of globulin. The presence of the magnesium sulphate does not seem materially to affect the result.

Tests for albumin in the urine.—A great many reactions have been proposed for this purpose, and much has been written on the subject. The fact seems to be that while in the majority of cases the presence of albumin or globulin in the urine can be demonstrated with perfect certainty by the application of one or two simple tests, yet every now and

then cases are met with in which some doubt exists. We believe that by carefully applying the following tests the presence of albumin and globulin can be clearly shown, even when present in very small amount. Before proceeding to apply any of the tests, it is a *sine qua non* that the urine should be *absolutely clear*. It may therefore be necessary to filter it. If it be acid, this can be proceeded with at once ; should it be alkaline, enough acetic acid should first be added to render it faintly acid. It may be necessary to filter more than once. Should the urine still not be clear, the turbidity is probably occasioned by the presence of bacteria. These can best be removed either by (1) simply shaking up the urine with powdered barium carbonate, and filtering, or (2) adding to the urine a little caustic soda till a precipitate of earthy phosphates appears. This carries down with it all bacterial débris, and on filtration the urine will now be found to be clear. It should then be slightly acidified with acetic acid, and the following tests proceeded with :—

(*a*) *Boil* an inch or so of the urine in a test tube. If it remains perfectly clear while the reaction is still acid, no albumin is present. Turbidity may be due to coagulated albumin or to the throwing down of earthy phosphates. Add a drop of nitric acid. Any turbidity which remains is due to the presence of albumin ; mucin and nucleo-protein are re-dissolved by the nitric acid.*

(*b*) *Heller's test.*—Place $\frac{1}{4}$ in. of pure nitric acid in a test tube. With the aid of a pipette allow some of the urine to flow on to its surface. If after standing for half a minute no opaque white ring appears at the junction of the two fluids, the urine may be regarded as free from albumin, for the test

* If, as sometimes happens, only a few drops of urine be obtainable, the test may be modified by dropping the urine into a test tube full of boiling water held against a dark background. If the urine be albuminous, each drop gives rise to an opalescent cloud. Phosphates give the same reaction, but the cloud disperses on the addition of acetic acid.

is capable of revealing the presence of 0·002 per cent. If a ring forms it may be due to albumin, nucleo-protein, or proteoses. A diffuse haze at the upper part of the fluid may be due to mucin. In the case of proteoses the ring disappears on heating, and reappears on cooling. (The method of distinguishing nucleo-protein and mucin will be considered later.) If the urine be very concentrated, nitrate of urea or acid urates may separate out, usually in the form of a cloud without sharp margins. This can at once be distinguished by the fact that previous dilution of the urine with twice its volume of normal salt solution prevents its appearance. The precipitate also disappears on heating, and shows small crystals on examination with the microscope. The presence of resinous bodies—e.g. balsams—in the urine may also cause the appearance of a white cloud, which, however, is at once dissolved on the addition to the urine of half its volume of alcohol (rectified spirit).

It will often be noticed that a brownish-red transparent ring appears at the junction of the nitric acid and the urine. This is due to the oxidation of the urinary indigogens and the production from them of pigments. It occurs, therefore, in urines which are rich in indigogens, and has, of course, nothing to do with albumin. Such urines will be more fully considered later.

Effervescence on the addition of nitric acid may be due to decomposition of urea with the liberation of CO_2 and nitrogen. This only occurs if the nitric acid used contains some nitrous acid also.

As a substitute for nitric acid in the above test, one can use *Spiegler's solution.* It consists of—

Perchloride of mercury . .	4 parts.
Tartaric acid	2 ,,
Glycerine	10 ,,
Water	100 ,,

It gives a distinct white ring if as little albumin as 1 in 350,000 be present, and may reveal the presence of albumin even in normal urine.

Another substitute, probably the most satisfactory of all tests for albumin, is a saturated solution of pure *salicyl-sulphonic acid.* It has the advantage over nitric acid of being non-corrosive, and therefore easily carried about. To perform the test, add a few drops of the saturated acid solution to 10 c.c. of urine. A white precipitate indicates, as before, albumin, nucleo-protein, or proteoses. In the case of

the latter it disappears on heating the solution, reappearing on cooling.

(*c*) *Ferrocyanide of potash test.*—Take 2 in. of urine in a test tube. Add 10 drops of a 5 per cent. solution of ferrocyanide of potash, and then render strongly acid with acetic acid. If the urine remains clear, no albumin is present. Turbidity may be due to albumin, proteose, or nucleo-protein. Proteose may be distinguished by the nitric or salicyl-sulphonic acid test; nucleo-protein by the fact that it is precipitated by acetic acid alone without the aid of ferrocyanide.

(*d*) *Picric acid test.*—(Use a saturated solution of pure picric acid, without the addition of citric acid, as in Esbach's solution.) Place 2 in. of the picric acid in a test tube. Allow the urine to drop into it from a pipette. If no cloud forms around the drops, the urine is albumin-free. A cloud may be due to albumin, proteoses, or peptone. The precipitate produced by the proteoses and peptone disappears on heating. Quinine also gives a precipitate with picric acid which disappears on heating.

It will be observed from what has been said above that it is comparatively easy to be sure of the absence of albumin—not so easy to be certain of its presence. The substance which is most apt to be mistaken for serum albumin in the urine is nucleo-protein. We have already indicated some methods of distinguishing between the two, and shall return to the subject later when we consider "nucleo-proteinuria."

Quantitative estimation of albumin.—This can be done with sufficient accuracy for clinical purposes by means of Esbach's albuminimeter. The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid. The instrument consists of a thick glass test tube, with graduations on it from 0 up to 7.

Method.—Filter the urine if not already clear, and if alkaline render slightly acid with acetic acid. If the specific gravity be 1010 or more, dilute the urine sufficiently to bring the density below that level (to 1008). This is important, and is often overlooked. Fill the tube with the urine up to the mark U. Pour in the reagent (Appendix, 13) up to the mark R.

Close the tube with a rubber stopper, and gently invert it a few times to allow the fluids to mix. Set aside for twenty-four hours. At the end of that time read off the level of the surface of the precipitate. The figures on the scale represent grams of dried albumin per litre of urine.

Divide by 10 to get the percentage, and multiply the result by 4·375 to get the amount of albumin in grains per ounce of urine. If the urine requires to be diluted, the result must, of course, be multiplied the requisite number of times.

Very small quantities of albumin cannot be estimated by Esbach's method, as the instrument does not record less than 0·1 per cent. If after the first trial the level of the precipitate is found to be above the mark 4, the urine must be diluted and a fresh estimation made.

An excretion of 8 grm. of albumin daily represents an ordinary degree of albuminuria. This is equivalent to about ½ per cent.

ii. **Proteosuria.**—This is a more correct term than "peptonuria," formerly in use. It is very doubtful, as before remarked, if true peptone ever occurs in the urine. The clinical significance of the presence of proteoses in the urine is not yet finally determined. Recent investigations tend to show that they may occur in any "infective" disease—i.e. wherever disintegration of tissue is going on under the action of micro-organisms. Thus they are not uncommonly met with in the urine in pneumonia. They are most constant, however, in cases where a large collection of pus has formed in the body—e.g. in empyema or large abscess formation. They have also been found in considerable quantity in some cases of nephritis. The proteose in these cases is usually transient and of little importance. Permanent and abundant proteosuria may almost be regarded as pathognomonic of diffuse sarcomatous degeneration of the bone-marrow (myelopathic, or Bence-Jones proteosuria).

Detection of proteoses.—There are two classes of proteoses—primary and secondary—the latter standing nearest to

the peptones. From a clinical point of view the differentiation of the two is of no importance, but they differ somewhat in their chemical reactions, and this necessitates the application of different tests for their detection. We will assume first that the urine to be examined is free from albumin. Proceed as follows :—

(*a*) Add to the urine (filtered and acidified if necessary) a few drops of a saturated solution of salicyl-sulphonic acid. Boil, and filter whilst hot. If the filtrate becomes cloudy on cooling, proteose is present.

The presence of antipyrin, quinine, and certain resins in the urine is apt to give a similar reaction.

(*b*) Apply Heller's test as already described (p. 341). A white cloud which disappears on heating and reappears on cooling indicates the presence of primary proteoses. The cloud is situated towards the upper part of the tube—it does not form a sharp ring close to the nitric acid, as is the case with albumin. The secondary proteoses do not give this reaction unless in the presence of an excess of salt.

(*c*) Add to the urine an equal volume of a saturated solution of common salt, and then drop in acetic acid so long as a cloud forms. If this disappear on heating and reappear on cooling, proteoses are present. Both forms of proteose give this reaction.

If the urine be already albuminous the albumin should be removed before testing for proteose. To do this, bring the urine to boiling-point, add a drop or two of acetic acid, and boil for two minutes. Filter and test filtrate as above.

The Bence-Jones proteose already referred to is probably a hetero-proteose. It may be present in enormous amounts, and sometimes appears in the urine as a precipitate. It gives the ordinary proteose reactions, but is characterized by its behaviour on heating. Urine containing it becomes opaque at a comparatively low temperature (below 60° C.), and a sticky coagulum forms which floats on the surface or adheres to the sides of the tube. If the reaction be acid this coagulum *disappears entirely or almost so on boiling, and reappears on cooling.* It is very easily soluble, too, in dilute alkali. The body also gives a sharp ring with strong hydrochloric acid, *even when the urine is diluted with twenty times its volume of water.*

This ring disappears on heating and reappears on cooling (Bradshaw's reaction).

If the presence of true peptone be suspected, the urine must be saturated while boiling with sulphate of ammonium, and the filtrate tested for peptone by the ordinary reactions—the best being the occurrence of a white ring on floating the urine on Spiegler's solution; or, better, dialyse the urine for two hours and test the dialysate for peptone. No proteose passes through in that time.

iii. **Nucleo-proteinuria and mucinuria.**—We have already mentioned that both a nucleo-protein (or a substance very closely resembling one) and mucin occur normally in the urine, and it is probable that the so-called "mucus" of the urine consists mainly of the former. In catarrhal conditions of the urinary passages, however, and especially of the bladder, an excess of what is perhaps true mucus may appear in the urine, and to this the term "**mucinuria**" has been applied. As long as the urine is acid, mucin is insoluble and forms a deposit at the bottom of the vessel. Such a deposit may be distinguished from pus by the absence of pus cells on microscopical examination, and by the fact that on adding to it some caustic potash the solution is not decidedly ropy, as it is in the case of pus. If the urine be alkaline the mucin goes partially or entirely into solution. It may then be detected by adding to the urine a few drops of acetic acid. A white cloud, insoluble in excess and increased on boiling, indicates mucin. This will often succeed better if the urine be previously diluted with its own bulk of water, as the presence of a large quantity of salts tends to prevent the precipitation.* There is no

* Previous dilution of the urine has also the advantage that it prevents one from being deceived by a precipitate of urates which may be thrown down if acetic acid is added to a concentrated urine.

simple clinical test for distinguishing true mucin from nucleo-protein in the urine.

"Fibrinuria" has already been described (p. 313).

(2) BLOOD AND ITS DERIVATIVES

Blood may appear in the urine as a whole (hæmaturia), or blood pigment may appear without corpuscles (hæmoglobinuria). These two conditions can only be differentiated by examining the deposit for blood cells. There is here one source of fallacy. In alkaline urines, especially if they have stood for some time, the red cells are apt to swell up and disappear. The urine should, therefore, be examined as fresh as possible.

If urine contains only a small amount of blood or blood pigment it has a peculiar opaque appearance, to which the term "smoky" is applied. Large quantities of blood give to the urine a red colour varying in intensity with the amount of blood present. The blood corpuscles are apt to settle at the bottom, producing a flocculent deposit, which is brown or red according to the amount of the blood and the degree of its alteration.

The following tests depend upon the presence of blood pigment, and therefore give a positive reaction both in **hæmaturia** and in **hæmoglobinuria**:—

i. **Heller's test.**—Place 2 in. of the urine in a test tube and render it strongly alkaline with caustic soda. Boil. If blood pigment be present the deposit is brownish-red in colour while the supernatant fluid is bottle-green.

The precipitate consists of earthy phosphates which have carried down with them hæmatin derived from the blood pigment, and are therefore reddish in colour instead of being white or yellowish. If the urine be alkaline a few drops of calcium chloride solution should be added to form more earthy phosphates.

If the urine be very dark, as it may be (e.g. from the presence of bile), the supernatant fluid should be decanted off the precipitate and replaced by water. The test is sufficiently delicate to reveal the presence of 1 c.c. of blood in 1 litre of urine.

Fallacies.—If the patient be taking senna, santonin, or rhubarb, the test may yield a positive result even although no blood is present. If the coloration be due to hæmoglobin, however, the precipitate yields the spectrum of alkaline hæmatin (Fig. 70, p. 240), and this excludes all possibility of fallacy.

ii. **Guaiac test.**—Take 1 in. of urine in a test tube, add to it two drops of tincture of guaiac. A white precipitate forms, owing to partial precipitation of guaiac resin. Now add 1 in. of ozonic ether without shaking. If blood pigment be present a blue colour appears at the junction of the fluids.*

The blue colour is due to oxidation of the guaiac by oxygen derived from the ozonic ether, the blood pigment acting as the carrier. Ozonic ether is a solution of peroxide of hydrogen in sulphuric ether.

Fallacies.—If iodides be present in the urine a blue colour is produced on applying the test. It is distinguished from that due to blood by the fact that it appears much more slowly, and by its appearing simultaneously all through the fluid, not at the junction of the ether and the urine.

Pus gives with guaiac alone a greenish-blue colour, which disappears on heating.

The presence of much saliva in the urine (e.g. from the patient spitting into it) is also a possible source of fallacy, as it gives the guaiac test owing to the

* The tincture of guaiac must be prepared from fresh—i.e. unoxidized—resin, and the ozonic ether must contain in solution peroxide of hydrogen of 30-volume strength. It should give off bubbles of gas when poured into the test tube. If these points be not attended to, the test may fail. Sanitas is a very good substitute for ozonic ether in the above test, and is less expensive.

presence of an *oxidase*. As oxidases are present sometimes in urine, it is better to boil the urine before performing the test. The coagulum in this case is turned blue when blood is present.

Methæmoglobinuria.—Methæmoglobin may be formed from hæmoglobin in any acid urine after it has stood for some time. Not infrequently, however, methæmoglobin is present in the urine when passed. It has been said to indicate that the hæmorrhage has its origin in the kidney. The characteristic smoky tint of the urine in hæmaturia of renal origin is largely due to methæmoglobin. The pigment present in "paroxysmal hæmoglobinuria" consists largely of methæmoglobin. Spectroscopic examination is the only satisfactory test for methæmoglobin. The urine, if very dark in colour, should first be diluted, and it should always be filtered. It should then be examined in a layer 5 cm. thick—a small flat glass bottle does well enough. If methæmoglobin be present there will be a band visible in the red, in addition to two bands nearly in the position of those due to oxyhæmoglobin (*see* Fig. 70, p. 240).

Hæmatoporphyrinuria.—Hæmatoporphyrin (iron-free hæmatin) occurs normally in the urine in very small amount, and may be considerably increased without affecting its colour. When present in large quantities the urine has a dark port-wine colour. Such a urine does not give the guaiac reaction. If examined with the spectroscope in a thin layer it may possibly show the characteristic spectrum of so-called alkaline hæmatoporphyrin, that being the form met with even in acid urines. Often, however, no distinct spectrum can be obtained on direct examination of the urine. In such a case the pigment can be extracted by shaking up the urine with a little amylic alcohol or acetic ether, after the addition of a few drops of acetic

acid. The extract so obtained shows the bands of alkaline hæmatoporphyrin, viz. four bands, one at the junction of the red and yellow, a second in the yellow, a third in the green, and a fourth (the broadest) between the green and the blue (*see* Fig. 70, p. 240). On adding a drop or two of hydrochloric acid the bands of acid hæmatoporphyrin are obtained, viz. two bands, one in the orange (narrow) and one at the junction of the yellow and green (broader). The latter is the characteristic band, and consists really of two halves, a lighter half on the side next the narrow band and a very dark half on the side away from it.

Hæmatoporphyrin sometimes appears in large amount in the urine of patients who are taking sulphonal, but much more commonly in females than in males. It is a sign of very grave significance, as such cases often terminate fatally. The excretion of port-wine-coloured urine by a patient who is taking sulphonal is always an indication for the immediate stopping of the drug and for the free administration of alkalis.

Urine which contains blood or hæmoglobin contains also, of course, some albumin, and it is often difficult to say whether the blood is sufficient to account for all the albumin present or whether true albuminuria exists as well. We have found that if human blood be added to normal urine in an amount sufficient to produce distinct smokiness, the quantity of albumin amounts to merely a trace. Even when the quantity added is sufficient to render the urine distinctly red, the amount of albumin, as shown by Esbach's method, is only $\frac{1}{2}$ per 1,000.

(3) SUGARS IN THE URINE

The sugars which are of most practical importance in the examination of the urine are glucose and lac-

tose. It is possible that lævulose may sometimes occur along with glucose. Cane sugar and maltose may conceivably appear in the urine if excessive quantities of either be ingested. Under rare conditions pentoses may also occur.

Glucose in the urine.—Glucose (dextrose or grape sugar), $C_6H_{12}O_6$, is by far the commonest variety of sugar met with in the urine. The condition is spoken of generally as "glycosuria." This must be distinguished from "diabetes." Diabetes—or, more correctly, diabetes mellitus—is a disease of which glycosuria is the chief symptom, but every patient with glycosuria has not necessarily got diabetes. It has long been disputed whether or not traces of glucose occur in normal urine. Recent researches appear to have finally settled the question. Traces of glucose *do* occur in normal urine, but not in an amount capable of detection by the reagents usually employed. If, therefore, glucose be detected by any of the tests we are about to describe, its presence may be regarded as pathological.

Tests for glucose in the urine.—A group of these tests depends upon the fact that glucose can become oxidized at the expense of certain metallic oxides, this oxidation occurring most easily at a temperature near boiling, and in the presence of free caustic alkali. Copper is the metal usually employed in the test. If one takes a solution of caustic soda and adds to it a few drops of a very dilute solution of sulphate of copper, a blue precipitate forms. This is hydrated cupric oxide ($CuOH_2O$). If now one boils the blue precipitate it becomes black from the separation of cupric oxide (CuO). In the presence of certain substances, however—such, for example, as a tartrate—the cupric hydrate formed on the addition of the sulphate goes into solution instead of being

precipitated. A deep-blue fluid then results, which remains unaltered on boiling. If an oxidizable substance such as glucose be present, however, the blue cupric hydrate is reduced on boiling to cuprous hydrate (Cu_2OH_2O), which is not capable of being held in solution, and accordingly appears as a yellow precipitate, or it may be further dehydrated to cuprous oxide (Cu_2O), which is red. Now, glucose is not merely capable of reducing cupric hydrate, but it is also one of the substances which, like the tartrates, are capable of holding it in solution. Hence, if a solution of glucose be rendered alkaline with caustic soda, and a few drops of cupric sulphate solution added, the cupric hydrate formed is dissolved and a blue solution results. On raising this to the boiling-point the glucose reduces the cupric hydrate, and yellow cuprous hydrate or red cuprous oxide is precipitated.*

Such is a brief account of the chemistry of the copper test for sugar. The two chief methods of applying it are :—

i. **Trommer's test.**—Take 2 in. of the urine in a test tube, add one-eighth of its volume of caustic potash, and then drop in carefully some 1 per cent. solution of sulphate of copper, shaking after each addition. If any considerable quantity of glucose be present the cupric hydrate formed is at once dissolved, and a blue solution results. Continue to add sulphate of copper until a little cupric hydrate remains undissolved. Boil the upper part of the fluid and it becomes yellow from separation of cuprous hydrate ; and on prolonged boiling this becomes red (Cu_2O). If more cupric hydrate has been formed than the amount of sugar present is capable of holding in solution, the excess yields black cupric oxide on boiling, which obscures the result. Hence the advantage of Fehling's reagent, in which solution of all the cupric hydrate is ensured by the addition of a tartrate. The formula for the reagent will be found in the Appendix (14).

If even a small quantity of glucose be present, the

* Cuprous hydrate is obtained when creatinin is present ; in the absence of the latter, red cuprous oxide is thrown down.

reduction in Trommer's test begins before the boiling-point is reached. Glucose is the only substance likely to be found in the urine which will do this.

ii. **Fehling's test.**—As a preliminary to carrying out the test, one must always make sure that the reagent is good. This is necessitated by the fact that Fehling's solution alters on keeping, with the result that on boiling it deposits a precipitate of cuprous oxide. The exact nature of the alteration is not fully understood. To test the Fehling's solution, add to it an equal volume of water, and boil for two minutes. If the solution remains clear, it is to be regarded as safe. Should a precipitate occur, a little more caustic soda should be added and the liquid filtered. It is then ready for use. Add to 1 in. of Fehling in a test tube a few drops of the urine (freed from albumin), and boil. If any considerable quantity of glucose is present, a yellow or red precipitate will appear. Should none be evident, add as much urine as there was Fehling, and boil for two minutes. Set aside. If after standing the solution still remains quite clear, there cannot be more than a mere trace of sugar present.

"If the proportion of sugar," says Allen,* "be moderate—that is, under 0·8 per cent.—the precipitation of the yellow or red cuprous oxide does not take place immediately, but occurs as the liquid cools, the appearance being somewhat peculiar. The liquid first loses its transparency, and passes from a clear bluish-green to an opaque light-greenish colour. This green milky appearance is said to be very characteristic of dextrose, but it would be more correct to say that its appearance indicated the presence of some substance interfering with the normal reaction of sugar." Creatinin is the chief substance which may act as such an interfering agent, and its presence renders the indications of Fehling's test uncertain when only small quantities of sugar are present. Allen has accordingly proposed the following modification of Fehling's test by taking advantage of the fact that a slightly acid

* "Chemistry of Urine," p. 61.

solution of cupric acetate will precipitate most of the "interfering" substances without affecting any form of sugar. He proceeds as follows :—

> Heat 7–8 c.c. of the urine to boiling in a test tube, and, without removing any precipitate of albumin, add 5 c.c. of the cupric sulphate solution used in preparing Fehling. Partially cool the liquid and add 1–2 c.c. of a saturated solution of sodium acetate containing enough acetic acid to give it a feebly acid reaction. Filter. To the filtrate add 5 c.c. of the alkaline tartrate mixture used for Fehling, and boil for twenty seconds. If more than 0·2 per cent. of sugar be present, cuprous oxide separates before the boiling-point is reached. With smaller quantities precipitation takes place during the cooling of the solution, which becomes greenish, opaque, and suddenly deposits cuprous oxide as a fine yellow precipitate.

Certain *precautions and fallacies* in the use of Fehling's test have still to be mentioned.

In the first place, the urine must be free from albumin. If necessary, add a drop or two of acetic acid to the urine, boil and filter. Neutralize the filtrate with a little calcium carbonate.

Fehling's test cannot be applied to strongly ammoniacal urine, as the free ammonia would prevent precipitation of cuprous oxide. In that case Pavy's modification should be employed (p. 358).

If the amount of glucose present be more than is required for reduction of all the cupric oxide, some of it is apt to be caramelized, especially on prolonged boiling. The whole liquid and precipitate then becomes of a dark-brownish colour.

The fallacies attendant upon the use of Fehling's test are due to the fact that other substances in the urine besides glucose can reduce cupric oxide. The chief of these are uric acid, creatinin, and hippuric acid among the normal ingredients ; of the abnormal constituents, the chief are lactose, glycuronic and glycosuric acids, and the products of certain drugs—e.g.

chloral, chloroform, glycerine, benzoic acid, salicylates, carbolic acid, etc.; "alkapton" urines, and those to which formalin has been added, also reduce Fehling. In a doubtful case, if the specific gravity of the urine be high, it should be reduced by the addition of water to about 1015. Any reduction of Fehling then obtained after boiling for ten seconds, either immediately or on standing for a minute or two, almost certainly indicates the presence of sugar in pathological amount, provided the patient be taking no drugs.

If one is still in doubt, the following additional tests should be employed :—

iii. **Nylander's test.**—The reaction depends upon the power of glucose to reduce bismuth compounds in alkaline solution with the formation of the black suboxide. To 10 c.c. of urine add 1 c.c. of the reagent (Appendix, 14*a*), shake, and boil for two or three minutes. If sugar be present a fine black precipitate of bismuth suboxide settles down.

This test is very delicate, and is not given by the reducing bodies often present in excess in normal urine. Glycuronic acid, lactose, and some drugs (e.g. rhubarb, senna, salol, turpentine, antipyrin, etc.), however, give a positive reaction.

iv. **Picric-acid test.**—Take 1 in. of urine in a test tube. Add $\frac{1}{4}$ in. of saturated solution of picric acid and a few drops of caustic potash; heat. If sugar be present, the solution becomes of a very dark *opaque* red colour, owing to the reduction of the picric to picramic acid.

Fallacies.—(*a*) Normal urine causes some darkening of the fluid when the test is carried out as above. This is owing to the creatinin which it contains. The colour is not nearly so dark as that produced by sugar, and the solution always remains transparent when held up to the light. (*b*) If the picric acid be impure, it may darken spontaneously when heated with caustic potash. It is as well, therefore, to test the picric acid employed, before using it.

v. **Phenyl-hydrazin test.**—Place 60 c.c. of urine in a 100 c.c. beaker. Add 1 grm. of sodium acetate and rather less pure phenyl-hydrazin hydrochlorate (the colourless crystals are best). Place in a water-bath, stirring from time to time, and scraping off any deposit which forms on the sides. Continue till the bulk is reduced to 10 or 15 c.c. Allow to cool, and examine the deposit after two hours. If there are no crystals, sugar is absent.

The following is a simpler form of the test for clinical purposes :—

Fill any ordinary test tube to a depth of ½ in. with powdered phenyl-hydrazin hydrochlorate, add another ½ in. of powdered acetate of soda. Half fill the tube with urine and bring to boiling-point. Continue boiling for two minutes, set aside for half an hour, and examine some of the deposit with a microscope.

When glucose is present, bright, sulphur-yellow, needle-shaped crystals will be found arranged in tufts, sheaves, or rosettes. If sugar be absent, only brown or yellowish globules or granules are seen, and in such a case the reduction of the Fehling's solution cannot have been due to glucose. Glycuronic acid, lactose, and the pentoses, however, yield crystals which might be mistaken for those given by glucose, so that if the phenyl-hydrazin yields a positive result at all it is always well to proceed to the fermentation test.

vi. **Fermentation test.**—This is really the one absolutely certain test for glucose, that being the only fermentable substance which is ever found in the urine. Neither lactose nor glycuronic acid—the two substances most liable to be mistaken for glucose—is fermentable.

The following precautions must be observed in carrying out the test : (*a*) The urine must be acid. Alkaline urine would putrefy ; therefore render it acid, if necessary, by adding tartaric acid. (*b*) Boil the urine for ten minutes, so as to drive off any air it may contain. Use German yeast. Shake the urine up with a small piece of it, so as to form an emulsion free from lumps, then place the urine so prepared in a tube. Special fermentation tubes are manufactured. If one of these is not obtainable, an ordinary test tube inverted in a bath of mercury will do. A Doremus (Southall's) ureometer tube does

extremely well. The long limb of it should be filled with the urine completely, no air bubbles being left. Set aside the tube in a warm place, and examine after a few hours. If a distinct bubble has appeared at the top of the tube, the urine is fermentable, and contains at least 0·5 per cent. of glucose. Care must be taken to ascertain that the yeast is active. It should be tested with a dilute solution of glucose. It is also well to have a control tube full of normal urine to which yeast has been added, as the yeast is apt to give off a little gas.

If these precautions are observed, the test is trustworthy and delicate.

The only drawback to the fermentation test is that it may not give any reaction if the urine contains less than 0·1 per cent. of sugar. If, then, no gas be produced on fermentation, one should apply Fehling's test to the urine after it has been submitted to the action of yeast for twenty-four hours. If no reduction be now obtained, then the urine has contained a trace of glucose, though not enough to ferment appreciably.

Quantitative estimation of sugar.—i. **Fehling's method** consists in titrating the urine with a known quantity of Fehling's solution at boiling temperature, and observing when all the cupric oxide has been reduced to cuprous oxide, as evidenced by the discharge of all the blue colour from the solution. Owing to the formation of the red precipitate, it is not an easy matter to determine accurately when the blue colour has disappeared, and for this reason Fehling's method is not recommended for quantitative work, one of the following modifications being preferable.

ii. **Ling and Rendle's method.** — In this method the complete reduction of the cupric salt is determined by using a solution of ferrous thiocyanate as *indicator*. This is prepared by dissolving with gentle warmth 1 grm. of ferrous ammonium sulphate and 1·5 grm. of ammonium thiocyanate in 10 c.c. of water; cooling, and adding 2·5 c.c. of concentrated HCl. If the solution thus obtained has a brownish-red colour due to the ferric salt, it may be reduced by shaking with a little zinc dust. This indicator shows

the presence of a cupric salt. When such a salt is brought into contact with the ferrous thiocyanate, the ferrous salt becomes converted into the ferric salt, which is blood-red in colour. Fehling's solution therefore gives a blood-red colour until all the cupric salt in it has become reduced to cuprous by the sugar.

One proceeds as follows: If ordinary diabetic urine is being examined, it should be diluted to the extent of 1 in 20 (5 c.c. of urine to 95 c.c. of water).* Fill a burette with it. Measure 10 c.c. of Fehling's solution into a flask of 100 c.c. capacity, add a little strong caustic soda solution,† and water to 50 c.c. Boil. Whilst it is boiling, run in the urine till the fluid above the precipitate of cuprous oxide is judged to have lost all colour. A drop of the fluid is now removed and tested with a drop of the indicator upon a slab of porcelain; if *no* red colour is produced the reduction of the Fehling's solution is complete.

Calculation.—10 c.c. Fehling = 0·05 grm. glucose. Suppose 10 c.c. of the diluted urine has been used, and 5,000 c.c. is the amount of urine passed in twenty-four hours: then

$$\text{Sugar in 24 hours} = \frac{5{,}000 \times 0{\cdot}05}{10} = 25 \text{ grm.}$$

but the urine was diluted 1 in 20,

$\therefore$ sugar in 24 hours $= 25 \times 20 = 500$ grm.

The result must be multiplied according to the degree of dilution.

iii. **Pavy's method** is also convenient for clinical purposes. It differs from Fehling's in this—that a quantity of strong ammonia is added to the alkaline cupric tartrate solution. Ammonia is able to dissolve cuprous oxide, the solution being colourless. Hence, when the solution is titrated at the boiling-point with a solution containing glucose, the mixture gradually fades until every trace of blue has gone, and one is not confused by the throwing down of a red precipitate.

* The urine must be diluted to such an extent that not more than 5–10 c.c. are required to reduce 10 c.c. of Fehling.

† This facilitates the separation of the cuprous oxide.

Pavy's solution has also the advantage of keeping indefinitely, and of being possessed of an originally deeper colour. The method of preparing it will be found in the Appendix (15).

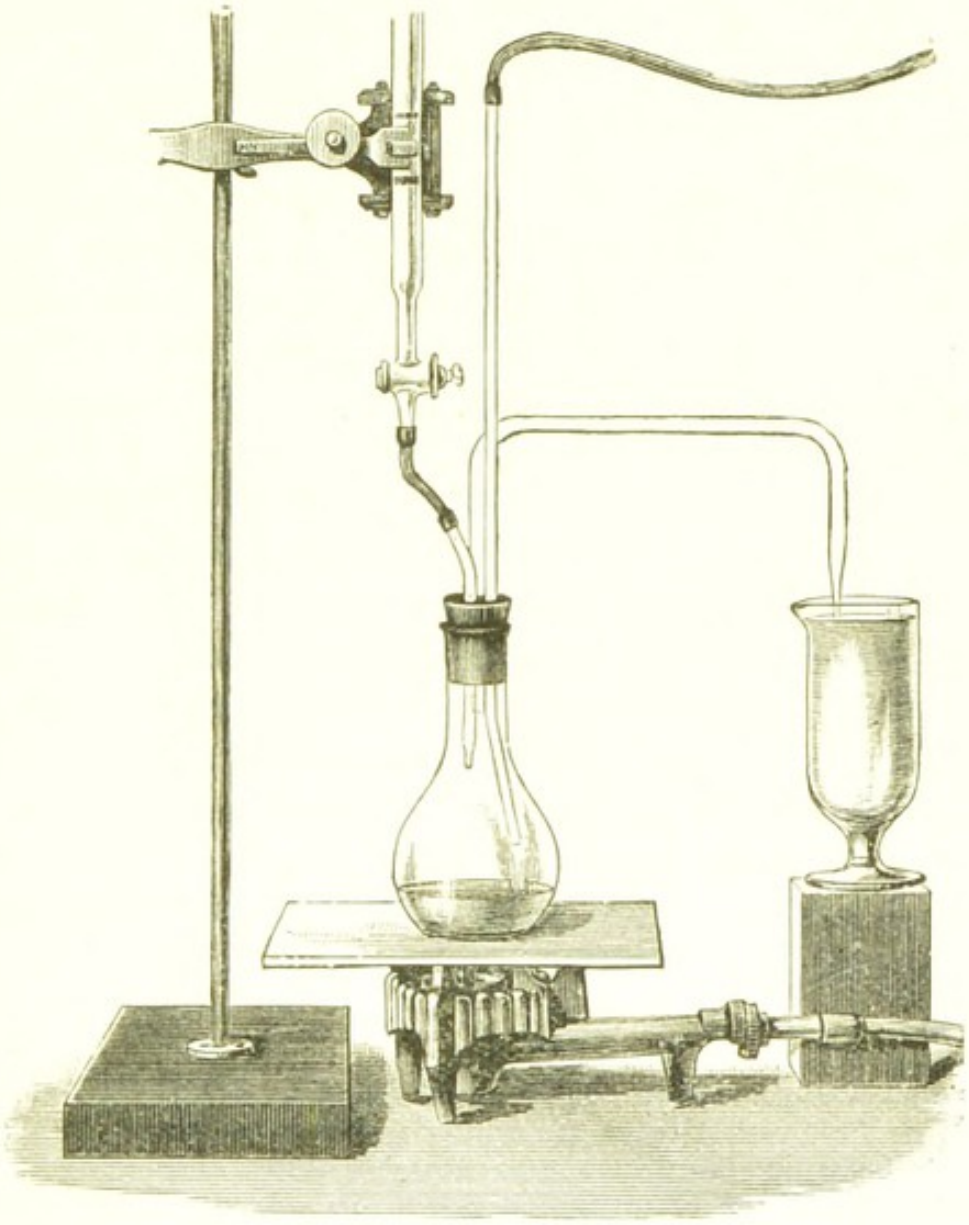


Fig. 88.—Pavy's apparatus.

Method.—Fill a burette with urine diluted 1 in 20 (5 c.c. urine to 95 c.c. of water) for ordinary diabetic urine. The outflow from the burette should be regulated by a stopcock or screw. The end of the burette is connected with a tube passing through the stopper of a 150 c.c. flask. Another hole in the stopper allows the passage of an exit tube for the escape of the fumes of ammonia (Fig. 88). Place in the flask 10 c.c.

of the solution, diluted with 20 c.c. of water.* When the solution is boiling, run in the urine. It should be run in at the rate of 60–100 drops per minute—not too fast, or the limit of reduction may be overstepped ; and not too slowly, or all the ammonia may be driven off and some cuprcus oxide thrown down. When the blue colour has entirely faded, take a reading of the burette.

The calculation is performed in the same way as for Fehling's method, but it must be remembered that 10 c.c. of Pavy's solution only = 0·005 grm. of glucose, i.e. it is ten times less strong than Fehling's. The accompanying table (p. 361) saves the trouble of calculation. It gives the sugar in grams per 1,000 c.c. The same table may be used for Ling and Rendle's and Gerrard's methods (provided that 10 c.c. of Fehling has been taken), the number of parts per 1,000 being always multiplied by 10 to allow for the greater strength of Fehling's solution ; e.g. if 10 c.c. diluted urine is used in Pavy's method, reference to the table shows that this means 0·5 grm. sugar per 1,000 c.c. ; by Fehling's method it would be equivalent to 5 grm. per 1,000 c.c. In each case the result must be multiplied by 20 if the urine has been diluted 1 in 20, and if it be desired to express the result in grains per ounce the amount per 1,000 c.c. should be multiplied by 0·4375.

* Some writers recommend that 50–100 c.c. of Pavy's solution be taken. This necessitates the addition of a little talc or pumice-stone to prevent bumping, and also of some arrangement to neutralize the fumes of ammonia given off. A jar of acidulated water (as shown in figure) does this quite well. A layer of liquid paraffin poured on its surface serves as a valve and prevents any air from getting in and causing re-oxidation of the cuprous oxide. Sutton leads the fumes away by an elastic tube closed at the end by a plug of glass rod, with a transverse slit just above the plug. This allows the fumes to escape, and prevents the return of fluid in case of a vacuum forming. Pavy himself uses only 5–10 c.c. of his solution. If the urine is sufficiently diluted this gives quite accurate results, and the small amount of ammonia given off is no inconvenience.

Estimation of Sugar

Table showing the Amount of Sugar, expressed in parts (by weight) per 1,000 (by volume), corresponding with cubic centimetres, in 10ths, required to decolorize 10 c.c. of the Ammoniated Cupric Test.

C.c. to decolorize	Parts per 1,000	C.c. to decolorize	Parts per 1,000	C.c. to decolorize	Parts per 1,000
1·0	5·000	5·7	·877	10·4	·480
·1	4·545	·8	·862	·5	·476
·2	4·166	·9	·847	·6	·471
·3	3·846	6·0	·833	·7	·467
·4	3·571	·1	·819	·8	·462
·5	3·333	·2	·806	·9	·458
·6	3·125	·3	·793	11·0	·454
·7	2·941	·4	·781	·1	·450
·8	2·777	·5	·769	·2	·446
·9	2·632	·6	·757	·3	·442
2·0	2·500	·7	·746	·4	·438
·1	2·380	·8	·735	·5	·434
·2	2·272	·9	·724	·6	·431
·3	2·173	7·0	·714	·7	·427
·4	2·083	·1	·704	·8	·423
·5	2·000	·2	·694	·9	·420
·6	1·923	·3	·684	12·0	·416
·7	1·851	·4	·675	·1	·413
·8	1·785	·5	·666	·2	·409
·9	1·724	·6	·657	·3	·406
3·0	1·666	·7	·649	·4	·403
·1	1·612	·8	·640	·5	·400
·2	1·562	·9	·632	·6	396
·3	1·515	8·0	·625	·7	·393
·4	1·470	·1	·617	·8	·390
·5	1·403	·2	·609	·9	·387
·6	1·388	·3	·602	13·0	·384
·7	1·351	·4	·595	·1	·381
·8	1·316	·5	·588	·2	·378
·9	1·281	·6	·581	·3	·375
4·0	1·250	·7	·574	·4	·373
·1	1·219	·8	·568	·5	·370
·2	1·190	·9	·561	·6	·367
·3	1·162	9·0	·555	·7	·364
·4	1·136	·1	·549	·8	·362
·5	1·111	·2	·543	·9	·359
·6	1·086	3	·537	14·0	·357
·7	1·063	·4	·531	·1	·354
·8	1·041	·5	·526	·2	·352
·9	1·020	·6	·520	·3	·349
5·0	1·000	·7	·515	·4	·347
·1	·980	·8	·510	·5	·344
·2	·961	·9	·505	·6	·342
·3	·943	10·0	·500	·7	·340
·4	·925	·1	·495	·8	·337
·5	·909	·2	·490	·9	·335
·6	·892	·3	·485	15·0	·333

In both methods the urine must be freed from albumin (if necessary) by adding two drops of acetic acid, boiling, neutralizing with calcium carbonate, filtering, and making up to the original volume with water.

iv. **Gerrard's cyano-cupric method.**—This method depends upon the fact that the colourless double cyanide of potash and copper is capable of holding cuprous oxide in solution just as ammonia does in Pavy's process. If, therefore, Fehling's solution is titrated with a sugar solution in the presence of this cyanide, the blue colour fades gradually, no precipitate being thrown down. The end point is thus very sharp, and, as there is no tendency to re-oxidation, the process may be safely conducted in an open porcelain basin.

To prepare the double cyanide.—Dilute 100 c.c. of Fehling with about 300 c.c. of water, boil, and run in cautiously an approximately 5 per cent. solution of cyanide of potash. When the colour is just gone, dilute to exactly 500 c.c., and keep in a well-stoppered bottle.

Method of titration.—To 30 c.c. of the above solution add 10 c.c. of Fehling, and boil in a porcelain basin. Run in the diluted urine cautiously till the colour of the solution has just gone. As the double cyanide undergoes no reduction, one has only to reckon with the 10 c.c. of Fehling added. This process is rapid and accurate enough for clinical use. As a mixture of the double cyanide and Fehling's solution will keep quite well for a number of weeks, it is easy to prepare it in bulk and to measure out each time such an amount as will correspond to 10 c.c. of Fehling.

v. **Polarimetric method.**—In this method advantage is taken of the fact that the amount of sugar in urine may be estimated by observing the degree of rotation of polarized light produced by a layer of urine 1 or 2 decimetres thick. The calculation is based on the fact that theoretically 100 grm. of sugar dissolved in 100 grm. of water, observed as a layer 1 decimetre thick, rotates polarized light through a definite number of degrees, 52·5° to the right for dextrose, 52·4° to the right for lactose, and other numbers for other sugars. In the case of dextrose, therefore,

$$\text{a rotation of } 1^\circ = \frac{100}{52{\cdot}5} \text{ grm. of sugar in 100 c.c.}$$
$$= 1{\cdot}905 \text{ grm. in 100 c.c.}$$

when seen through a tube 1 decimetre long.

A table may therefore be constructed by multiplying this figure by the different numbers up to 9. When a tube 2 dcm. long is used it is necessary to divide the reading by 2; 1° therefore = 0·952 grm. of sugar.

The following is a table for use with 1 dcm. and 2 dcm. tubes :—

	With 1 dcm. tube	*With 2 dcm. tube*
1° rotation	= 1·905 grm. of sugar per 100 c.c.	0·952
2° ,,	= 3·810 ,, ,, ,,	1·905
3° ,,	= 5·715 ,, ,, ,,	2·852
4° ,,	= 7·620 ,, ,, ,,	3·810
5° ,,	= 9·525 ,, ,, ,,	4·762
6° ,,	= 11·430 ,, ,, ,,	5·715
7° ,,	= 13·335 ,, ,, ,,	6·667
8° ,,	= 15·240 ,, ,, ,,	7·620
9° ,,	= 17·145 ,, ,, ,,	8·572

By merely altering the position of the decimal point the amount of glucose causing a given degree of rotation is easily calculated. For example, suppose, using a 1 dcm. tube, a rotation of 4·3° is observed; then—

$$4^\circ = 7{\cdot}620$$
$$0{\cdot}3^\circ = 0{\cdot}5715$$
$$= 8{\cdot}1915 \text{ grm. of sugar per 100 c.c. of urine.}$$

Various forms of polarimeter are in use, and for the principle of action of the different instruments advanced manuals must be consulted. To make a reading, proceed as follows :—

The urine must be *clear* and not high-coloured. If necessary treat every 50 c.c. of urine with 10 c.c. of a 25 per cent. solution of lead acetate and use the filtrate. Ascertain that the zero of

the polarimeter is correct by observing when an equal illumination of the field is given by a tube of water. If an error exists, this must be added to or deducted from the subsequent reading. Carefully fill the observation tube to overflowing, slide on the cover-glass so that no air bubbles are included, place in polarimeter. Upon observation the field is found to be unequally illuminated. Equal illumination is now restored by careful rotation (clockwise = dextro-rotation), and the amount read off on the scale provided, usually with the aid of a vernier. Several observations should be made, as a rule, and the mean reading taken.

It is to be borne in mind that the method is inapplicable if the urine contains lævo-rotatory substances such as *protein*, lævulose, *β*-oxybutyric acid, and the compound glycuronates. Naturally also the dextro-rotatory substance should be ascertained to be glucose. Protein may be removed by heating with a little acetic acid or treating with lead acetate, as mentioned above.

Apart from this, the method is simple and sufficiently exact for clinical purposes.

vi. **Fermentation method.**—An approximate estimate of the amount of sugar present may be arrived at by noting the change in specific gravity that follows fermentation. The specific gravity of the urine is taken before and after fermentation with yeast for twenty-four hours. Every degree of specific gravity lost represents 1 gr. of sugar per ounce of urine.

In an ordinary case of diabetes, 3 litres or so (about 5½ pints) of urine will be passed daily, containing on an average 100 grm. of glucose (3–4 per cent.).

In recording the amount of sugar passed in a case of diabetes it is better to make an accurate estimation every third day, or even once a week, rather than a hurried and unreliable calculation every day.

Lactosuria.—Lactose is sometimes found in appreciable quantity in the urine of women who are nursing. It reduces Fehling's solution, and gives with

the phenyl-hydrazin test yellow rosettes of phenyl-lactosazone which are smaller than the sheaves yielded by glucose, but it gives no reaction with the fermentation test. It may be estimated by titration with Fehling's solution, it being remembered that the reducing power of lactose is to that of glucose as 7 is to 10; i.e. if 7 parts of glucose reduce a given quantity of Fehling, it will require 10 of lactose to effect the same result.

Pentosuria.—This rare condition consists in the presence in the urine of pentoses, i.e. carbohydrates containing only 5 atoms of carbon. They have the general formula $C_5H_{10}O_5$. The pentoses do not exist free in nature, but can be obtained easily by hydrolytic decomposition of complex carbohydrates belonging to the gum class, present in many fruits (e.g. cherries). They are distinguished from the hexoses, such as glucose, in not furnishing lævulinic acid in decomposition with sulphuric acid or HCl; when gently heated with phloroglucin and HCl they give a cherry-red reaction; with orcin and HCl a green colour, changing to greenish-blue precipitate.

They furnish osazones (M.P. 156–160), are not fermentable, and, on distillation with HCl, furfurol is given off.

They are optically active, and reduce Fehling's and Nylander's solutions.

The best test for pentoses is with Bial's reagent (1 grm. of orcin, 500 c.c. of HCl, specific gravity 1151, 25 drops of 10 per cent. solution of ferric chloride). Boil 5 c.c. of this reagent in a test tube, *remove from flame*, add 5 drops of urine. A green ring at junction is diagnostic of pentoses. Glycuronic acid and compound glycuronates (p. 371) give the tests for pentoses, including Bial's, but there is no risk of confusion if the test tube be first *removed from the flame.*

Pentoses occasionally occur in diabetic (hexose) urine. They are also present after the ingestion of

certain fruits (cherries, grapes, plums). But their chief interest is in the connection with so-called "pentosuria," a rare anomaly of metabolism not necessarily attended by morbid symptoms, probably harmless and needing no treatment.

(4) BILE IN THE URINE

Both bile pigment and bile acids may be present. Usually they occur together, but the pigment much more abundantly than the acids. The usual cause of the entrance of the bile constituents into the urine is some obstruction in the bile passages. As long as the urine is fresh, bilirubin is the form of bile pigment always found in it. After it has stood for some time, biliverdin is apt to be formed as the result of oxidation.

Urine which contains bile is greenish or brownish-yellow in colour, and somewhat more viscid than normal, so that the froth which forms on the top after shaking is unusually permanent. Salol urine may closely resemble urine which contains bile, but the froth in the latter case is also greenish; in salol urine it is not.

Tests for bile pigment. Gmelin's test.—Place some of the urine in a conical glass, and run a little impure nitric acid down the side so as to form a layer at the bottom. Oxidation of the bile pigment occurs, the most highly oxidized product (choletelin) forming a yellowish-red ring nearest the acid. Above this is a reddish ring, then violet (bilicyanin), and highest of all, green (biliverdin). Of these rings the green is alone characteristic of bile; all the others may be yielded by urinary indigogens.

The test, as thus carried out, is not very sensitive, and may fail even when 5 per cent. of bile is present. The sensitiveness of the reaction can be increased by filtering the urine repeatedly through an ordinary filter-paper. The latter becomes impregnated with the bile pigment, and if a drop of impure nitric

acid be placed upon it a play of colours can easily be seen.

The following modifications of it are much more delicate, and should always be employed in doubtful cases. They will reveal the presence of 0·2 per cent. of bile.

i. To 50 c.c. of urine add 5 c.c. of 10 per cent. barium chloride solution and 5 c.c. of chloroform. Shake for several minutes. Set aside for ten minutes. The chloroform and precipitate of phosphates fall down, carrying with them any bile pigment. If there is still any of the precipitate suspended, move the jar gently to and fro for a little, when it will settle down. Now draw off the chloroform and precipitate with a pipette ; if some urine is removed at the same time, no matter. Place in a flat dish, and set the latter over a basin of hot water till all the chloroform has evaporated. Allow to cool, and pour off any fluid from the precipitate. The latter will be yellowish. Place impure nitric acid in drops here and there on the surface of the precipitate. If bile pigment be present, a play of colours appears round each drop.

ii. Render 10 c.c. of the urine alkaline with caustic soda, and add a little 10 per cent. of calcium chloride solution. Collect and wash the precipitate, and place it along with the filter-paper in a small porcelain dish. Pour on it 10 c.c. of acid alcohol (5 c.c. of strong HCl to 100 c.c. of alcohol). Heat the yellowish solution in a test tube. If bile be present, it becomes green or bluish.

Iodine test.—If a 10 per cent. alcoholic solution of iodine is poured on the top of the urine in a test tube an emerald-green layer appears where the two fluids join, if bile be present.

Tests for bile acids.—Pettenkofer's test for bile acids is inapplicable in the case of urine, for even normal urine gives, with strong sulphuric acid, a purplish colour which might be mistaken for a positive reaction.

The simplest test is **Hay's sulphur test.**

Sprinkle some powdered sulphur upon the surface of the urine. If bile salts be present it will sink ; with normal urine it floats. This test depends upon the fact that bile salts lower the surface tension of fluids in which they are dissolved.

Oliver has proposed the following test. It depends upon the power of bile acids to precipitate peptone in acid solution. The peptone solution is prepared as in the Appendix (16).

Oliver's test.—Filter the urine until quite clear, acidify it if necessary, and dilute it till the specific gravity is less than 1008. Take 60 minims of the solution in a test tube and add to it 20 minims of the urine. If bile acids be present a decided milkiness appears at once, and is dense in proportion to the amount of acids. It may disappear on agitation, but reappears on adding more of the solution. The test is extremely delicate, and nothing as yet found in the urine interferes with it.

(5) PUS IN THE URINE (PYURIA)

The naked-eye characters of a urine which contains pus have already been described (p. 319). On chemical examination such a urine is, of course, always albuminous. It is often difficult to decide, just as it is in hæmaturia, whether all the albumin is accounted for by the pus alone or whether there is true albuminuria in addition. Reinecke has proposed the following method for enabling one to form a conclusion in this matter. He shakes up the urine of twenty-four hours thoroughly, so as to diffuse the pus evenly through it. He then counts the pus cells present, by means of a hæmocytometer, just as in the method for estimating the red blood-corpuscles in the blood, only without previous dilution. He finds that 100,000 pus cells per cubic millimetre should correspond to 1 per cent. of albumin (Esbach). If there be more albumin than this with that number of corpuscles, then albuminuria is present in addition to pyuria. Obviously, the method can only afford approximate indications. Moreover, it is inapplicable if the urine be ammoniacal, or if it contain much mucus. It should be added that if the number of pus cells exceeds 3,000

per cubic millimetre, the urine should be diluted with 1 per cent. salt solution prior to counting.

Tests for pus.—As already mentioned, urines which contain pus give, on the addition of guaiac alone, a green colour, which, however, disappears upon heating.

If caustic potash be added to the deposit of pus, a ropy, gelatinous mass results. For the microscopical recognition of pus in the urine, *see* p. 385.

(6) SOME RARER ABNORMAL CONSTITUENTS OF URINE

i. **Urinary indigogens.** — We have seen (p. 326) that indol is excreted in the urine in the form of potassium indoxyl sulphate—the so-called "indican." Small quantities of potassium skatoxyl sulphate, derived from skatol, are also to be found in human urine. On oxidation these compounds yield coloured substances, indigo blue and indigo red. Hence they are called urinary indigogens. To detect their presence one oxidizes them in one of these ways :

(*a*) Remove albumin, if present, by boiling. Add to some of the urine in a test tube an equal quantity of hydrochloric acid and a few drops of nitric acid, and boil. Cool and shake up with a little chloroform. The chloroform dissolves out the products of oxidation and becomes of a violet tint from the mixture of indigo blue and indigo red, if excess of indigogens be present. The presence of iodides in the urine must be excluded before applying this test.

(*b*) Mix 5 c.c. of urine with an equal volume of strong HCl containing 0·4 per cent. ferric chloride (Obermayer's reagent). Add 3 c.c. of chloroform and shake well. On settling, the chloroform becomes tinged blue if indican be present.

This test may be used roughly as a quantitative one, judging the amount of indican by the intensity of the blue colour produced. It is always to be borne in mind, however, that this is liable to the fallacy that indigo red may also be produced, and thus the intensity of the colour be misleading.

Traces of the indigogens are normally present in

the urine. The reddish-yellow transparent ring which appears above a layer of nitric acid when the latter is added to the urine is due to their partial oxidation.* They are increased in all conditions associated with excessive putrefaction. Hence they are much increased whenever the intestinal contents are unduly retained, e.g. in chronic constipation and intestinal obstruction. They are also increased in some fevers.

ii. **Acetone.**—Hydroxybutyric acid, aceto-acetic acid, and acetone may all occur in the urine. The relationship between the three may be seen from the following formulæ :—

β-hydroxybutyric acid	= $CH_3CH(OH)CH_2COOH$.
Aceto-acetic acid	= CH_3CO, CH_2COOH.
Acetone	= CH_3, CO, CH_3.

β-hydroxybutyric acid is formed first, probably, by destruction of proteins ; it then becomes oxidized, yielding aceto-acetic acid,

$$CH_3CH(OH)CH_2COOH + O = CH_3COCH_2COOH + H_2O.$$

The aceto-acetic acid is very easily decomposed into acetone and CO_2,

$$CH_3COCH_2COOH = CH_3COCH_3 + CO_2.$$

Only aceto-acetic acid and acetone require to be detected in the urine. Hydroxybutyric acid always occurs along with the first of these.

Test for aceto-acetic acid.—The urine must be fresh and unboiled, as the acid readily decomposes. Take some urine in a test tube, drop in a solution of perchloride of iron, diluted until it is of a pale sherry colour, as long as a precipitate of phosphate of iron falls. Filter, and add to the filtrate another drop or two of the iron solution. The solution becomes claret-coloured if aceto-acetic acid be present. On boiling the urine the colour disappears.

* In part also the reddish colour so obtained is due to the pigment urorosein, which is produced from its chromogen by the action of mineral acids. Its clinical significance is unknown, but it is often met with in the urine of chlorotic patients, and is probably of intestinal origin.

Antipyrin, salol, salicylates, carbolic acid, and some other drugs give a similar colour with perchloride of iron, but it is not affected by heat.

Tests for acetone.—Urine containing acetone has a peculiar fruity odour. It reduces Fehling's solution. A test for its presence is based upon its ready conversion into iodoform :—

$$C_3H_6O + 4KOH + 3I_2 = 3KI + KC_2H_3O_2 + CHI_3 \text{ (Iodoform)} + 3H_2O.$$

To 1 in. of the urine add 5 drops of 10 per cent. caustic soda or potash. Heat gently. Then drop in a saturated solution of iodine in potassium iodide until the liquid has a yellowish-brown colour. Then add a little more caustic potash or soda. Iodoform appears as a yellowish turbidity, which settles down into a crystalline precipitate. It may be recognized by its odour. Under the microscope it consists of hexagonal plates often gathered into stars.

Another test for acetone consists in adding to the urine a few drops of freshly prepared sodium nitro-prusside solution, ammonia till alkaline, and saturating the liquid with crystals of ammonium sulphate. A deep-violet colour similar to potassium permanganate solution develops in from one to fifteen minutes (Rothera's test).

If only traces of acetone be present, it is better to distil the urine after the addition of a little phosphoric acid, and test the distillate as above.

Acetone and the substances from which it is derived are especially apt to appear in the urine in cases of diabetes, and are to be regarded as of grave import, their appearance being often followed or accompanied by the development of coma. They are also found in some fevers and in the condition termed "acidosis."

iii. **Glycuronic acid** ($C_6H_{10}O_7$) is probably derived in the body from dextrose. Mere traces of it exist in combination in normal urine. It is very prone to form ethereal or glucosidal compounds if suitable substances are introduced into the circulation. Hence it appears in the urine in considerable quantity, in

paired combination with aromatic substances, etc., after the administration of such drugs as chloral, benzoic acid, chloroform, morphia, etc. This circumstance gave rise to the old belief that such drugs produce glycosuria ; in reality the substance which is excreted after their use is glycuronic acid, not glucose.

Occasionally glycuronic acid occurs spontaneously in the urine. It is then very apt to be mistaken for glucose. The error is a serious one, for the pathological significance of glycuronic acid in the urine is much less grave than that of glucose. Glycuronic acid reduces Fehling's solution, and gives a yellow crystalline precipitate with the phenyl-hydrazin test. It can be best distinguished from glucose in the following ways :—

(*a*) It does not ferment with yeast.

(*b*) It gives a deep-red colour with phloroglucin and HCl.

But (*c*) it does not reduce Bial's reagent when the urine is added to the boiling reagent (cf. Pentoses, p. 365).

iv. **Cystin** $(C_3H_6NSO_2)_2$ is sometimes found as a deposit in acid urines. It is recognized by its characteristic crystals (*see* Fig. 93, *b*, p. 381). It is soluble in alkalis ; hence the deposit disappears when the urine putrefies, an odour of sulphuretted hydrogen being evolved. If urine which contains it is boiled with a little caustic potash and acetate of lead, a black precipitate of sulphide of lead appears.

Cystin is a product of protein metabolism, and is probably derived from cystein, the normal oxidation of which has somehow been interfered with. It is often associated with the appearance in the urine of diamines, such as cadaverin and putrescin. It is about twice as common in males as in females, and has rarely been observed after 50 years of age. It is

apt to occur in several members of the same family, and may appear either persistently or intermittently. It is of interest as being due to an inborn error of metabolism, but is of little pathological significance, except from its tendency to form calculi.

(7) DRUGS IN THE URINE

Antipyrin.—After its use the urine may be red and dichroic, leading to the suspicion that blood is present. On adding a little dilute perchloride of iron a purplish-red colour develops, which persists on boiling, but disappears on adding an acid. Urines containing antipyrin produce a partial reduction of Fehling's solution on boiling.

Bromides.—Add a little hydrochloric acid and a few drops of a weak solution of bleaching powder. Shake with chloroform, and the latter becomes brownish-red from the solution of the free bromine.

Carbolic acid (*see also* section on Colour of Urine, p. 313).—The best test for it is to add a little bromine water. The appearance of a whitish precipitate (tribromophenol) indicates the presence of phenol.

Chloral, chloroform, etc., lead to the appearance of glycuronic acid (p. 371).

Iodides.—Acidify the urine with a little pure nitric acid, and shake up with chloroform. The latter becomes of a rose-red colour.

Iron.—Add a few drops of nitric acid. Boil, cool, and add a little 10 per cent. ferrocyanide of potash. A precipitate of prussian blue forms if iron is present.

Rhubarb and santonin have been referred to under the Colour of the Urine (p. 312).

Salicylates and **salol** appear in the urine as salicyluric acid. Such a urine gives a bluish-violet

colour on the addition of a little perchloride of iron ; it also partially reduces Fehling's solution.

Tannin gives a bluish-black colour with perchloride of iron.

(8) CAMMIDGE'S PANCREATIC REACTION

This test is believed by many, when taken in conjunction with clinical evidence and the results of a chemical examination of the fæces, to be of help in the diagnosis of pancreatitis. It is carried out as follows :—

A specimen of the twenty-four hours' urine is filtered several times through the same filter-paper, and, if acid in reaction and free from protein and sugar, 2 c.c. of strong hydrochloric acid (specific gravity 1016) are mixed with 30 c.c. of the clear filtrate, and the mixture is gently boiled on the sand bath in a small flask having a long-stemmed funnel in the neck to act as a condenser. After ten minutes' boiling the flask is well cooled in a stream of water and the contents made up to 30 c.c. with cold distilled water. The excess of acid present is neutralized by slowly adding 8 grm. of lead carbonate. After standing for a few minutes to allow of the completion of the reaction, the flask is again cooled in running water and the contents filtered through a well-moistened close-grained filter-paper until a perfectly clear filtrate is secured. The filtrate is then well shaken with 4 grm. of powdered tribasic lead acetate and the resulting precipitate removed by filtration, a clear filtrate being obtained by repeating the filtration several times if necessary. Since the large amount of lead now in solution would interfere with the subsequent steps of the experiment, it is removed either by treatment with a stream of sulphuretted hydrogen or, what is equally satisfactory and less disagreeable, by precipitating the lead as a sulphate. For this purpose the clear filtrate is well shaken with 2 grm. of finely powdered sodium sulphate, the mixture heated to the boiling-point, then cooled to as low a temperature as possible in a stream of cold water, and the white precipitate removed by careful filtration ; 10 c.c. of the perfectly clear transparent filtrate are made up to 18 c.c. with distilled water and added to 0·8 grm. of phenyl-hydrazin hydrochlorate and 2 grm. of powdered sodium acetate contained in a small flask fitted with a funnel condenser. The mixture is boiled on a sand

bath for ten minutes, then filtered hot through a filter-paper moistened with hot water into a test tube provided with a 15 c.c. mark. Should the filtrate fail to reach the mark, it is made up to 15 c.c. with hot distilled water; but this is rarely necessary, as after a little practice it is possible to regulate the boiling process so that the final result always comes out at between 15 and 16 c.c.

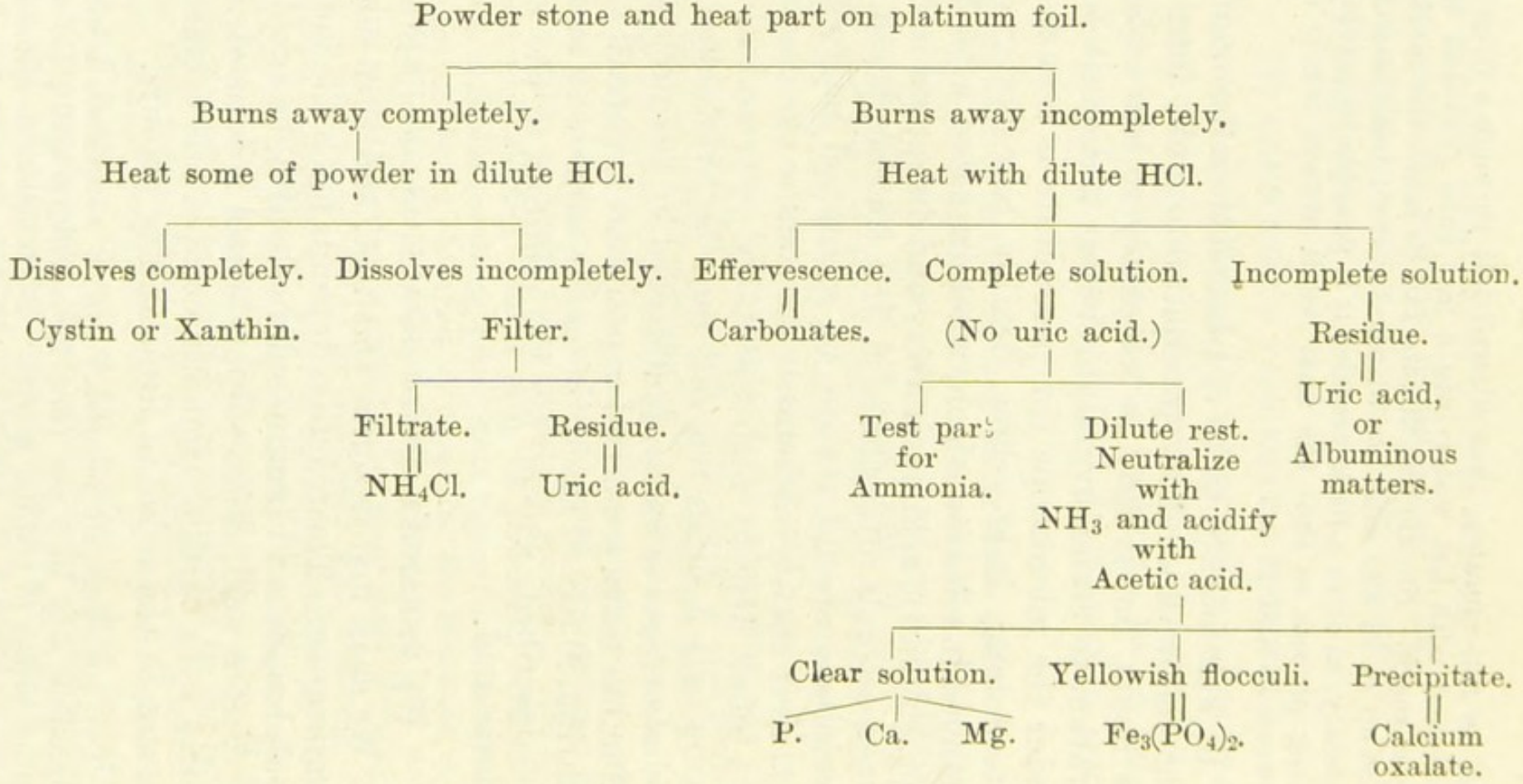
In well-marked cases of pancreatic inflammation a light-yellow flocculent precipitate should form in a few hours, but it may be necessary to leave the preparation to stand overnight before a deposit occurs. Under the microscope the precipitate is seen to consist of long, light-yellow, flexible, hair-like crystals, arranged in sheaves which, when irrigated with 33 per cent. sulphuric acid, melt away and disappear in ten to fifteen seconds after the acid first touches them. The precipitate should always be examined microscopically, as it may be difficult to determine the characters of a small deposit with the naked eye, and so cases giving only a slight reaction may be overlooked. To exclude traces of sugar, undetected by the preliminary reduction tests, a control experiment is carried out by treating 30 c.c. of the urine in the same way as in the test described, except for the addition of the hydrochloric acid.

(9) EHRLICH'S DIAZO REACTION IN URINE

We shall first describe this test, and then state its significance. The reaction depends upon the fact that if sulphanilic acid (amino-sulpho-benzol) be acted upon by nitrous acid, diazo-sulpho-benzol is formed, which unites with certain aromatic compounds occasionally present in the urine to form aniline colours.

Two solutions are necessary—(*a*) a saturated solution of sulphanilic acid in 5 per cent. hydrochloric acid; (*b*) a ½ per cent. solution of sodium nitrite. Both solutions should be as fresh as possible.

Scheme for Analysis of Urinary Calculi. (*After Salkowski*)



(*a*) Add to some urine in a test tube an equal quantity of (*a*); then add three drops of (*b*), and shake till a froth forms. Render alkaline with ammonia. If the liquid becomes of a port-wine colour while the froth is also red, the reaction is positive.

The test has the following significance :—

i. If the urine of a supposed typhoid in the second or third week fails to give the reaction, the diagnosis is probably wrong. In very mild cases, however, the reaction may be absent.

ii. The reaction is present in measles, but not usually in German measles (rötheln). It is thus of value in distinguishing between the two.*

iii. It is very constantly present in tuberculous disease which is advancing rapidly.

III. MICROSCOPICAL EXAMINATION OF URINARY DEPOSITS

The examination of deposits in urine and other fluids liable to decomposition is greatly facilitated by using a centrifuge. For ordinary clinical purposes, Beck's is perhaps as convenient as any ; where a large number of examinations are undertaken it is better to employ one driven by electricity or water power.

Unorganized Deposits

The first group of urinary deposits includes the various salts and crystalline substances that are found in urine, either when freshly voided, or more often when it has stood for some time. The following occur in **acid** urine (Fig. 89).

i. **Uric acid.**—This appears under a variety of forms, and, unless the urine is almost devoid of colouring matter, assumes a reddish-brown colour in consequence of its absorbing a considerable amount of

* Dr. Lloyd Jones informs us that he has found the reaction in not a few cases of German measles.

pigment. To the naked eye the appearance resembles that of a shower of grains of cayenne pepper collected at the bottom of the specimen. Under the microscope the crystals are either rhombic prisms or some modification of that form. Often the more obtuse angles are rounded off and the edges continued in curved lines, so that pointed oval shapes result. Numerous crystals may be joined together to produce rosettes and other composite forms. Some of the more common are represented in the accompanying figure (Fig. 90).

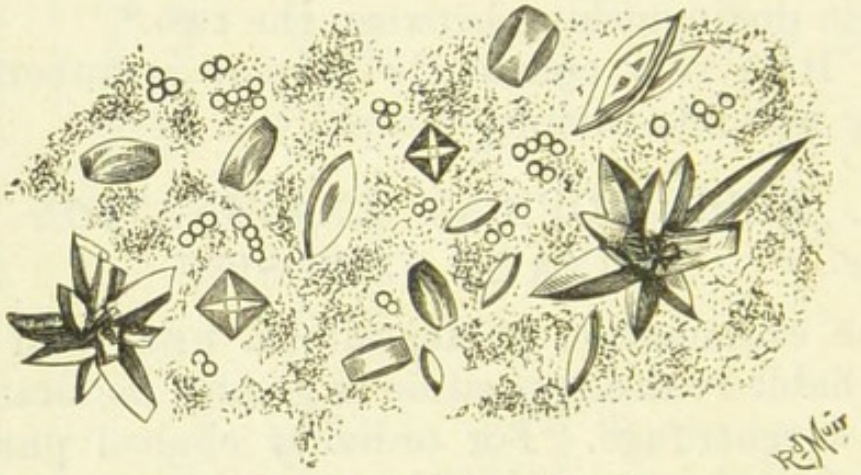


Fig. 89.—Deposit in acid urine.

2. **Urate of soda** occurs rather frequently in the urine of recently born infants, when it produces a yellow stain on the napkin. In adults it is found very seldom. The appearance presented under the microscope is that of spheres, either solitary or in clusters, having a more or less crystalline structure, and possessing numerous spines radiating from their surface (Fig. 91).

3. **Amorphous urates** ($MH\bar{U}$, $H_2\bar{U}$).—These are urates of potassium, sodium, and ammonium. They have a considerable affinity for the urinary pigments, and hence are generally more or less pink or brick-coloured. In very pale urines they are colourless, and resemble rather closely a deposit of phos-

phates. Microscopically they consist of small granular particles, arranged in moss-like clumps. On heating a urine from which they have separated out, they will be found to redissolve before the boiling-point has been reached.

Uric acid and urate of soda can be preserved in Canada balsam—the water being got rid of by passing

Fig. 90.—**Uric acid.** (*Finlayson, after Funke.*)

them through alcohol, then letting a drop dry on the slide, and adding balsam in xylol.

4. **Hippuric acid** appears in human urine chiefly after the administration of benzoic acid or its salts. It occurs as colourless four-sided prisms, insoluble in hydrochloric acid, but soluble in ammonia.

5. **Oxalate of lime.**—This deposit is rarely abundant. The small colourless crystals lying on the top of the mucous deposit that settles at the bottom of the urine glass give the impression of an undulating

snowy surface. They also adhere to irregularities on the surface of the glass, producing the appearance of scratches. Two forms are found under the microscope. The first, which is by far the commoner, consists of small octahedral crystals. When, as is generally the case, they are slightly flattened along one axis, they appear like squares crossed by two diagonal lines, or like long octahedra, according as the short axis lies in or perpendicular to the line of sight. The other form in which oxalates occur is that of minute dumb-bells or oval biscuit-shaped discs. Some writers consider that this form is not really due to oxalate, but to

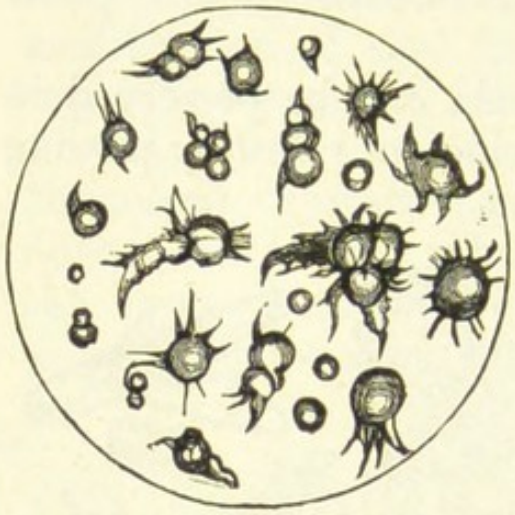


Fig. 91.—Urate of soda. (*After Roberts.*)

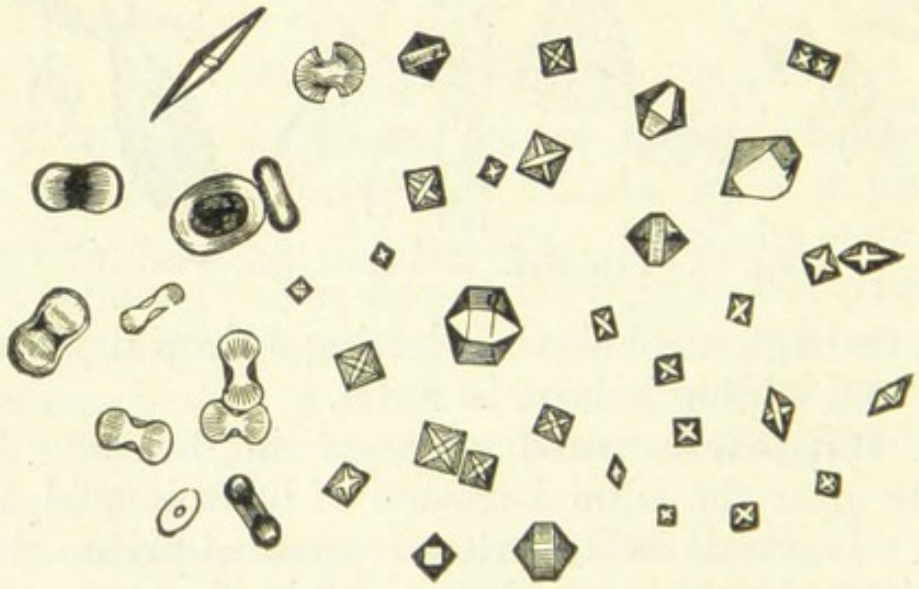


Fig. 92.—Oxalate of lime. (*After Finlayson.*)

carbonate of lime ; yet, though carbonates frequently enough assume this shape, there can be little doubt that under certain conditions oxalates do so too. For

permanent specimens octahedral oxalates are best mounted in glycerine jelly, dumb-bell oxalates in balsam (Fig. 92).

6. **Cystin** is a rare deposit in human urine, but when it occurs the precipitate is often copious, and is not unlike a sediment of fawn-coloured urates. The addition of a few drops of acetic acid to a urine containing cystin in solution determines its precipitation. From urine it is deposited as hexagonal tablets, soluble in ammonia, and, when the ammonia

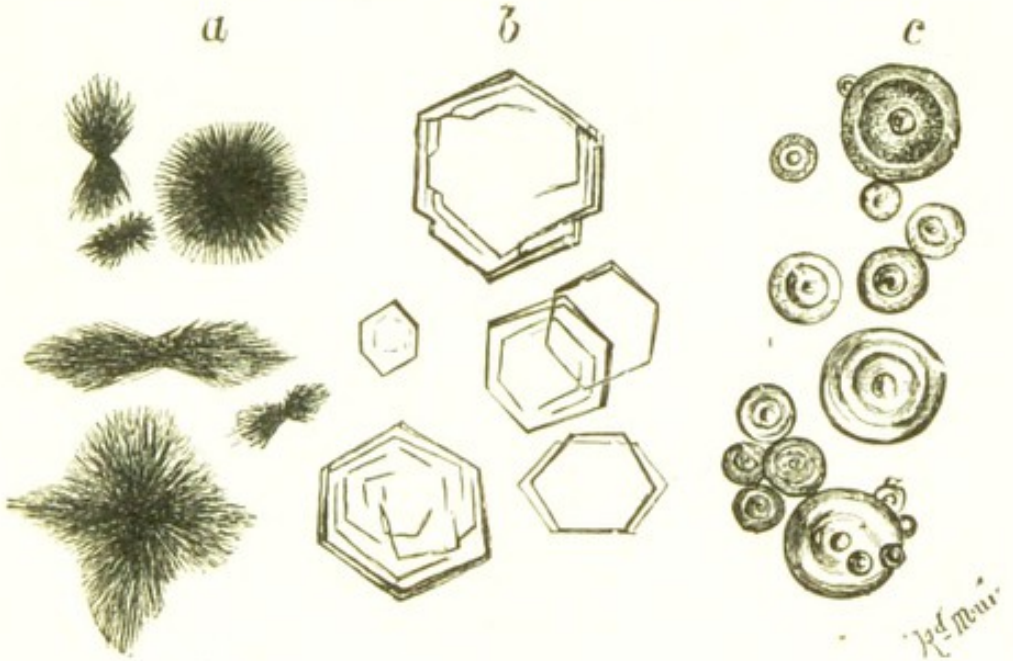


Fig. 93.—*a*, Tyrosin crystals; *b*, cystin; *c*, leucin.

evaporates, recrystallizing as hexagons or prisms (Fig. 93, *b*).

7. **Xanthin** is of extremely rare occurrence; the crystals are said to be similar to "whetstone" crystals of uric acid, but are soluble in ammonia, in warm hydrochloric acid, and in nitric acid.

8. **Tyrosin** is generally found associated with leucin, but occurs independently also. It forms colourless sheaves of fine needle-like crystals. A similar appearance may be presented by several other deposits; therefore, if there be any doubt as to the

nature of the sediment, a chemical analysis may be necessary (Fig. 93, *a*).

9. **Leucin** occurs in urine as yellow spherical masses without obvious crystalline structure. Leucin and tyrosin are found together in acute yellow atrophy of the liver (Fig. 93, *c*).

In **alkaline** urine the following occur :—

1. **Phosphates.**—These may be salts of phosphoric acid and calcium, or of phosphoric acid with ammonium and magnesium.

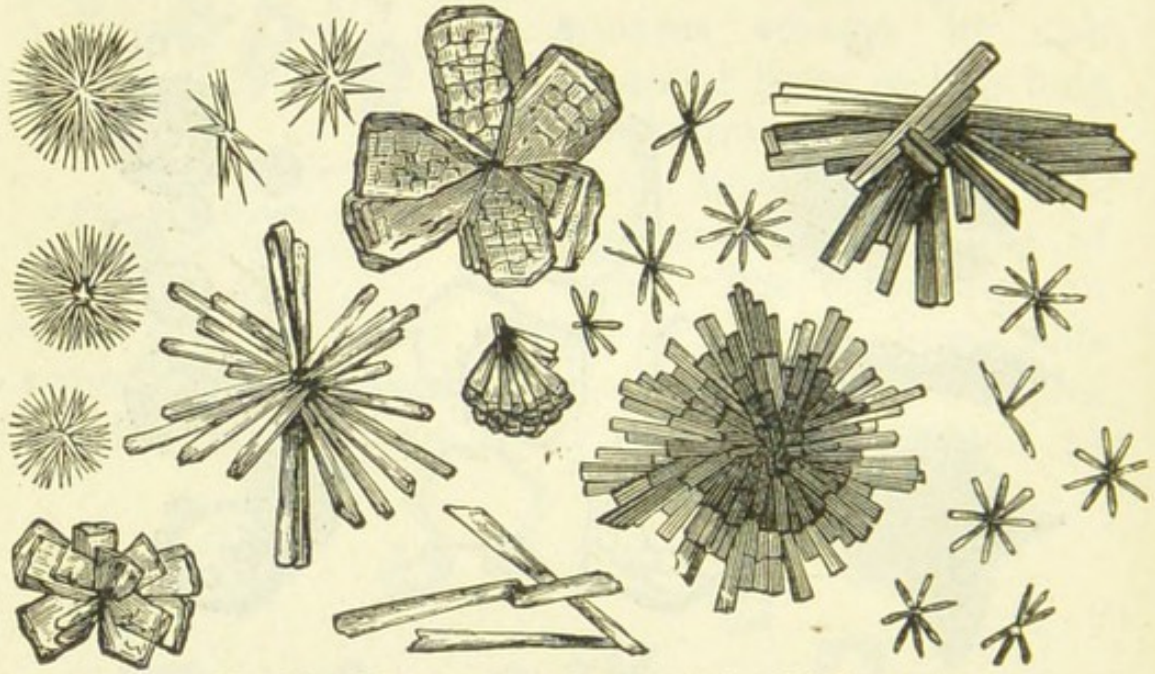


Fig. 94.—Stellar phosphates. (*After Finlayson.*)

(*a*) **Phosphate of lime** is found either in an amorphous or in a crystalline form, the latter being also known as *stellar phosphates* (Fig. 94).

Amorphous phosphate of lime occurs in small white granules, as a deposit at the bottom of alkaline urine. To the naked eye the sediment is white and flocculent; unlike urates, it has no affinity for urinary pigment. The deposit is increased on heating.

Stellar phosphates are rather uncommon. They consist of colourless prismatic crystals, which occur either singly or more often in radiating clusters. They

are found in very faintly acid as well as in neutral and alkaline urine. Roberts is inclined to regard the presence of this deposit in abundance as an accompaniment of some grave disorder.

(*b*) **Ammonium magnesium,** or **"triple" phosphate,** is deposited in ammoniacal states of the urine. To the naked eye the sediment appears very white, and when the crystals are large they may be visible as bright points. Sometimes the deposit also clings

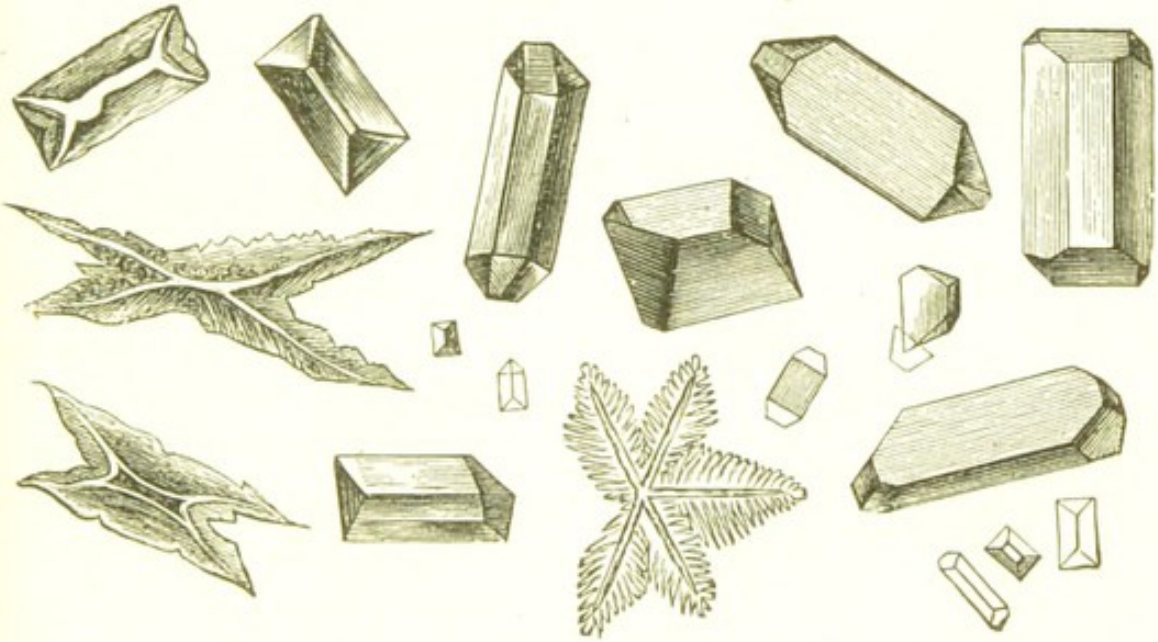


Fig. 95.—Triple phosphates. (*After Finlayson.*)

to the sides of the glass and forms a film on the surface of the urine.

The crystals are incomplete, triangular, colourless prisms, which may offer considerable variations in appearance, according to their length and degree of perfection. Often they are described as "knife-rest" or "coffin-lid" crystals. If the ammoniacal change is well marked, and still more if excess of ammonia is added to healthy urine, the deposit takes the form of feathery stars, and is then known as a precipitate of "feathery" phosphates (Fig. 95).

It is difficult to keep these crystals permanently,

but they may be preserved fairly well in a solution of ammonium chloride.

2. **Urate of ammonia** ($MH\bar{U}$) occurs in alkaline urine, and is very commonly present in cases of cystitis. Microscopically, it occurs in small spherical masses, which are practically indistinguishable in many instances from those of urate of soda, except that they are generally darker and more opaque, and, unlike the soda salt, are associated with crystals of triple phosphate. The spheres may have smooth surfaces, or they may be beset with innumerable spiny processes (Fig. 96).

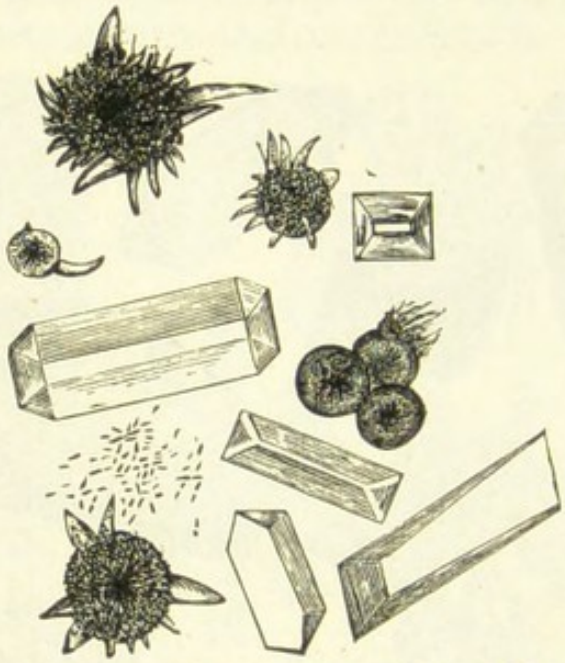


Fig. 96. — Deposit in alkaline fermentation of urine, showing urate of ammonia, triple phosphates, and Bacterium ureæ. × 200.

3. **Carbonates** generally occur in human urine as granular particles, which dissolve in acetic acid with evolution of CO_2. As phosphates give off no gas on solution in acetic acid, it is quite easy to distinguish between them. On rare occasions in human urine, and commonly in horses' urine, carbonate of lime appears in the form of dumb-bells or of spheres with a radiating crystalline structure.

4. **Cholesterin** has occasionally been found in the urine ; it occurs in characteristic thin, rhomboidal, colourless plates, with a notch at one of the corners (*see* Fig. 140, p. 583).

Other sediments, such as indigo, lime and magnesia soap crystals, and hæmatoidin, have been observed, but are of little importance.

Organized Deposits

1. **Red blood-corpuscles** are present in cases of hæmaturia, but under certain conditions are rapidly disintegrated, and should therefore be examined for in recently voided urine. According to the density of the urine they appear fairly normal, or swollen, or shrunken and crenated.

2. **Leucocytes** and **pus corpuscles** occur where there is irritation and suppuration of the urinary tract. According to the length of time which has elapsed, the cells may be indistinguishable from ordinary leucocytes, or they may be very granular and fatty. The addition of acetic acid clears up the cell body and brings two or three nuclei into view. Where pus is present, examine carefully for pathogenic microbes, especially for gonococci and tubercle bacilli.

3. In cases of **chyluria** the urine contains nucleated granular corpuscles similar to leucocytes, and very finely divided fatty material, which appears simply granular under the microscope. A few red blood-corpuscles are often present. The urine and blood should be carefully examined for the presence of filariæ, particularly if the patient comes from a district where these parasites exist.

In **lipuria** the fat may occur in larger globules which refract light strongly, and which are sometimes free in the fluid, at other times enclosed in cells or tube casts.

It must not be forgotten that fatty matter may reach the urine unintentionally from an oiled catheter, or may be added purposely in the form of milk by a patient who wishes to deceive the physician.

4. **Epithelium** from various parts of the urinary tract may be found in the urine. The following varieties are readily recognized :—

(*a*) *Renal epithelium.* This is polygonal, nucleated, and rather larger than a leucocyte. It may present fatty degeneration, or be more or less disintegrated (Fig. 97).

(*b*) *Epithelium from the bladder and urinary passages* presents various appearances, according to whether it is derived from the more superficial or the

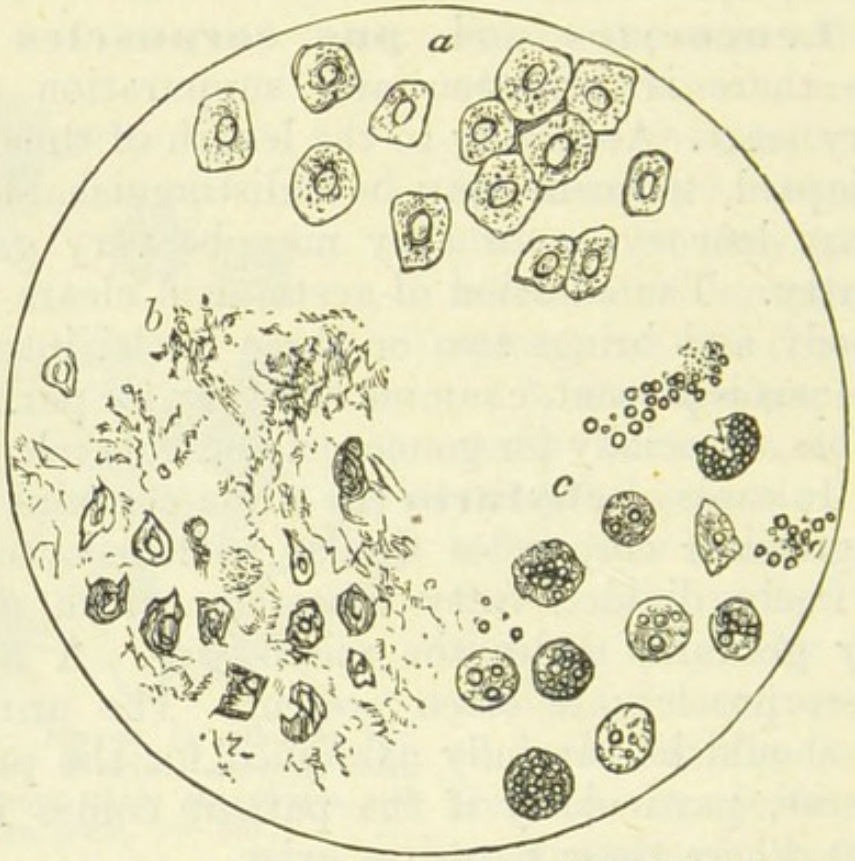


Fig. 97.—Renal epithelium. (*After Roberts.*)
a, normal; *b*, disintegrated; *c*, fatty. × 230.

deeper layers. Formerly, tailed cells were thought to indicate implication of the pelvis of the kidney; this is, however, inaccurate. They may equally well proceed from the deeper layers of the bladder epithelium, as may be seen from the accompanying diagram (Fig. 98).

(*c*) *Vaginal epithelium* is very commonly present in the urine of women. It is squamous, and the large cells appear sometimes singly, at other times in groups.

5. **Spermatozoa** occur at times in the urine,

where their characteristic appearance makes it easy to recognize them.

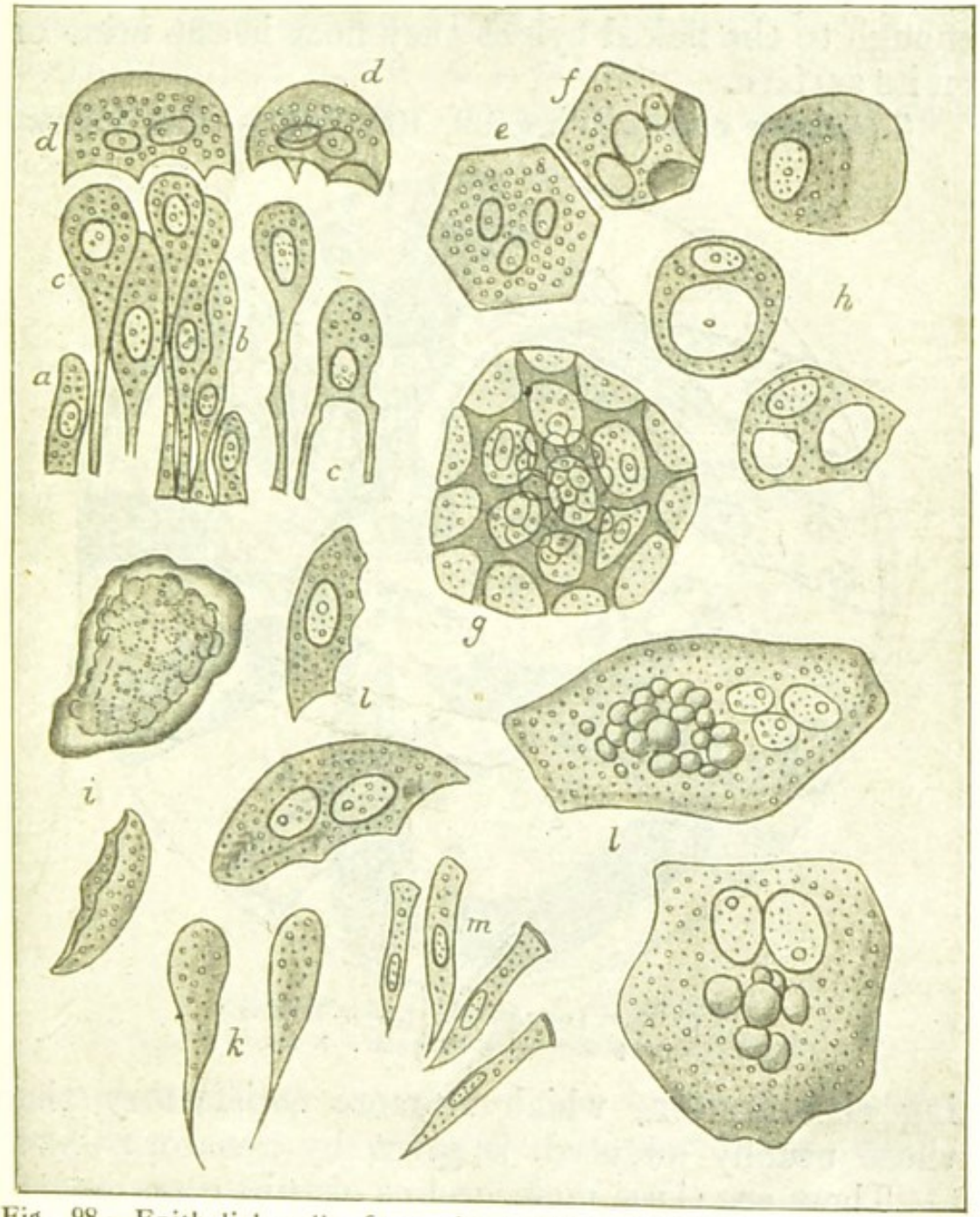


Fig. 98.—Epithelial cells from the urinary passages. (*From Sahli's "Klinische Untersuchungs-Methoden," after Bizzozero.*)

a, cells of the deepest layer; *b*, long cells of the second layer; *c*, "tailed" cells; *d*, flat cells of superficial layer; *e*, *f*, *g*, cells of superficial layer in surface view, with nuclei and indentations; *h*, *i*, *k*, *l*, epithelium from bladder, altered by action of urine; *m*, cells from male urethra.

6. **Prostatic threads** are found when there is chronic inflammation of the prostate, especially after

gonorrhœa. They consist of mucus, and are mostly voided with the first portions of the urine. They are much larger than tube casts, being visible readily enough to the naked eye as they float in the urine or on its surface.

7. **Tube casts** (Figs. 99, 100, 101).—The follow-

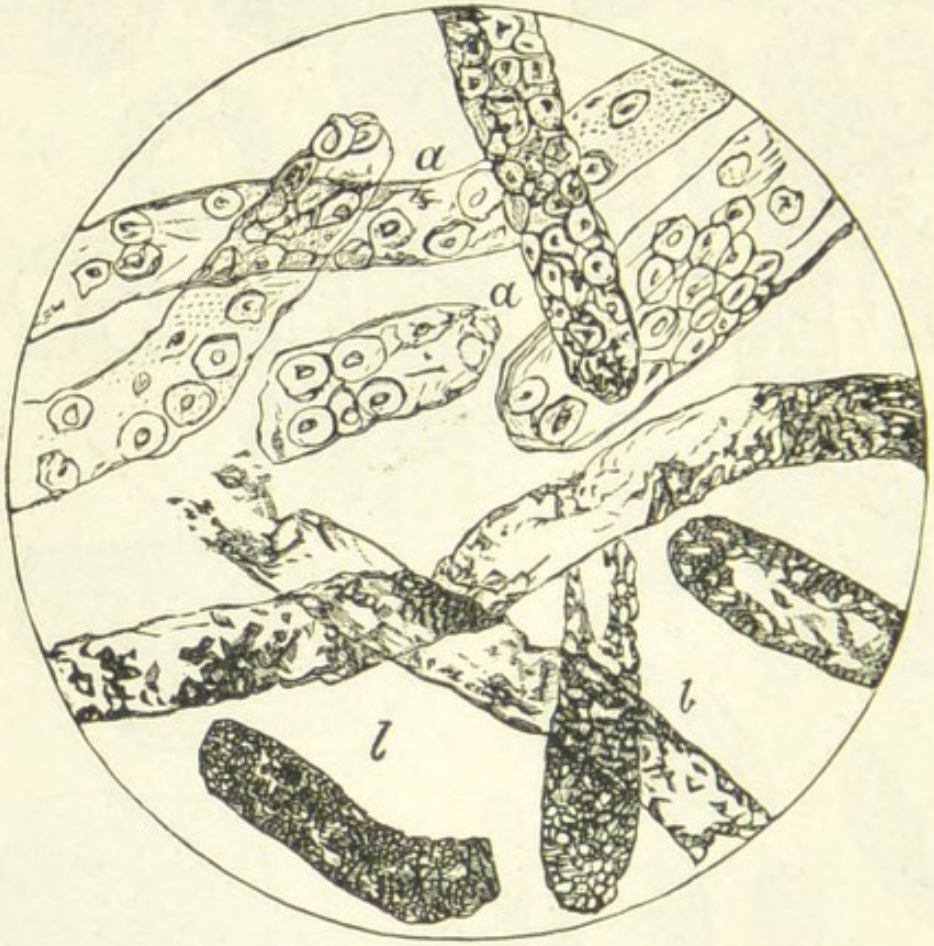


Fig. 99.—Tube casts. (*After Roberts.*)
a, epithelial; *b*, granular. × 230.

ing classification, which is more satisfactory than those usually adopted, is given by Senator:—

There are three main groups of tube casts:—

i. Casts wholly or mostly composed of cellular structures.

ii. Granular casts.

iii. Amorphous casts, having a homogeneous structure, and occasionally striated on the surface.

i. **Cellular.**—The cells may be epithelial or composed of red blood-corpuscles or leucocytes.

(*a*) *Epithelial.*—The casts may be completely covered with epithelial cells, as though the whole epithelium had scaled off a tubule, or the cells may have been separately detached and subsequently

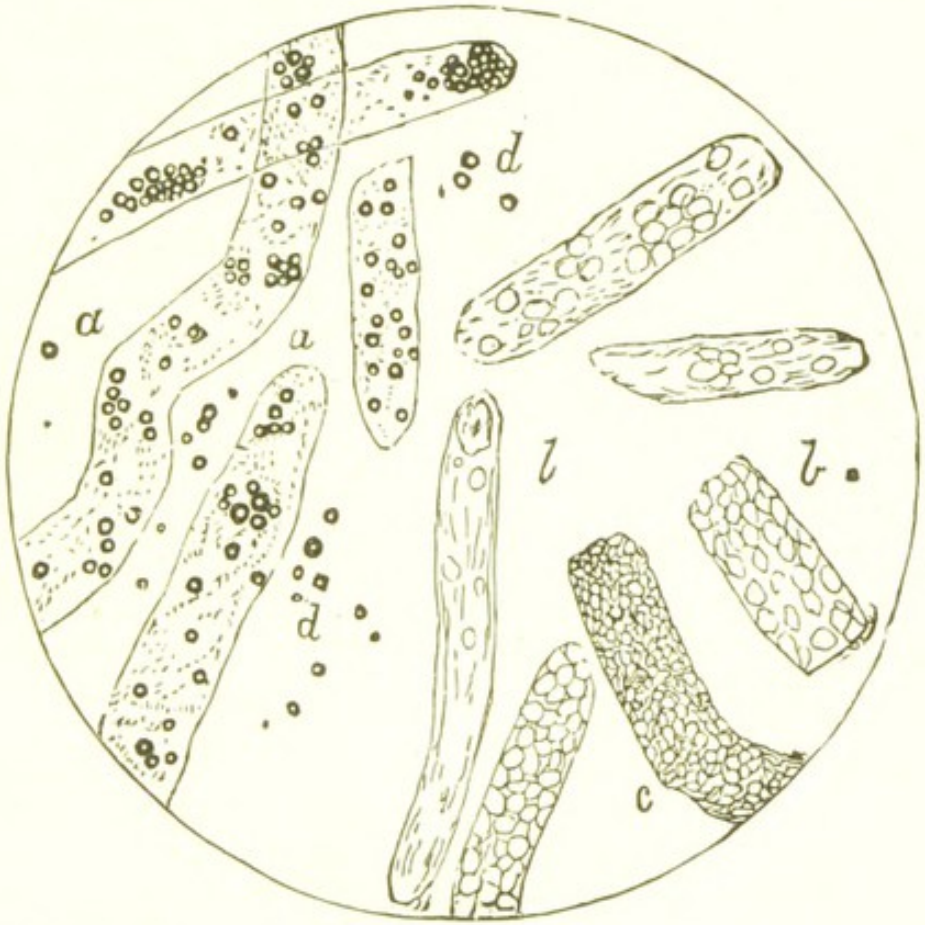


Fig. 100.—Tube casts. (*After Roberts.*)
a, fatty casts; *b*, *c*, blood casts; *d*, free fatty molecules. × 230.

moulded. The cells may or may not show a nucleus, and they may appear fresh, or affected by granular or fatty degeneration.

(*b*) The *red blood-corpuscle* casts exhibit a surface thickly covered with the minute round corpuscles.

(*c*) *Leucocytes* rarely form casts by themselves, but are fairly often found adhering to the surface of other casts.

ii. **Granular.**—The granules are sometimes coarse,

at other times fine. They are sometimes fatty, at other times they result from granular degeneration of protoplasm. They represent in some instances the relics of broken-down epithelium, in other cases they result from a granular change occurring in old amorphous tube casts.

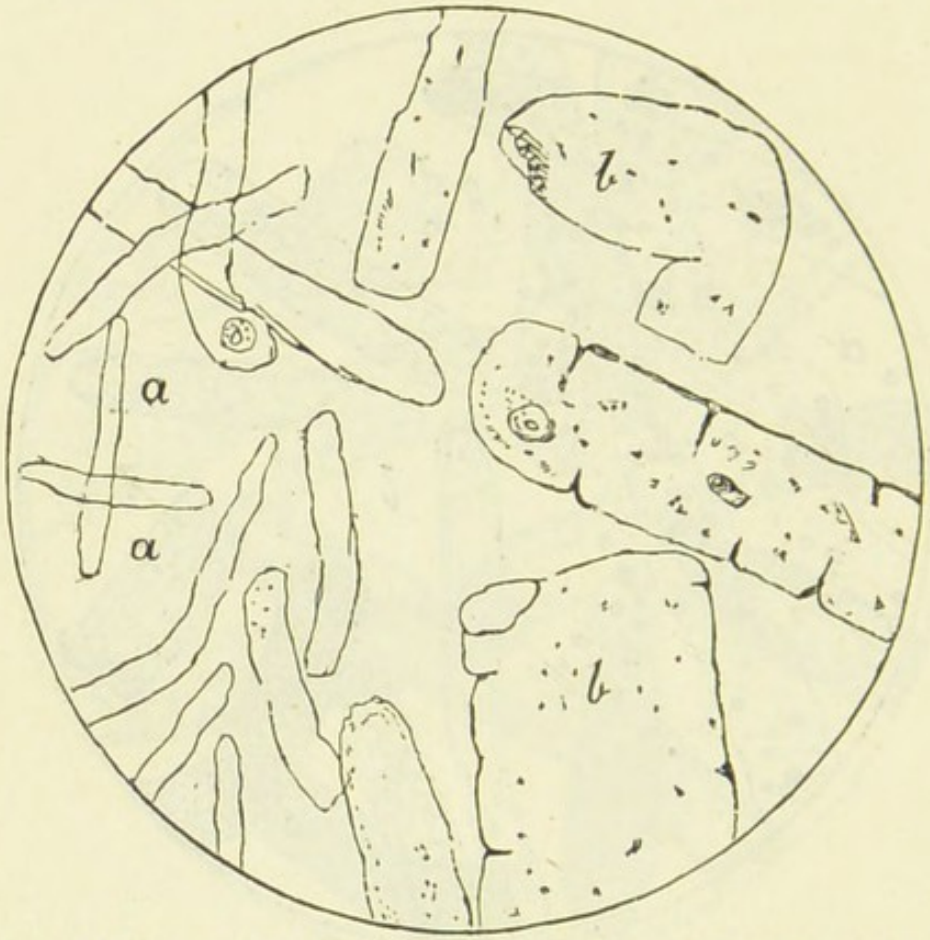


Fig. 101.—Hyaline (*a*) and waxy (*b*) tube casts. (*After Roberts.*) × 230.

iii. **Amorphous.**—This group contains two varieties, the hyaline and the waxy.

(*a*) *Hyaline* tube casts are pale, transparent, and homogeneous. Occasionally the surface is striated. They may be almost invisible, but are rendered more prominent by the addition of iodine solution. Their origin has been variously accounted for. Senator believes that in nearly all cases they are derived from epithelium which has undergone hyaline degeneration, or has yielded a secretion that is coagulable.

(*b*) *Waxy* casts are broader and more highly refractile than hyaline. Often they are more or less fissured. Possibly they may be formed from other casts which have remained long in the urinary tubules; they are *not* symptomatic of waxy disease of the kidney. Sometimes they give the amyloid reaction with iodine and sulphuric acid and with methyl violet.

Transition forms between the various groups are not uncommon; often, for instance, a cast is partly epithelial and partly hyaline.

A tube cast frequently picks up adventitious elements from the urine, and thus comes to contain bacteria, or crystals such as oxalate of lime.

In length, tube casts are very variable; occasionally they approach 1 mm. in length. One end may be spirally twisted, or in rare cases bifurcated. Formerly tube casts were called "fibrin cylinders," but now it is proved that fibrin rarely or never enters into their composition, the only exception being that in the case of red blood-corpuscle casts the blood discs may be bound together by a little fibrin.

It appears that tube casts, including their matrix, are essentially products of the cells which line the kidney tubules, and that the relative proportion of epithelial, granular, and hyaline casts is greatly influenced by the reaction and constitution of the fluid which bathes them. An acid urine yields, as a rule, granular casts, but, if the acidity is very high, casts which appear hyaline. An alkaline urine renders the majority of the casts hyaline. On the other hand, the salts of the urine, apparently by precipitating colloid material, tend to preserve the granular aspect of the casts, and to make them smaller by causing the colloid material to lose water.*

Structures called **cylindroids** have been described by Thomas and others. They resemble extremely long and narrow tube casts, but are usually considerably flattened. Some observers regard their presence

* Fischer, "Nephritis," p. 84 *et seq.* New York, 1912.

as quite immaterial, others looking on them with considerable suspicion as being nearly related, both in origin and clinical import, to tube casts.

Not to be confounded with either tube casts or cylindroids are the small strings of mucus which occasionally are present in a urinary sediment. Small clumps of micrococci and short so-called "prostatic threads" are also liable to be misinterpreted by an inexperienced observer. When there is reasonable cause for doubt, the addition of acetic acid or some other reagent will often settle the question.

To preserve tube casts permanently for examination, the writers have found the following methods the most serviceable :—

(*a*) Collect some of the sediment containing the casts, wash rapidly in water, and drop into a conical glass containing picrocarmine strain. When the sediment has collected at the foot of the glass, which will have occurred in twelve hours or so, wash the deposit once more in water, and drop it into a small quantity of Farrant's medium in a conical glass. It will gradually sink in this, and, after a day or two, samples can be removed from the foot of the fluid, and be permanently mounted in some of the fluid from which they have been withdrawn.

(*b*) Harris proposes the following method :—

Let urine stand for twelve hours, then remove deposit, and place in a pipette whose upper end is closed by a rubber stopper. The pipette should contain the following preservative fluid :—

Pot. acetate	60 grm.
Chloroform	10 c.c.
Distilled water	1,000 c.c.

When the sediment has sunk through the fluid it may be transferred to a slide along with a drop of the preservative, and permanently sealed there. Specimens keep fairly well.

(*c*) Boston's method is as follows :—

Collect some of the precipitate, wash it at least once with water to which a few drops of chloroform have been added, place a drop of the washed precipitate on a slide and examine under the microscope. If casts are present, evaporate the drop

nearly to dryness, add a drop of the mounting medium to the centre of the drop of urine, mix gently with a fine needle (avoiding the introduction of bubbles), moisten a cover-glass with the breath and allow it to fall gently on the specimen, lay aside for a few hours to harden, and then ring with zinc white.

The mounting medium is prepared as follows :—

Liquor. arsenicalis.	℥i.
Ac. salicyl.	gr. ½.
Glycerine	℥ii.

Warm slightly and add tears of gum acacia to saturation, then decant the clear supernatant fluid. A drop of formalin may be added if necessary. Specimens prepared by this method keep good for years.

The use of a centrifuge is of great value for securing tube casts in perfectly fresh urine, and for washing them rapidly in water prior to preserving them.

8. Tumours of the bladder, especially when villous, may often be detected by the presence of **fragments** of the growth in the urine. These show a core of connective tissue with its blood-vessels, coated with several layers of nucleated epithelial cells. In cancerous tumours, though their débris is commonly enough present, yet nothing at all so characteristic is to be seen as is found in the papillomata.

9. **Elastic fibres** are often present in cases of ulceration of the bladder. They may be detected either without special treatment, or after the use of caustic soda (as was described in Chap. VI.), preceded by a preliminary filtration of the yet acid urine to remove the phosphates, which would otherwise be precipitated copiously when an alkali was added.

Senator enumerates the following **parasites** of the urinary tract : Echinococcus, Cysticercus cellulosæ, Eustrongylus gigas, Distōma (or Bilharzia) hæmatobium, Filaria sanguinis hominis, Nephrophages sanguinarius (a member of the arthropoda related to the acari), rhabditis (sp. ?), and certain psorosperms. Several of these are so rare as to be of no practical

importance; echinococcus, cysticercus, and filaria having been described elsewhere,* Distōma hæmatobium alone needs to be referred to here.

D. hæmatobium.—The ova measure 0·12 mm. by 0·04. A spine projects at one pole or at a little distance from it. In urine the spine is usually situated at the pole; the form with a lateral spine predominates in ova obtained from the rectum (Fig. 102).

Fig. 102.—Ova of Bilharzia hæmatobium in urine. (*After Roberts.*)
a, × 50, in mucus; *b*, × 100, in urine freshly voided.

The adult male bilharzia is thicker and shorter than the female, and is provided on the ventral surface with a gynæphoric canal. The female is cylindrical and worm-like. The male measures 12 mm., the female about 16 mm., in length. Their habitat is in the blood-vessels of the portal system and in the venous plexuses of the bladder and rectum. The ova escape from the blood-vessels into the tissues of the body. Those which reach the rectum and bladder are discharged, and enable a diagnosis to be made. The parasite is very common in Egypt.

After the urine has been voided for some time, it

* Pp. 103 and 227.

becomes contaminated by numerous non-pathogenic fungi and infusoria ; but several pathogenic bacteria occur in the urinary tract, and in cases of doubt should always be sought for. The chief of these are the gono coccus, the tubercle bacillus, which must not be mistaken for the morphologically similar smegma bacillus, and the Bacillus coli communis.*

In cases of cystitis a great variety of bacteria may occur. Actinomyces, though rarely present in the kidney, has been found there.

Foreign bodies often occur in urine which has been set aside for examination. Besides hairs, feathers, moth-wing scales, cotton, woollen, and silk fibres, starch grains derived from dusting powders (readily recognized by their turning blue on the addition of a little dilute tincture of iodine), and, more confusing than any of these, pinewood dust swept from the floor, one occasionally finds fragments of the contents of dermoid tumours or abscesses that have opened into the bladder or ureter. Small shreds of striped muscle may in rare instances be voided with the urine, and are derived, in some cases at least, from a sloughing psoas abscess.

It may happen also that the patient has been sick, and sputum or vomited matter may be more or less abundantly mixed with the urine.

* For further information *see* Chap. XIV., pp. 600 and 511.

CHAPTER VIII

THE SKIN

For the examination of the skin and its appendages the patient should be stripped as completely as circumstances permit and placed in a good light.

One should first note the **colour** of the skin as a whole. In anæmia the skin is pale ; in chlorosis it has a greenish tint ; in pernicious anæmia it is lemon-yellow. In order to distinguish the yellowness of pernicious anæmia from jaundice, look at the conjunctiva. The best way to do that is to place one hand on the patient's forehead, ask him to look at the ground, and then raise the upper eyelid with the thumb. In jaundice the conjunctiva is seen to be yellow where it covers the sclerotic ; in anæmia it is white. In judging of the degree of anæmia, one should be guided more by the colour of the mucous membranes than by that of the skin itself. The conjunctiva lining the lower eyelid is usually taken as an index. It is easily seen by getting the patient to look up while one depresses the lower lid with one finger. Instead of being pale, the skin may be abnormally red or *flushed*. The flushing may be general or local. Its exact extent should always be noted, and whether or not it fades on pressure. The best way of telling whether any redness of the skin fades on pressure, or not, is to place a lens on the skin and press it down. It will then be seen whether or not the skin becomes pale under the lens.

The term **tache cérébrale** is applied to the red flush which appears in some cases of intracranial

disease when the skin is stimulated. To elicit its presence, draw the finger-nail firmly across the patient's forehead. A red line soon develops along the track of the nail, and persists for some time. It is due to a disordered vasomotor supply, but is found in other conditions besides those of cerebral irritation, and is therefore not of much diagnostic value.

Taches bleuâtres are steel-blue spots, which may occur in large numbers, usually on the trunk and thighs. They are deeply placed in the skin, irregular in outline, and without diagnostic significance. They are probably always associated with the presence of pediculi.

Rarer alterations in colour of the skin are those due to the taking of nitrate of silver and those which occur in Addison's disease. The former constitutes what is known as **argyria.** It consists in a leaden-grey hue of the whole skin, which is unaffected by pressure. The pigmentation of **Addison's disease** consists in a bronzing, which appears first on parts in contact with the air, and next on those which are exposed to pressure. It is made up of small brownish spots, which fade off at their margins into healthy skin. The lips and buccal mucous membrane should always be examined in cases of supposed bronzing. In Addison's disease they often exhibit marks of pigmentation of a dark bluish-black colour, which have been compared to the stains produced by sucking a pen. Diffuse pigmentation of the skin is also a common occurrence in pregnancy (chloasma), and in many cases of pulmonary tuberculosis, and also after the prolonged administration of arsenic.

Having noted any alteration in the colour of the skin, one should look for the presence of any **eruption.** If any such be observed, the patient should be questioned about it on the lines laid down on p. 10.

The exact situation and extent of the eruption should be noted, and whether it is symmetrical or confined to one side only. These general facts having been noted, one should pass to a description of the minute characters of the eruption. In order to do this, it must be borne in mind that every cutaneous eruption consists of a **primary lesion,** to which secondary lesions may or may not be superadded. The following is a description of the different primary lesions which may be met with :—

1. **Macules** or spots.—Any abnormal change in the colour of the skin confined to a limited area. Always note whether or not they fade on pressure. The spots of typhoid fever, for example, fade on pressure, whilst those due to hæmorrhages into the skin, as in the bites of fleas, do not.

2. **Papules.**—Solid projections above the surface which are not larger than a pea. The term **tubercle** or **nodule** is applied to any solid projection from the skin which is larger than a pea, but not larger than a cherry. Anything larger than that is called a **tumour.** Always note whether the top of a papule is rounded as in some forms of eczema, pointed as in acne, or flattened as in lichen. As regards the base, observe whether it infiltrates the skin widely or not. The wider the infiltration, the more extensive and severe the inflammation.

3. **Vesicles.**—Elevations of the horny layer of the epidermis by transparent or milky fluid which are not larger than a pea. If larger than that they should be described as **bullæ** or **blebs.** Always note whether or not there is an area of redness around the base of a vesicle, for such redness indicates that the vesicle is planted upon an inflamed base—a fact which may be of diagnostic value.

4. **Pustules.**—Small elevations of the skin con-

taining pus. Always observe whether there is much infiltration around them or not.

5. **Wheals.**—Slightly elevated portions of skin, the centre of which is paler than the periphery.

Having stated which of these primary lesions it is that composes the eruption, one should next note whether the lesions are isolated (discrete), or whether they run together. It must also be remembered that an eruption may be made up of more than one kind of primary lesion. Thus, papules may be mingled with pustules, or pustules with vesicles, and so on.

Next look for **secondary lesions.** These are either produced mechanically, or are the result of changes which take place in the primary lesion in the course of its growth or decline. The commonest secondary lesions of mechanical production are **excoriations** due to scratching, and **fissures** (rhagades)—deep cracks going down to or through the corium, and produced by the stretching of the skin after it has become inelastic owing to infiltration. Fissures are often very painful.

The following are the secondary lesions produced by changes in those which are primary :—

1. **Desquamation.**—If the primary lesion be a dry one (macules or papules), a mere scaling off of epidermic cells occurs, and the eruption is then said to be "*scaly.*"

In moist lesions (vesicles, pustules, bullæ) the epidermic cells become glued together by the dried fluid, and a **scab** or **crust** forms. The scab may be serous, purulent, hæmorrhagic, or sebaceous, according to the nature of the contents of the primary lesion.

2. **Infiltration** may occur around the primary lesions, leading to the production of a leathery feeling in the skin. This is usually the result of prolonged chronic inflammation.

3. **Pigmentation** may occur around the primary lesions. This is also usually due to prolonged inflammation.

4. **Ulceration.**—Due to breaking down of the primary lesions and destruction of a part of the true skin.

The points to note in describing an ulcer are (1) the nature of the floor of the ulcer and the granulations covering it; (2) the character of the edge—smooth, raised, undermined, etc.; (3) the discharge—whether serous, purulent, watery, fetid, etc.; (4) the character of the surrounding skin, whether indurated, pigmented, etc.

5. ***Scar* formation.**—This only occurs where the true skin has been involved, i.e. where there has been an ulcer. Describe the scar, noting especially whether it be thin or thick, freely movable or adherent to the deeper tissues, pale or livid, pitted or not, surrounded by a zone of pigmentation or not.

Proceed now to the **palpation** of the skin. Pass the hand gently over it, pinching it up between the forefinger and thumb, and note the following points :—

Is it smooth or rough, thin or thick, dry or moist ? If there be any visible sweating, note whether it is general or local; whether it is attended or not by any flushing of the skin; and whether the sweat has any particular odour.

The **elasticity** of the skin should be investigated. If a fold of healthy skin be pinched up, it immediately flattens itself out again when released. Sometimes, however, it only does so very slowly, remaining for a considerable time in a creased condition. This indicates a diminished elasticity. It occurs not infrequently in debilitated and in old persons. It may also be often observed in babies exhausted from

diarrhœa, and is then believed to indicate interference with the renal functions.

The condition of the **subcutaneous tissue** should also be investigated. It may be infiltrated with fluid (œdema), with solid material (as in myxœdema), or with air (subcutaneous or surgical emphysema). The presence of **œdema** is usually recognized by the fact that if the skin be pressed with the finger, especially over a hard body such as a bone, a pit is left which persists for some little time. It must be borne in mind, however, that this is not an invariable guide. In some cases of œdema no pitting can be produced. This is especially apt to be the case where the œdema is of very long standing. The best place to look for slight degrees of œdema in cardiac disease is behind the malleoli of the tibia and fibula. In chronic renal disease, œdema can often be earliest detected beneath the conjunctiva. This subconjunctival œdema is seen by pushing up the lower lid over the sclerotic. A little drop of fluid resembling a tear is then squeezed up underneath the conjunctiva over the sclerotic.

Subcutaneous emphysema gives rise on palpation to a crackling sensation, which has been compared to that which is experienced in handling a bag of feathers. It starts in, and is usually confined to, the neighbourhood of the air-passages or air-containing organs, and is due to the establishment of an abnormal communication between such a passage or organ and the subcutaneous tissue. In rare cases it may be of bacterial origin.

Microscopical examination of the skin and its appendages is confined to the diagnosis of some parasitic diseases, of which the following are the chief (Figs. 103, 104) :—

1. **Scabies or itch.**—This is due to the *Acarus*

(*Sarcoptes*) *scabiei*. The female acarus is larger than the male, and forms burrows in the skin, in which the eggs are deposited. These burrows should be looked for between the fingers and on the inner aspects of the wrists. They are recognized with the naked eye as little short dark lines terminating in a sort of shining spot of skin. The eggs lie in the dark line, the insect in the shining spot. It may be picked out by means of a flat surgical needle passed along the black line to the clear spot. The use of a lens aids the operation—which is by no means invariably successful—and permits of the recognition of the insect. The latter may be placed on a slide under the microscope for more minute inspection (Fig. 103, *e*).

2. **Pediculosis.**—Three varieties of pediculus occur—*Pediculus capitis* on the head, *P. corporis* on the trunk, *P. pubis* on the pubic and axillary hairs. The eggs or "nits" of *P. capitis* are stuck on to the hairs (Fig. 103, *b'*). From their position on the hairs one can judge roughly of the duration of the condition, for they are fixed on at first near the root of the hair, and are then carried up with the latter in its growth. The higher up the nits are, therefore, the longer have pediculi been present. *P. corporis* should be looked for in the seams of the clothes, especially where the latter come into close contact with the skin—e.g. over the shoulders. The bites of the parasite produce hæmorrhagic spots, each with a dark centre and a paler areola. Marks of scratching should always be looked for on parts accessible to the patient's nails.

The microscopic characters of the pediculi are shown in Fig. 103, and require no verbal description. It will be noticed that *P. corporis* is the longest and narrowest of the three, *P. pubis* is shortest and broadest; *P. capitis* is between the two in size. *P. pubis* is also distinguished from the others by being yellowish-

Fig. 103.—Animal parasites of the skin.

a, flea; *b*, Pediculus capitis; *b'*, ovum of ditto, with embryo ("nit") *c*, P. corporis; *d*, P. pubis; *e*, Acarus scabiei (female, male, ovum, larva); *f*, Demodex folliculorum.

brown in colour. *P. capitis* and *P. corporis* are both greyish in colour, though the latter varies considerably with the colour of the skin of its host. The shape of the thorax and abdomen forms a distinguishing character between *P. capitis* and *P. corporis* (Fig. 103, *b*, *c*, *d*).

3. **Ringworm.** — Investigation by Sabouraud and others has shown that two distinct classes of parasites are capable of producing the appearances which are included under the name "ringworm." One of these is not a trichophyton. It goes by the name of Microsporon Audouini, and is the cause of the commonest and most contagious and intractable form of the disease. Its operations are chiefly confined to the scalp, but 90 per cent. of all cases of ringworm in that situation are due to its presence. The other parasite belongs to the trichophyton family, but it is probable that there are several varieties of it, as there are also of the microsporon. It is the commonest producer of ringworm of the beard and skin (tinea barbæ and tinea circinata), but only occurs in about 10 per cent. of the cases of ringworm of the scalp. It succumbs much more readily under treatment than does the microsporon. The trichophyton may be situated mostly outside the hair (*ectothrix* or *endo-ectothrix*), or mainly in the hair substance (*endothrix*). Intermediate forms are also described. It is characterized microscopically by having, as a rule, fairly large so-called spores, which are arranged in chains, and the joints of its mycelium are placed at regular intervals. The microsporon has, on the whole, small spores, which are scattered irregularly, and the joints of its mycelium are at unequal intervals (Fig. 104).

A useful method of detecting hairs which are affected by ringworm consists in dabbing over the diseased patch with a piece of wool soaked in chloroform.

On evaporation of the latter, the affected hairs are whitened, and look as if covered with hoar-frost. They can thus readily be distinguished from healthy hairs

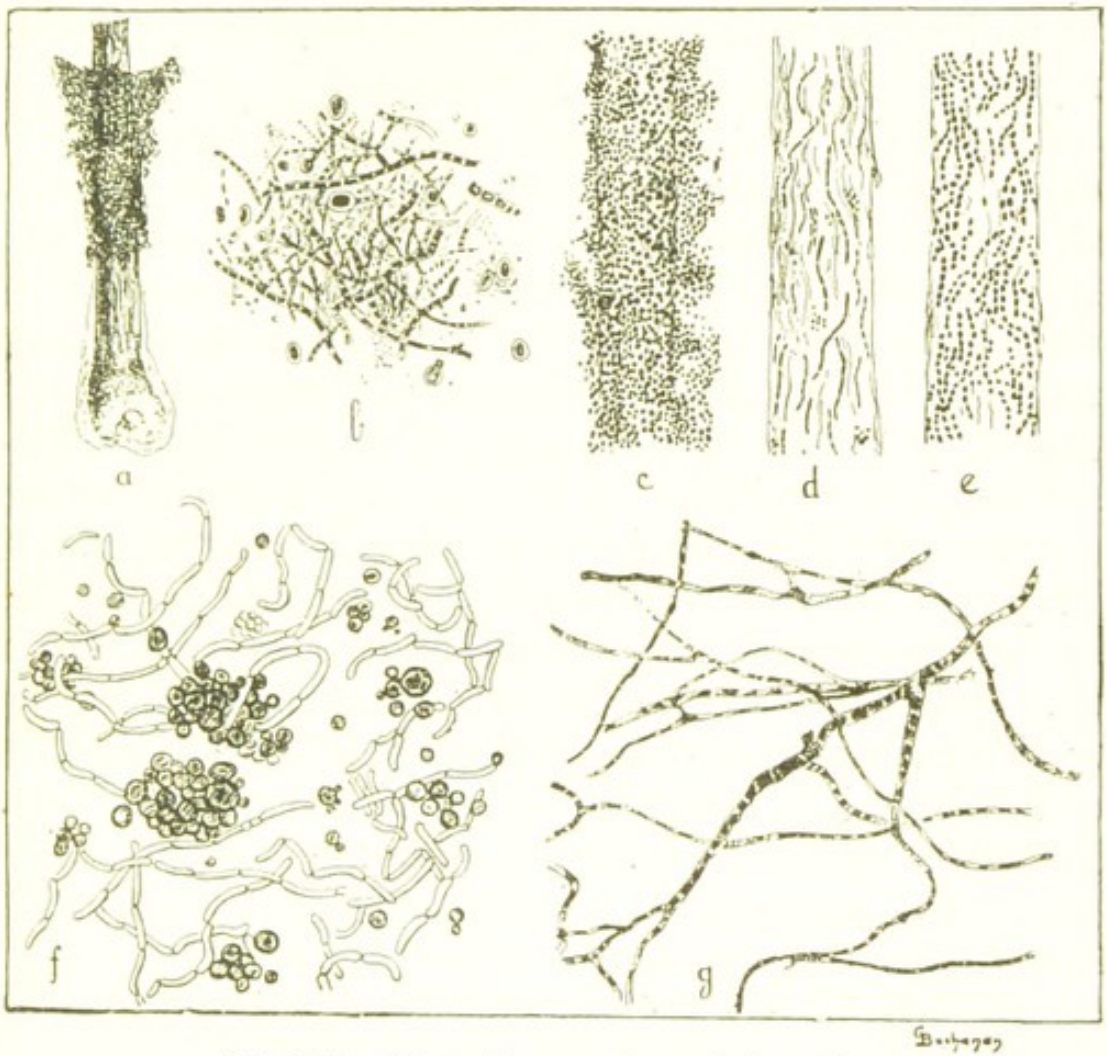


Fig. 104.—Vegetable parasites of the skin.

a, favus (Achorion Schönleinii), showing affected hair, × 70; *b*, favus (Achorion Schönleinii), showing fungus in a crust, × 290; *c*, ringworm (Microsporon Audouini) in hair, surface view, with mosaic of spores, × 200; *d*, ringworm (Microsporon Audouini) in hair, optical section, with mycelium, × 200; *e*, ringworm (trichophyton) in hair, with spores arranged in chains ("rosary" arrangement), × 200; *f*, Microsporon furfur (from pityriasis versicolor), spores in clusters, × 250; *g*, trichophyton mycelium, from a culture, × 250.

of the same size by the aid of a lens. Hairs affected by favus are not similarly whitened by chloroform.

Microscopical examination.—If one is dealing with a patch of ringworm of the skin, it is sufficient to scrape off some of the scales with a blunt penknife, to place them in a drop of 10 per cent. liquor potassæ,

and cover. The mycelium of the fungus will be recognized as branching, refractile threads, amid which the spores are scattered in groups or rows.

If a hair be similarly examined, it will be found to be broken up and full of spores (Fig. 104, *c*). No mycelium can be seen. For diagnostic purposes, in simple cases a broken hair should be extracted by broad-pointed forceps, traction being made in the axis of growth, so that the bulb may be obtained intact. It should be washed in ether, soaked for a quarter of an hour in liquor potassæ, a drop of glycerine then run under the cover-slip, and the edge sealed down with melted paraffin. The spores will be seen in the substance of the hair and in its sheath. Fatty particles are the only thing likely to be mistaken for them. A drop of ether will dissolve fat particles, but leaves the spores unaffected. Liquor potassæ, however, causes the spores to swell. Its use should therefore be avoided if one wishes to distinguish the two varieties of fungus. For that purpose staining is of great help. It should be carried out as follows :—

(1) Soak the hair for some minutes in ozonic ether to bleach it and to remove grease, and allow to dry on a slide.

(2) Steep for ten to thirty minutes in a mixture of 10 parts of a 5 per cent. alcoholic solution of gentian violet and 30 of aniline water (Appendix, 28).

(3) Dry between blotting-paper, and steep in Gram's solution for five minutes. This fixes the stain.

(4) Dry again, and soak for from ten minutes to some hours if necessary in pure aniline to which enough iodine has been added to give it a distinctly brown colour.* This decolorizes everything except the fungus, but the process should be controlled under the microscope.

(5) Wash in pure aniline for a few seconds, then in xylol, and mount in balsam.

Adamson's method of differential staining is briefly as follows :—

* Nitric acid may also be used as a decolorizer.

Place the hair in a drop of liquor potassæ, and cover. After twenty minutes run in some 15 per cent. alcohol (this hardens the hair). Remove the cover-glass, wash away any remaining liquor potassæ with more alcohol, mop up the latter, and dry over a flame. Stain in aniline gentian violet for half an hour. Place in Gram for three minutes. Decolorize in aniline oil for half an hour. When the fungus is clearly visible, and the hair no longer much stained, remove the oil with blotting-paper. Add a drop of concentrated alcoholic eosin, wash off with aniline in a minute, wash off latter with xylol, and mount in xylol balsam.

On examination with the high power the two varieties of fungus can be differentiated by the characters already described. It should be noted that the difference in size of the spores of the two varieties is really not very great after all. The arrangement of the spores and the character of the mycelium are the points to which attention should be directed.

4. **Favus.**—This disease, which is characterized to the naked eye by the production of yellow cup-shaped crusts or *scutula*, is caused by a parasite named, after its discoverer, the *Achorion Schönleinii*. The hair and accompanying crust may be examined in liquor potassæ, some pressure being exerted on the cover-glass in order to flatten out the mass of epidermic scales by which the parasite is apt to be obscured. The hair will be found completely filled with mycelium, and the medullary canal obliterated. The joints of the mycelium are branched, and often present a resemblance in shape to metacarpal and metatarsal bones. The spores are rather large, and arranged in rows or groups (Fig. 104, *a*, *b*).

5. **Tinea versicolor.**—This is produced by the *Microsporon furfur*. A scraping should be examined in caustic potash. The fungus shows a refractile mycelium which interlaces freely, and includes bunches of round spores in its meshes (Fig. 104, *f*). The spores may be stained with saffranin, differentiated by weak

acetic acid, and the mycelium counterstained with methylene blue.

6. **Demodex folliculorum** (Fig. 103, *f*) is a minute acarus, about $\frac{1}{120}$ in. in length, which is sometimes found in the sebaceous contents of comedones. It has a disproportionately large abdomen marked with transverse rings which give it at first sight the appearance of a minute worm. It possesses a suctorial proboscis and styliform jaws, and from the thoracic portion of the body four pairs of stunted legs project. It is simply a parasite living in sebaceous matter, and is of no pathological importance. It stains an intense red by the Ziehl-Neelsen method, whilst the sebaceous débris of the follicle stains blue.

7. **Erythrasma.**—This disease, which consists of brownish-yellow patches with slightly desquamating surface and well-defined margins, usually situated in the axillary or genital regions, is produced by the *Microsporon minutissimum*, a fungus which is made up of a network of extremely fine filaments irregularly segmented. It may be examined by the same method as the *Microsporon furfur*.

CHAPTER IX

THE NERVOUS SYSTEM

I. ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

MUCH recent knowledge regarding the structure and mechanism of the central nervous system is still too unsettled to admit of being used at the bedside; but

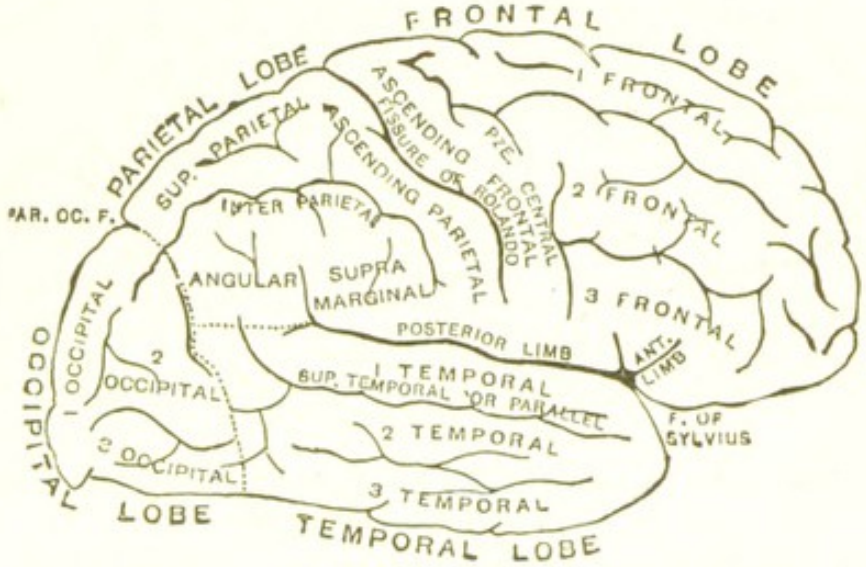


Fig. 105.—Outer aspect of right hemisphere, showing convolutions.

if the student wishes to investigate a case of nervous disease intelligently he must first have a clear grasp of some well-established facts in the anatomy and physiology of the brain and spinal cord. A few paragraphs devoted to these subjects will therefore not be out of place.

1. **Anatomy and physiology of the motor and sensory paths.**—The reader will remember that the **motor area** of the brain is situated in front of the fissure of Rolando (Figs. 105, 106), the leg

centre being highest up, the arm centre next to it, and the centres for the face, lips, and tongue being lowest. For the more exact localization of the various centres the figures in Plate 10 should be consulted.

The **motor fibres** start from the pyramidal cells in the above convolutions, and pass in the white matter of the hemispheres to the internal capsule (i.e. the knee-shaped band of white matter which is bounded on its outer side by the lenticular nucleus, and on its inner side by the optic thalamus and caudate nucleus).

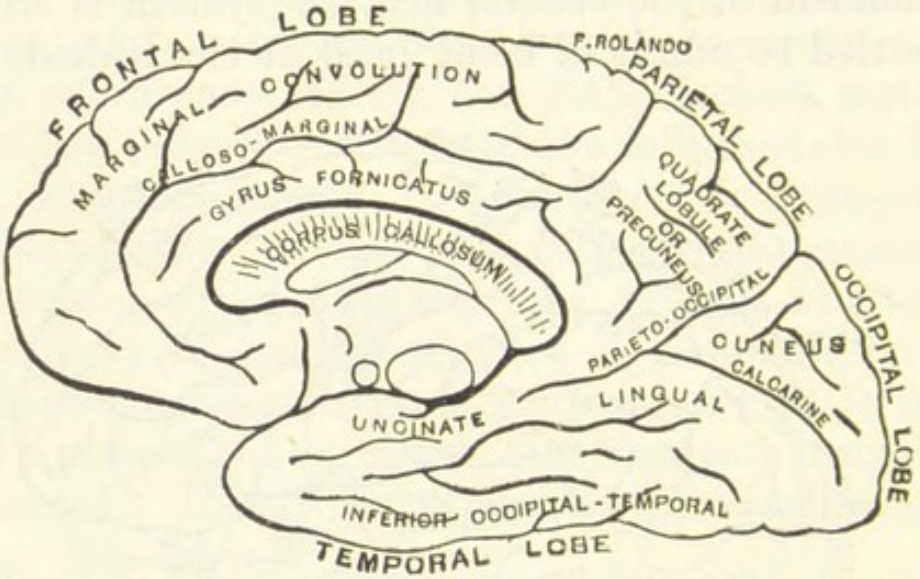
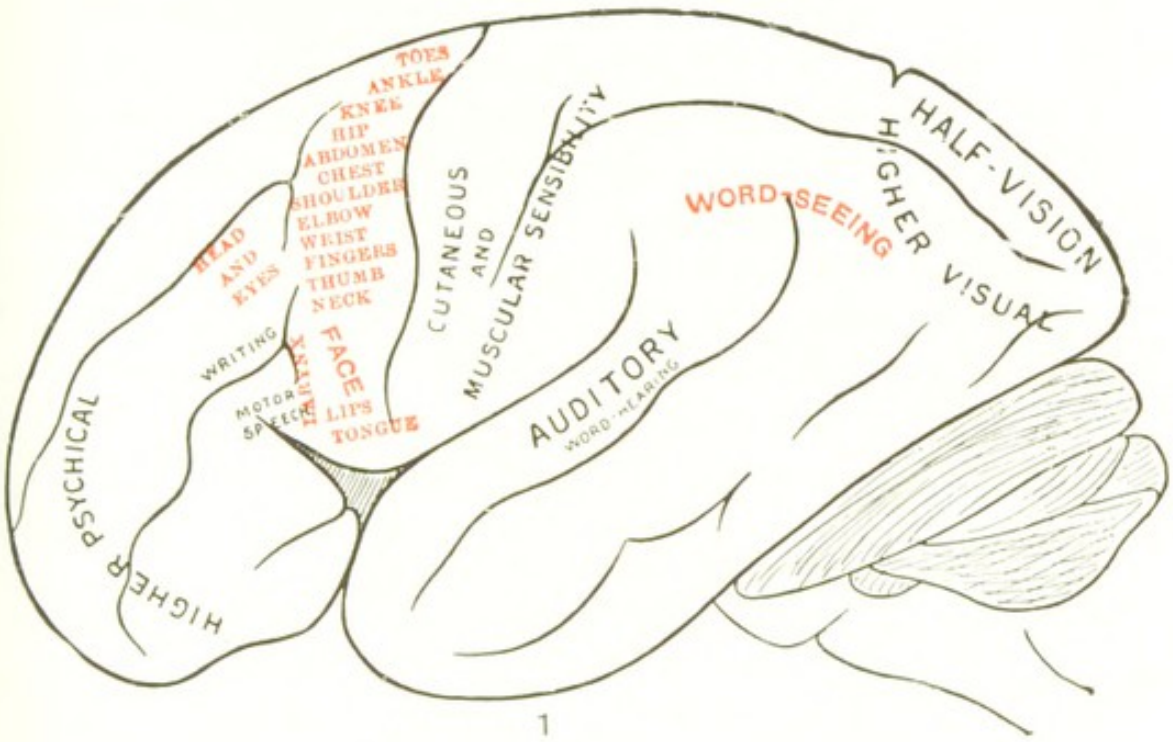
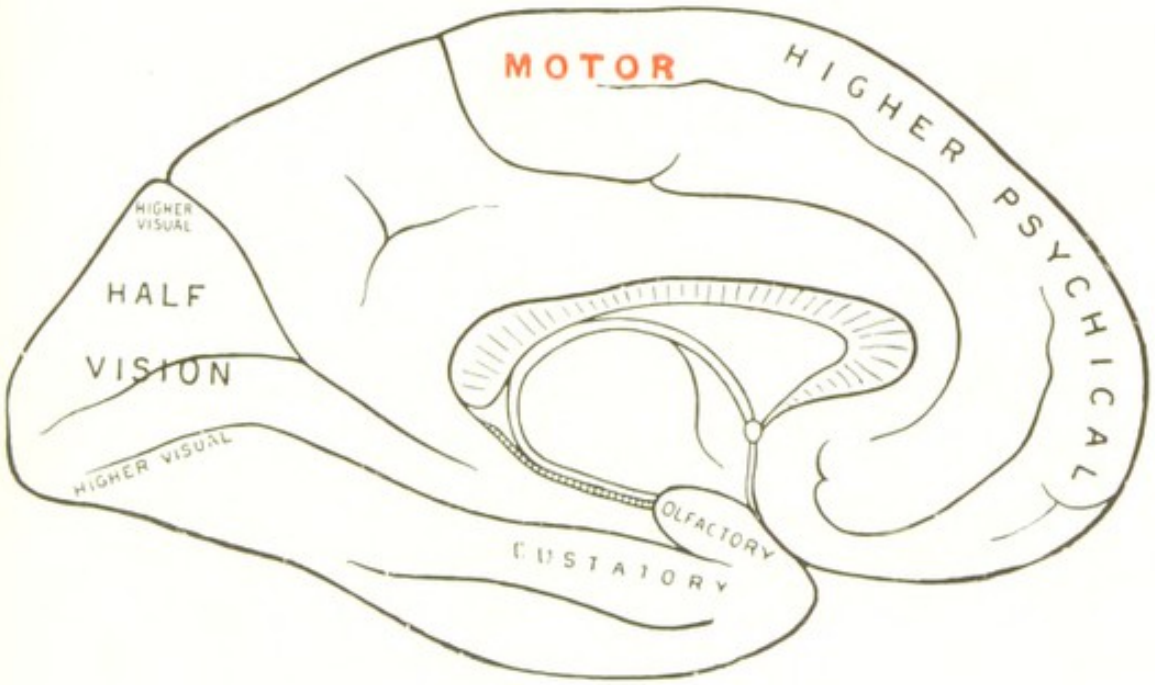


Fig. 106.—Mesial aspect of right hemisphere, showing convolutions.

The motor fibres occupy the anterior two-thirds of the posterior limb of the internal capsule, the fibres for the face being farthest forward, those for the leg farthest back, the fibres for the arm being between (Fig. 107). It is in the internal capsule that hæmorrhage most frequently occurs, and, owing to the close approximation of all the fibres at this point, a comparatively small lesion is able to produce a widespread result. From the internal capsule the motor fibres descend to the crus cerebri, occupying the middle third of its ventral aspect. As they descend in the crus the fibres for the leg are to the



1



2

Plate 10.—(1) Outer, (2) Mesial Aspects of Left Hemisphere, showing Functional Areas.

(*After Purves Stewart, "Diagnosis of Nervous Diseases."*)

outer side, the fibres for the face are nearest the middle line, and those for the arm are between the two. Below this level the fibres for the different limbs no longer run separate, but those for the arm and the leg are diffusely scattered over the cross-section of the pyramidal tract; consequently a lesion cannot produce a

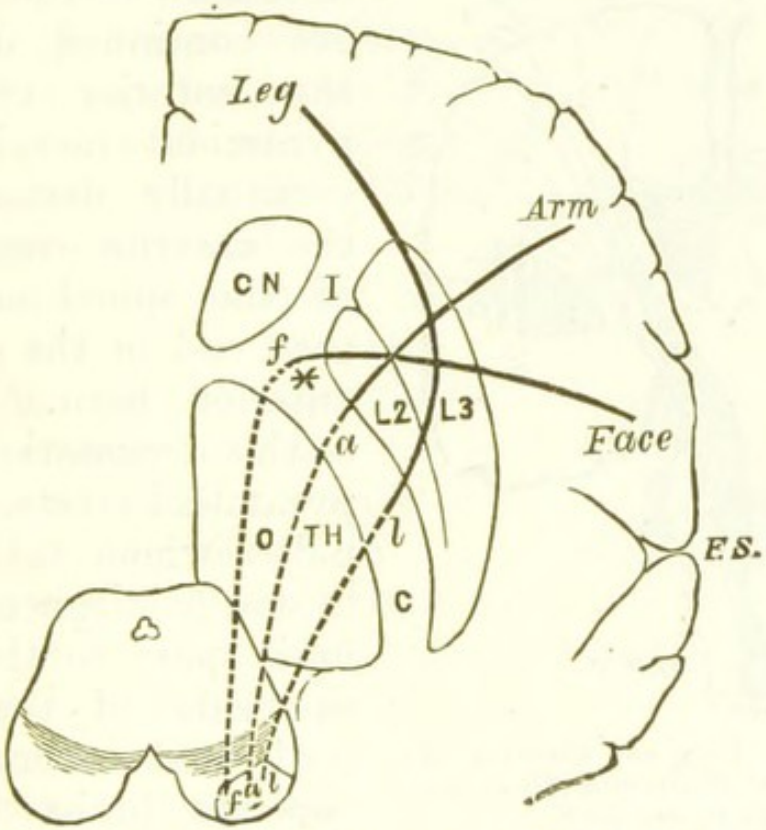


Fig. 107.—Diagram to show relative positions of the face, arm, and leg fibres in their course from cortex to crus. The section through the cortex and crus is vertical; through the internal capsule it is horizontal; * indicates the elbow of the internal capsule.

f, face; *a*, arm; *l*, leg fibres.

monoplegia, or paralysis of a single limb. Entering the pons, the fibres are no longer quite on the surface, but are covered by a layer of transversely placed fibres. In the upper part of the medulla they form a well-marked bundle, the anterior pyramid, lying quite on the surface. At the lower part of the medulla the greater number of the fibres cross to the opposite side, forming what is known as the *decussation of the pyramids*, and run down in the crossed pyramidal tract of

the cord, to end at different levels in the grey matter of the anterior cornu (Fig. 108). The motor impulses are here transferred to the cells of the anterior cornu and through the anterior roots, which arise from them, to the motor nerves and muscles. The small number of fibres which do not decussate in the medulla are continued down in the anterior or direct pyramidal tract, but they eventually decussate in the anterior commissure of the spinal cord, and then end in the opposite anterior horn. Owing to this decussation of the pyramidal tracts, the impulses which take origin in one hemisphere of the brain pass to the opposite side of the spinal cord, and innervate the opposite limbs and the muscles of this side of the body. A small number of fibres also descend in the crossed pyramidal tract of the same side. The existence of this small number of motor fibres which do not decussate at all, but end in the anterior cornu of the same side as that on which they took origin in the brain, explains the fact that after a unilateral cerebral lesion the knee-jerk on the same side may be exaggerated as well as that of the opposite leg. The pyramidal fibres to the motor cranial nerves decussate in the brain-stem close above the nuclei in which they terminate.

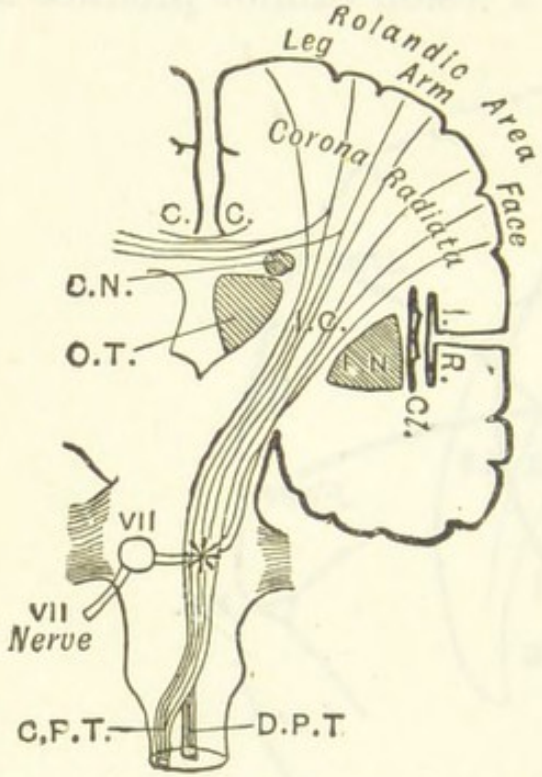


Fig. 108. — Diagram showing the course of the motor fibres from the cortex to the cord.

C C, corpus callosum; C N, caudate nucleus; I R, island of Reil; *Cl*, claustrum; *, site of facial decussation; C P T, crossed pyramidal tract; D P T, direct pyramidal tract.

In thus tracing the course of a motor impulse, we have spoken of nerve cells and nerve fibres. It would be better, however, to discard these names in favour of the modern terminology, which describes a nerve cell, its dendritic processes, and the fibre connected with it (axis cylinder process) as a **neurone**. Thus, anatomically, the motor impulses are conveyed by means of two neurones. One of these consists of the pyramidal cell in the cerebral cortex and the motor fibre arising from it (i.e. its axis cylinder process) and ending in the anterior cornu. The other is the anterior cornual cell and the fibre raising from it and ending in the muscle. There is apparently no direct anatomical continuity between these two neurones, but the nerve impulse is able to pass from the one to the other by contact (Fig. 109).

This conception has also the advantage of making clearer some well-

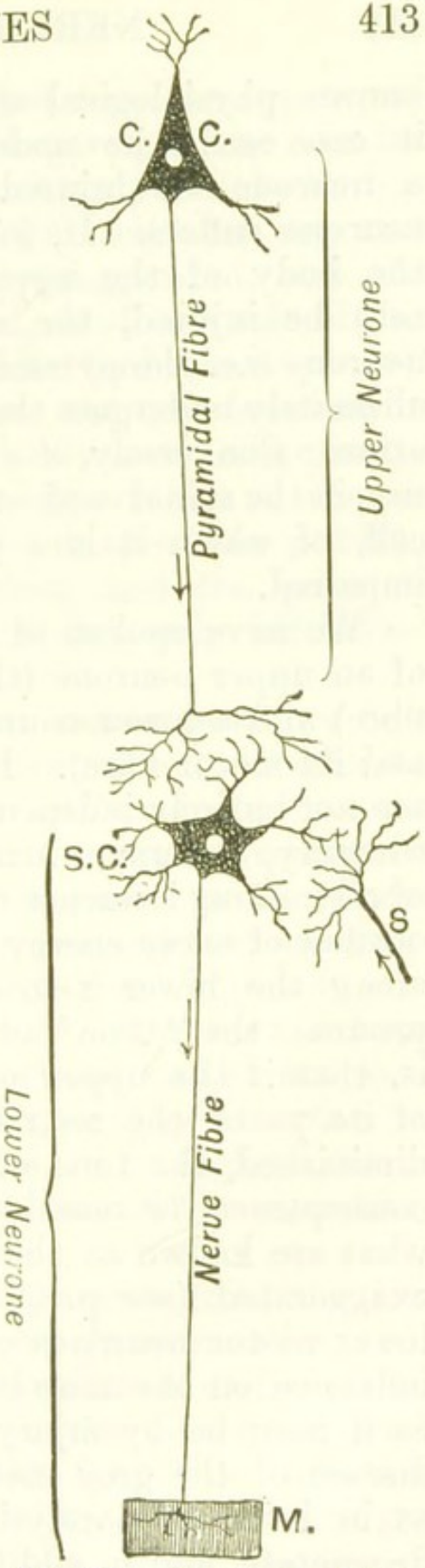


Fig. 109.—Upper and lower neurones of motor path.
C C, cerebral cell; S C, spinal cell; S, sensory fibre; M, muscle.

known physiological and pathological facts. Thus, it can easily be understood that if one part of a neurone be injured the health of the whole neurone suffers. If, for example, what one may call the body of the upper neurone (i.e. the cortical cell) be injured, the axis cylinder process of the neurone (i.e. the pyramidal fibre) is also affected, and ultimately undergoes the process spoken of as degeneration. Conversely, if a motor fibre be cut across—say, in the spinal cord—the health of the cortical nerve cell, of which it is a process, becomes secondarily impaired.

We have spoken of the motor path as consisting of an upper neurone (the cortical cell and its motor fibre) and a lower neurone (the anterior cornual cell and its motor fibre). It must be realized that these are not entirely independent of one another. On the contrary, the upper neurone seems to exercise a sort of restraining influence on the lower, which checks the outflow of nerve energy that is constantly proceeding along the lower neurone to the muscles, and that produces the "tone" of the latter. The result of this is, that if the upper neurone suffers damage in any of its parts, the restraint on the lower neurone is diminished, the tone of the muscles is raised; as a consequence the muscles become rigid or spastic, and what are known as their "tendon reflexes" become exaggerated (*see* p. 501). On the other hand, the lower motor neurones exert an important nutritive influence on the muscles, and when this is cut off, as it may be by injury of the peripheral nerves or disease of the grey matter of the anterior cornua, as in infantile paralysis, the muscles atrophy and degenerate, and in addition lose their tone.

Consequently, though paralysis may result from lesions of both the upper and the lower motor neurones,

considerable differences exist between the types produced. In *upper motor neurone paralysis* the muscles do not waste; their tone is increased, and the limb is consequently rigid; the electrical reactions are normal; the tendon reflexes are exaggerated, and the paralysis is usually widespread, as of one side of the body (hemiplegia) or of a whole limb (monoplegia). In *lower motor neurone paralysis* the muscles atrophy and lose their tone, and the paralysis is consequently flaccid; their electrical excitability is lost or the reaction of degeneration can be obtained (*see* p. 520); the tendon jerks are abolished, and the palsy is usually restricted and may involve only individual muscles or groups of muscles.

The anatomical paths for **sensation** are less accurately known than the motor paths. All afferent impulses from the periphery are conducted to the spinal cord by the sensory nerves, and through the spinal ganglia and the posterior spinal roots. These constitute *the peripheral or primary afferent neurones*. But only a small proportion of these impulses ever reach consciousness as sensations. The rest are concerned in the spinal reflex functions, in the maintenance of the tone of the muscles, or they terminate in the portions of the spinal cord, or in higher centres such as the cerebellum, which control the co-ordination of muscular activity.

Sensory impressions come not only from the skin and superficial tissues, but also from muscles, tendons, and joints. When sensation is disturbed by disease it may be seen that in the peripheral level sensory impulses are divided into three distinct systems—(1) **epicritic sensibility**, by which light touch, its localization, and the milder degrees of temperature which we call "warm" and "cool" can be appreciated; (2) the **protopathic system,**

which carries impressions of cutaneous pain, as that produced by a prick, and the extreme degrees of heat and cold; (3) **deep sensibility**, which is conveyed in the afferent fibres that run from muscles, tendons, bones, and joints. This third system underlies the recognition of position and movement, and, when cutaneous sensibility is lost, heavy touches or the pain produced by pressure can be appreciated through it. Owing to the wide anastomosis and distribution of the fibres of this system it frequently escapes in areas in which cutaneous sensibility is lost; then firm touches, as those produced by a finger or the point of a pencil, may be felt, though light touches cannot be appreciated.

After they have entered the spinal cord, the various sensory impulses are rearranged and grouped into other systems. The majority of the peripheral neurones that have carried them hither terminate in the grey matter of the posterior horn at or near the level at which they enter, and from this grey matter the secondary sensory tracts take origin. These cross immediately, or within a few segments, to the opposite antero-lateral column of the cord, and in it ascend to the brain-stem (Fig. 110). Touch, pain, and temperature are carried in this crossed secondary path. Other peripheral fibres, however, do not terminate in the grey matter of the spinal cord, but run cerebralwards in the posterior column of the same side as that on which they entered the cord; these posterior column fibres carry the impulses on which depends the appreciation of position, of movement, and of size and shape. Vibration and the power of discriminating Weber's compass-points are also conveyed in the posterior column, and this also contains a path for touch. Consequently, at any level of the spinal cord we have in each half of it two sensory paths conveying sensory

impressions cerebralwards : one, in the antero-lateral column, carries touch, pain, and temperature from the opposite half of the body; and a second, in the posterior column, conveys the appreciation of posture, weight,

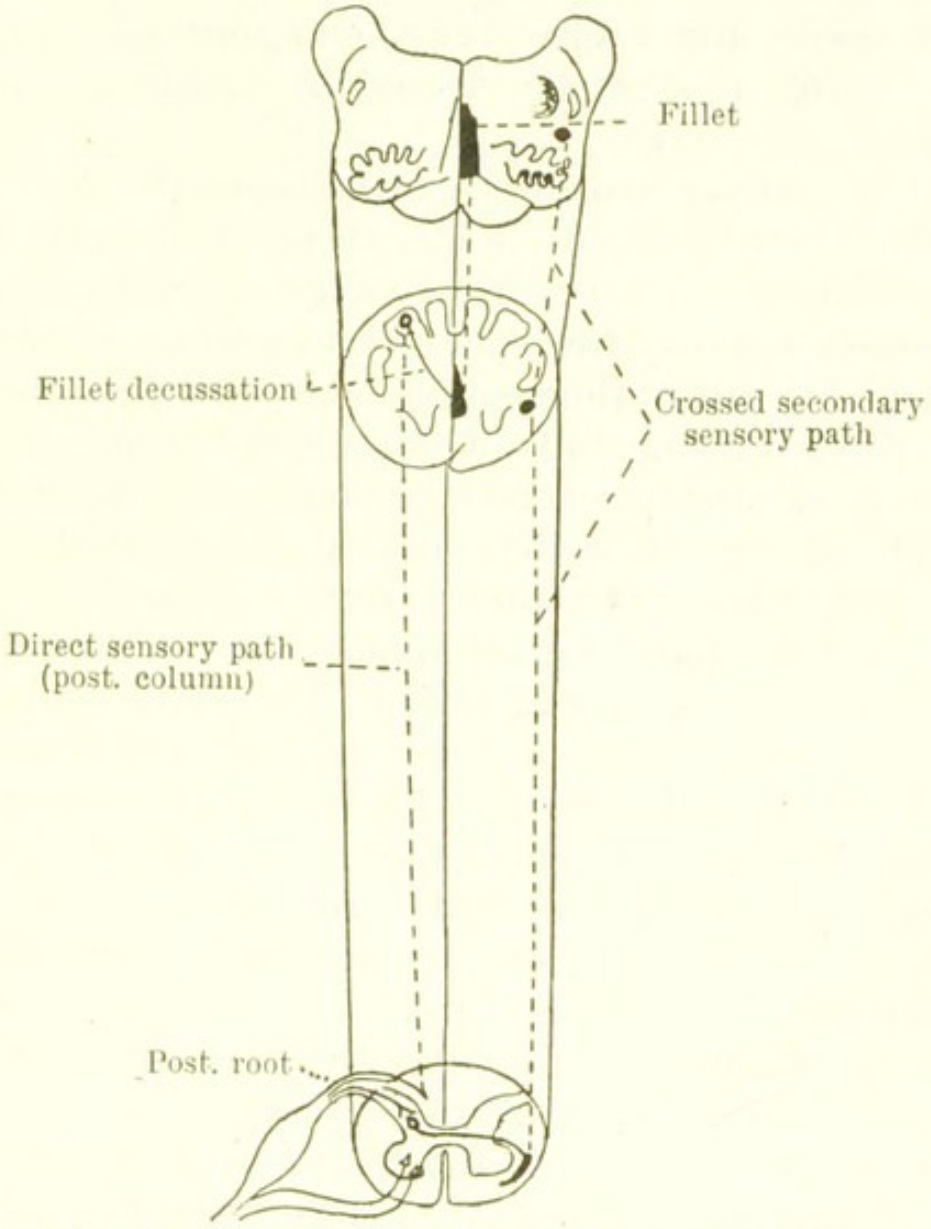


Fig. 110.—Diagram of the sensory paths in the spinal cord and the medulla oblongata.

size, shape, and other qualities of sensation from the same side of the body (Fig. 110). A unilateral lesion of the spinal cord, therefore, produces the Brown-Séquard phenomenon, in which pain and thermal sensibility are lost below the level of the lesion on the

opposite side of the body, while on the side of the lesion there is, in addition to motor paralysis, disturbance of the sense of position and of movement, and loss of the recognition of weight, size, shape, of vibration and of the compass test. As touch has a double path, one on the same and another on the opposite side of the spinal cord, it is rarely affected by unilateral spinal lesions.

At the upper end of the spinal cord the posterior column fibres terminate in the nuclei of Goll and Burdach, and the impulses they carry are taken up by **secondary sensory fibres,** which immediately cross to the opposite side of the medulla in the fillet decussation. Therefore, in the medulla oblongata all sensory impressions are carried in secondary tracts which lie on that side of the nervous system opposite to the half of the body from which they come. But even here all do not run in a single path, for pain and temperature seem to pass through the lateral part of the bulb, while the posterior column sensory elements are conveyed by the mesial fillet (Fig. 110). Higher in the brain-stem, however, all sensory elements probably run closely together in the mesial fillet, and those which have come from the spinal cord are probably joined by the secondary fibres from the nuclei of the sensory cranial nerves. Finally, the fibres of the fillet terminate in the optic thalamus, no secondary sensory fibres pass uninterrupted beyond it, and from this a tertiary system of sensory fibres conveys sensory impressions to the cerebral cortex.

The exact extent of the **cerebral cortex** concerned in reception of sensation is still doubtful; it certainly lies mainly in the parietal lobes behind the fissures of Rolando. It seems very probable, however, that certain sensory qualities, as pain, never reach the cortex but affect consciousness through subcortical centres,

Points of Origin and Exit of Nerve Roots from Cord and Spinal Canal

Roots	Level of Surface Origin	Point of Exit
C. 1	Just above arch of atlas.	Between atlas and occiput.
2	Ranges from just above to just below spine of atlas.	Above axis.
3	At or just above spine of axis.	Above 3rd C.
4	From spine of axis to spine of 3rd cervical.	Above 4th C.
5	Ranges from lower edge of spine of axis to that of 4th cervical.	Above 5th C
6	Ranges from lower edge of spine of 3rd to that of 5th cervical.	Above 6th C.
7	Ranges between top of 4th to bottom of 6th spine.	Above 7th C.
8	Ranges between top of 5th to top of 7th spine.	Above 1st D.
D. 1	Ranges between above 6th and below 7th spine.	Between 1st and 2nd D.
2	Ranges from lower edge of 6th cervical spine to that of 1st dorsal.	Between 2nd and 3rd D.
3	Ranges from upper edge of 7th cervical spine to lower of 2nd dorsal.	Between 3rd and 4th D.
4	Ranges from top of 1st dorsal spine to that of 3rd dorsal.	Between 4th and 5th D.
5	Ranges from top of 2nd to top of 4th dorsal spine.	Between 5th and 6th D.
6	Ranges from lower edge of 2nd dorsal spine to upper of 5th.	Between 6th and 7th D.
7	Ranges from top of 4th to bottom of 5th dorsal spine.	Between 7th and 8th D.
8	Ranges from top of 5th to top of 6th spine.	Between 8th and 9th D.
9	Ranges from between 5th and 6th spines to top of 7th.	Between 9th and 10th D.
10	Ranges from lower edge of 6th to upper of 8th.	Between 10th and 11th D.
11	Ranges from top of 7th to top of 8th spine.	Between 11th and 12th D.
12	Ranges between top of 8th and bottom of 9th spine.	Between 12th D. and 1st L.
L. 1	Ranges between top of 9th spine and bottom of 10th.	Between 1st and 2nd L.
2	Ranges between 9th and 11th dorsal spines.	Between 2nd and 3rd L.
3	Ranges between top of 10th and bottom of 11th spine.	Between 3rd and 4th L.
4	Ranges between bottom of 10th and top of 12th spine.	Between 4th and 5th L.
5	Ranges between top of 11th and top of 12th spine.	Between 5th L. and 1st S.
S. 1	Ranges between lower border of 11th dorsal spine and top of 1st lumbar.	Between 1st and 2nd S.
2	Usually between 12th dorsal and 1st lumbar spines.	Between 2nd and 3rd S.
3	Usually between 12th dorsal and 1st lumbar spines.	Between 3rd and 4th S.
4	Usually between 12th dorsal and 1st lumbar spines.	Between 4th and 5th S.

probably through the optic thalamus. The course of the fibres and the position of the centres for the special senses are described in the section dealing with the cranial nerves (p. 439). The speech centres and their connections are described at p. 434.

2. **The spinal cord.**—The cord extends as far down as the interspace between the 1st and 2nd lumbar spines; the membranes are continued down as far as the body of the 2nd sacral vertebra.

The *cervical enlargement* reaches to the 7th cervical spine. Its largest part is opposite the disc between the 5th and 6th cervical vertebræ.

The *lumbar enlargement* lies opposite the three lowest dorsal spines, its widest part corresponding to the body of the 12th dorsal vertebra.

Physiologically, the cord is to be regarded as made up of a series of superimposed segments, from each of which a pair of nerve roots arises. To enable us to localize focal lesions of the cord it is necessary to be acquainted with the functions of each segment, and therefore with the area of supply of the pair of nerve roots arising from it.

The table on p. 419 shows the points of origin and emergence of the nerve roots from the cord and spinal canal. Plates 11 and 12 exhibit in a diagrammatic way the motor functions of the cervical and lumbar segments and the reflexes over which each segment presides. The sensory functions are exhibited in Figs. 111, 112, and 113.

Figs. 112 and 113 show the sensory distribution of the **posterior nerve roots** ("root areas").

Fig. 114 shows the position of the different **tracts of the cord** on transverse section.

The list on p. 424 shows the **nerve supply of the muscles** of the trunk and limbs. It may be found convenient for reference in the study of cases of

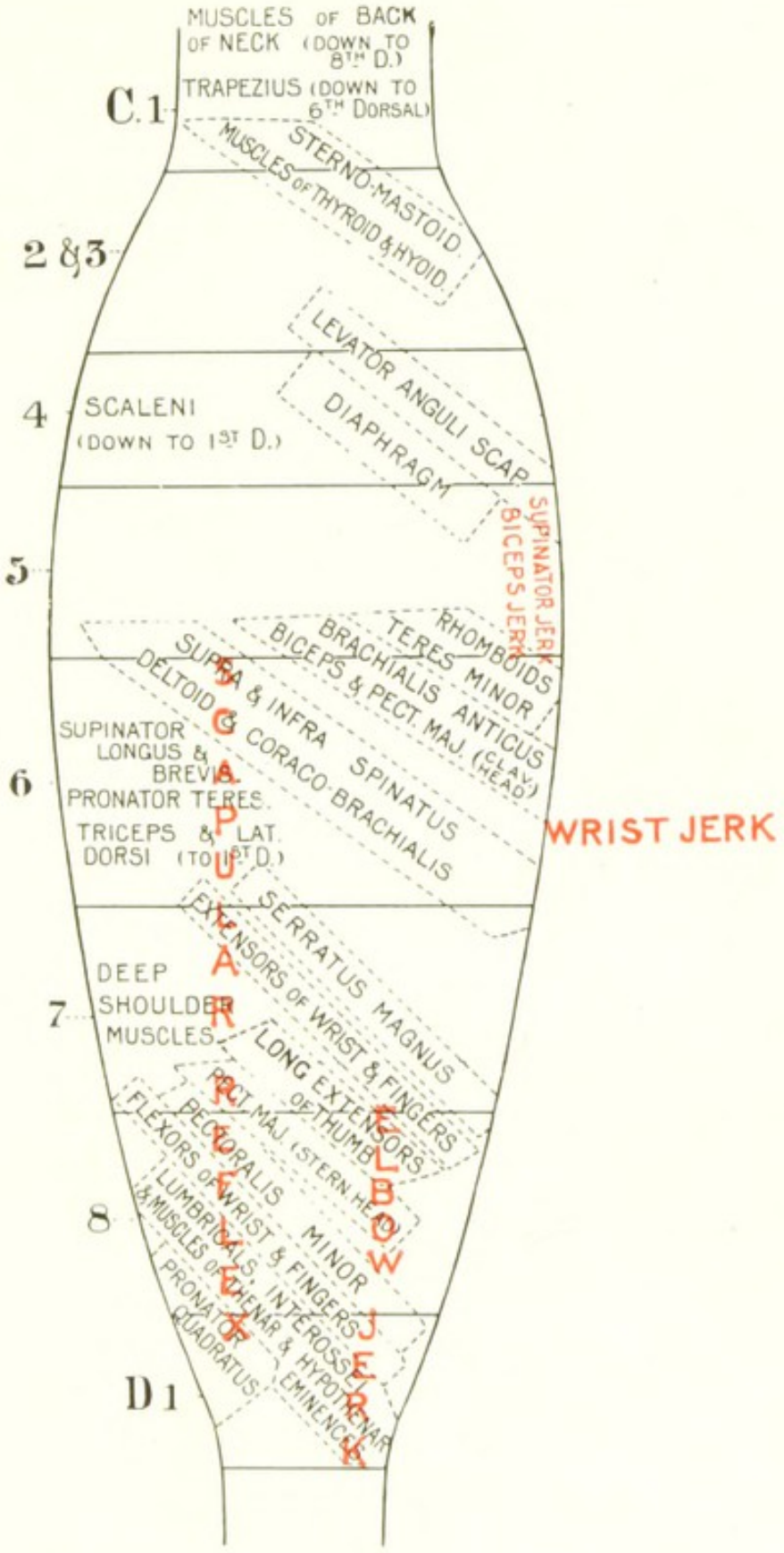


Plate 11. — Motor Segmental Function of the Cervical Enlargement.

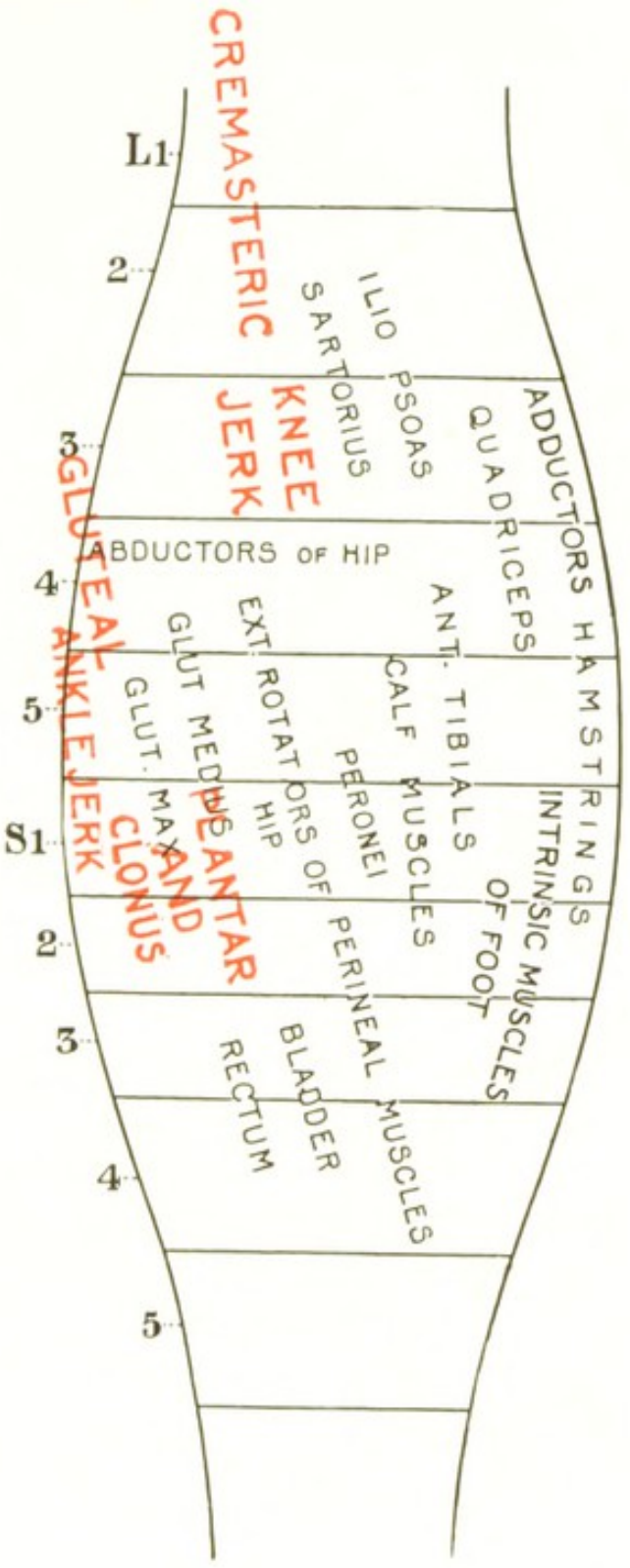


Plate 12. — Motor Segmental Functions of the Lumbar Enlargement.

peripheral paralysis. The nerve supply of the head is considered along with the cranial nerves (p. 439).

The peripheral distribution of the chief **sensory nerves** is sufficiently indicated in Figs. 115, 116, 117, and 118.

Vascular supply of the brain and spinal

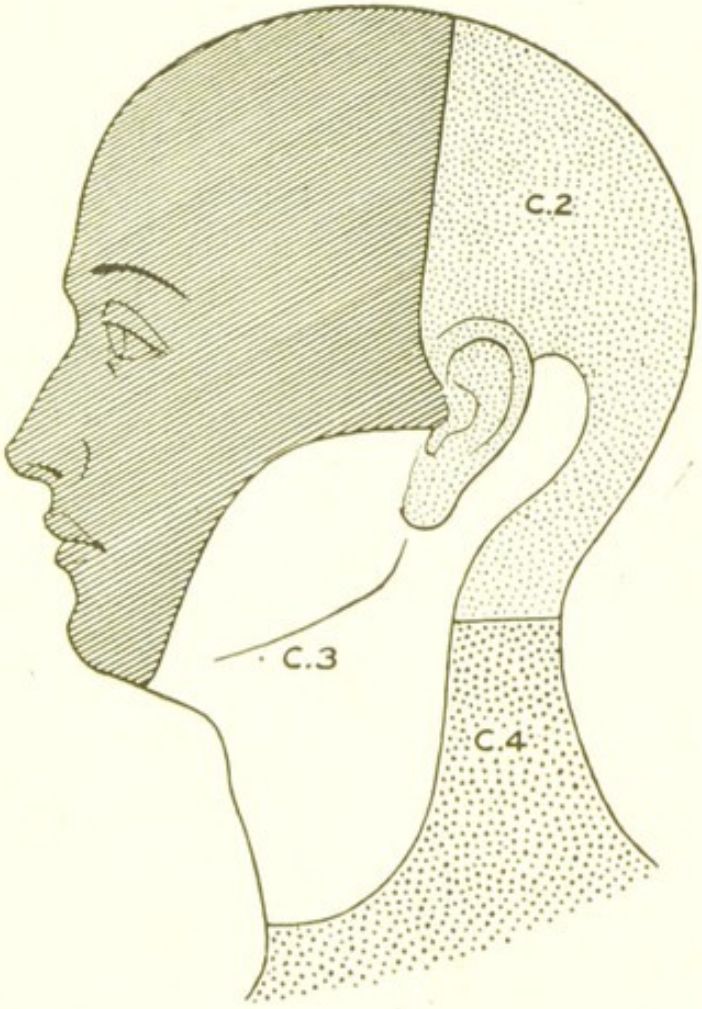


Fig. 111.—Lateral view of the skin areas supplied by the 2nd, 3rd, and 4th cervical segments.

cord.—The **brain** is supplied by the internal carotid and vertebral arteries. Owing to the position of origin of the left common carotid, an embolus can enter it more easily than it can the artery of the opposite side. Embolic lesions are therefore more frequent in the left than in the right cerebral hemisphere.

The two *vertebral arteries* unite at the lower border

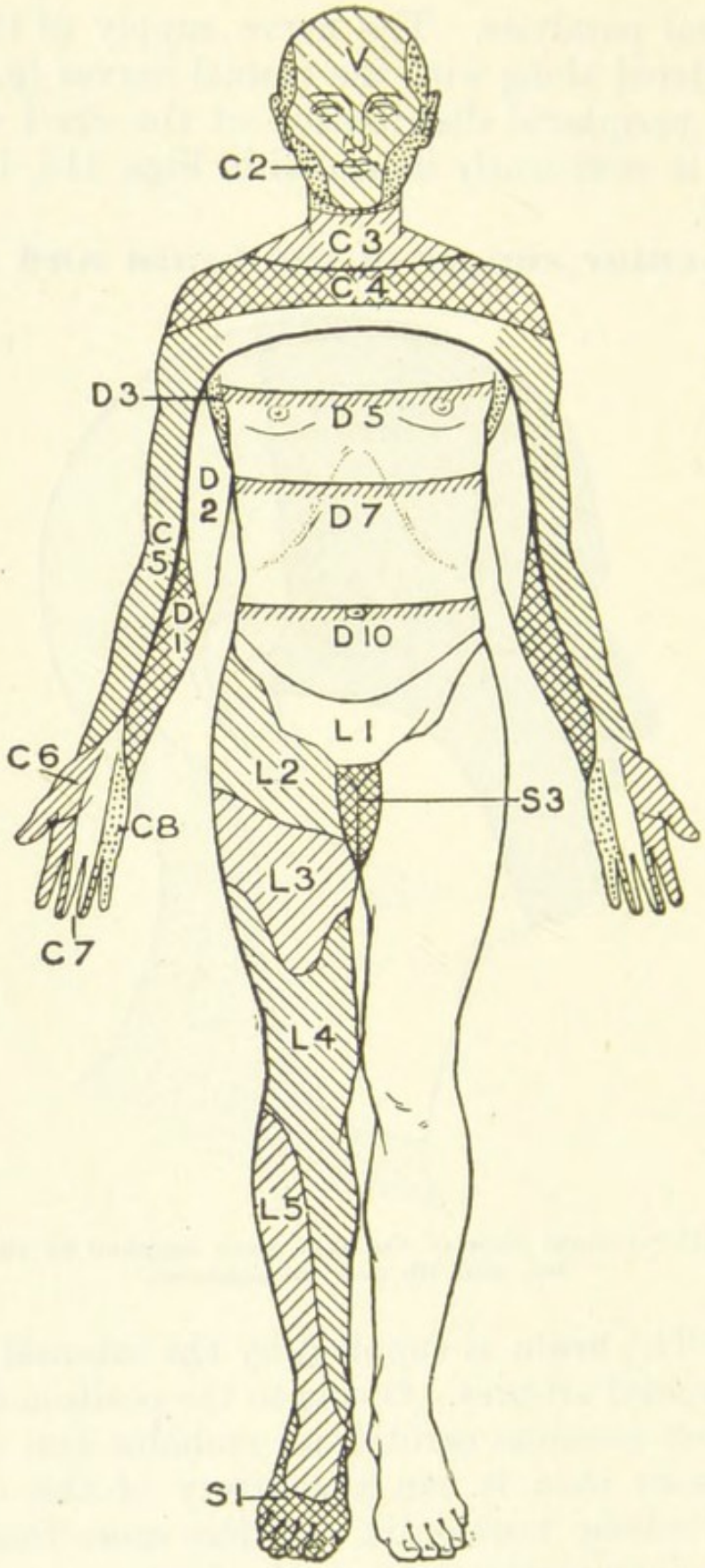


Fig. 112.—Segmental sensory areas of the cord. (*After Tooth.*)

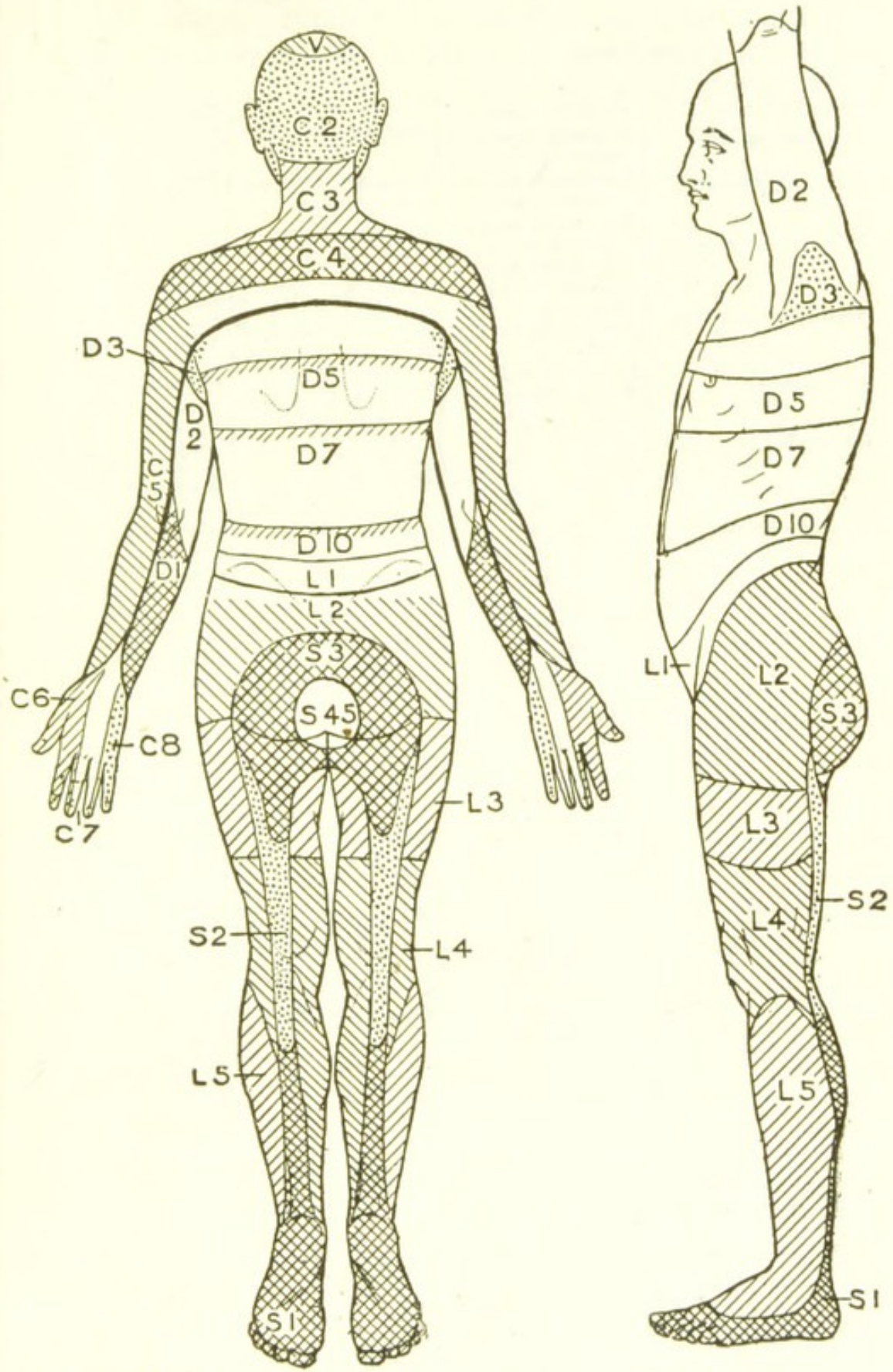


Fig. 113.—Segmental sensory areas of the cord. (*After Tooth.*

Nerve Supply of Trunk and Limb Muscles

Upper Limb

Post. Thoracic ..	Serratus magnus.
Suprascapulars ..	Supraspinatus. Infraspinatus.
Ext. Ant. Thoracic	Pectoralis major (upper part).
Int. Ant. Thoracic	Pectoralis major (lower part). Pectoralis minor.
Musculo-Cutaneous	Coraco-brachialis. Biceps. Brachialis anticus.
Subscapular	Subscapularis. Teres major. Latiss. dorsi.
Circumflex ..	Deltoid. Teres minor.
Musculo-Spiral ..	Triceps. Ext. carp. rad. long. Supinator long. B. anticus. Anconeus.
Post. Interosseus ..	Supinator brevis. Ext. carp. rad. brev. Ext. carp. uln. Ext. comm. digit. Ext. ossis metac. poll. Ext. primi intern. poll. Ext. secund. intern. poll. Ext. indicis. Ext. minimi digiti.
Median	Pronator radii teres. Pronator quadratus. Palmaris longus. Flexor carpi radialis. Flexor sublim. digit. Flexor longus pollicis. Opponens pollicis. Abductor pollicis Two outer lumbricals.
Median and Ulnar (jointly)	Flex. profund. dig. Flexor brevis pollicis.
Ulnar	Flexor carpi ulnaris. Adductor pollicis. Muscles of little finger. Interossei. Two inner lumbricals.

Trunk and Lower Limb

Intercostals	Intercostals. Rectus abdominis. External oblique.
Branches of Lumbar Nerves	Erector spinæ. Quadratus lumborum.
Genito-Crural ..	Cremaster.
Anterior Crural	Sartorius. Pectineus. Rectus femoris. Vastus externus. Vastus internus. Crureus. Psoas and iliacus.
Obturator	Gracilis. Obturator externus. Adductor longus. Adductor brevis. Adductor magnus (with sciatic).
Infer. Gluteal ..	Gluteus maximus.
Sup. Gluteal ..	Gluteus medius and minimus. Tens. vag. femoris.
Great Sciatic ..	Biceps femoris. Semitendinosus. Semimembranosus. Adductor magnus (with obturator).
Int. Popliteal ..	Gastrocnemius. Soleus. Tibialis posticus. Flex. comm. digit. Flex. long. hallucis.
Int. Plantar ..	Flex. brev. hallucis. Flex. brev. digit. Abductor hallucis.
Ext. Plantar ..	Adductor hallucis. Interossei. Flex. brev. min. Abductor min. dig.
Ext. Popliteal ..	Tibialis anticus. Ext. prop. hallucis. Ext. digit. longus. Peroneus longus. Peroneus brevis. Extens. brev. digit.

of the pons to form the *basilar*, which runs up the middle of the anterior surface of the pons, and ends by dividing into the two posterior cerebrals. It gives off lateral branches which run out transversely over the pons, and vertical branches which pass into

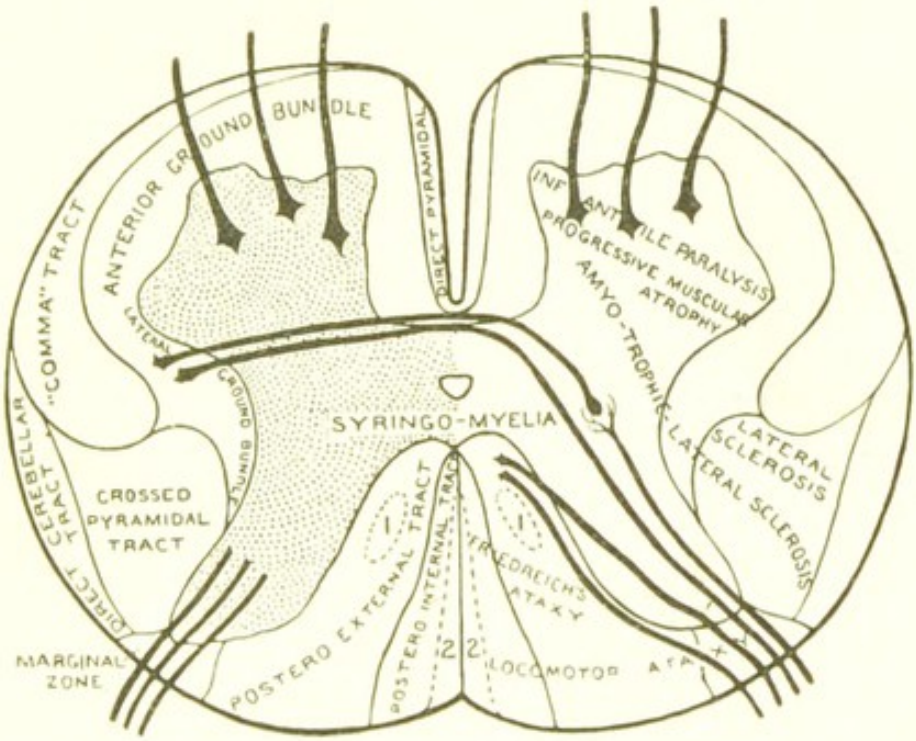


Fig. 114.—Scheme of a transverse section of the spinal cord, showing on the left side the position of the various tracts, and on the right side the names of the diseases affecting each part.

1, Schultze's descending tract in postero-external tract; 2, Septomarginal tract of Bruce and Muir in postero internal tract. Besides the names given on the diagram, the following synonyms should be noted: Postero-internal tract = Fasciculus gracilis = Column of Goll; Postero-external tract = Fasciculus cuneatus = Column of Burdach; Marginal zone = Lissauer's tract; Ascending antero-lateral tract = Tract of Gowers; Schultze's tract = "Comma" tract.

its substance. The latter not infrequently become thrombosed.

The *posterior cerebrals* supply the occipital lobes, the lower part of the temporo-sphenoidal lobes, with the uncinate gyrus, the inner part of the crus and the corpora quadrigemina, and the posterior part of the posterior limb of the internal capsule. Blocking of one of these arteries will therefore involve the visual centre and the sensory fibres.

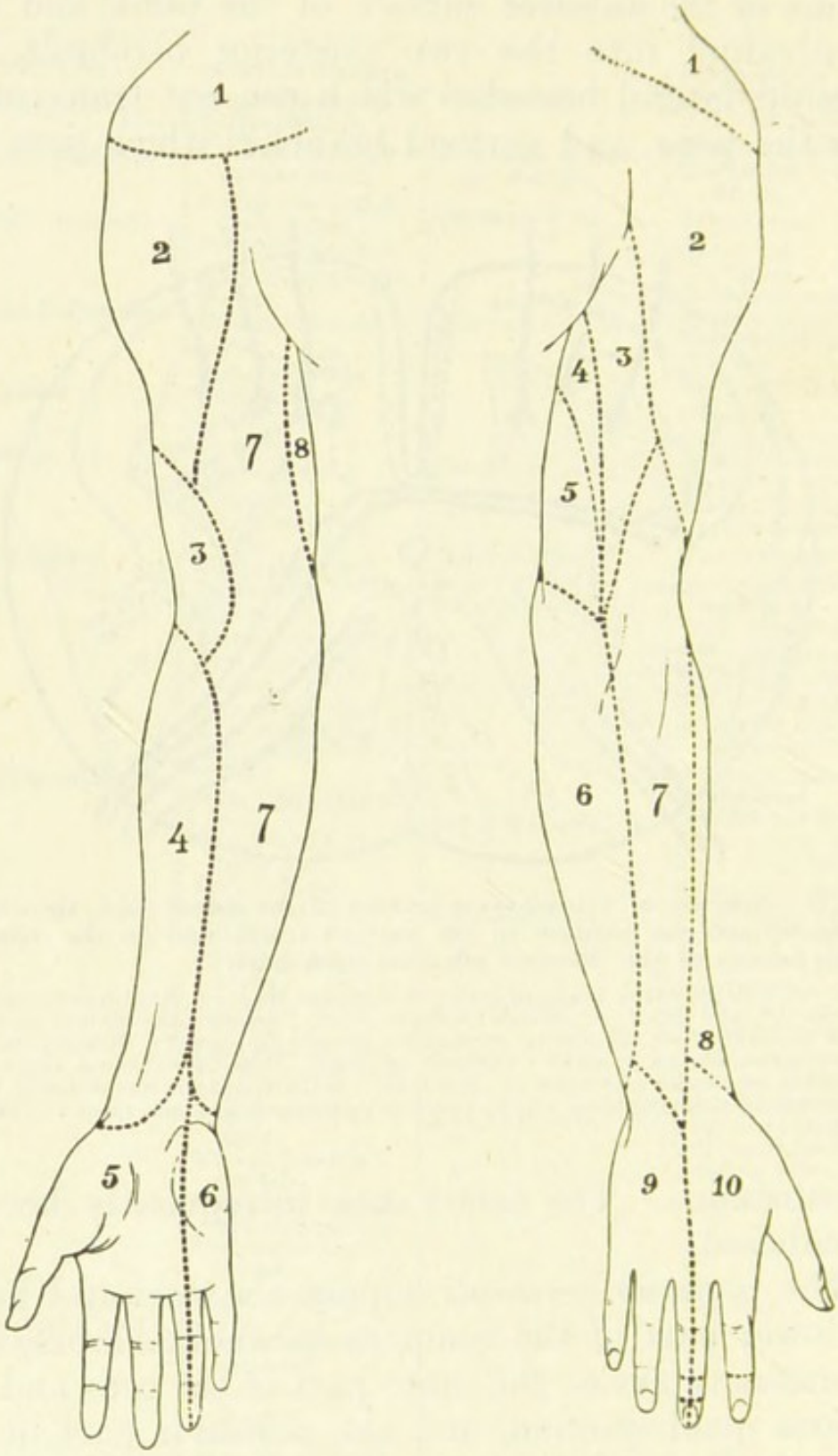


Fig. 115.—Cutaneous nerve supply of upper limb.

Anterior aspect: 1, cervical plexus; 2, circumflex; 3, ext. cut. of musc.-spiral; 4, ext. cutaneous; 5, median; 6, ulnar: 7, int. cutaneous; 8, nerve of Wrisberg. *Posterior aspect:* 1, cervical plexus; 2, circumflex; 3, int. cut. of musc.-spiral; 4, intercosto-humeral; 5, nerve of Wrisberg; 6, int. cutaneous; 7, ext. cut of musc.-spiral; 8, musc.-cutaneous; 9, ulnar; 10, radial.

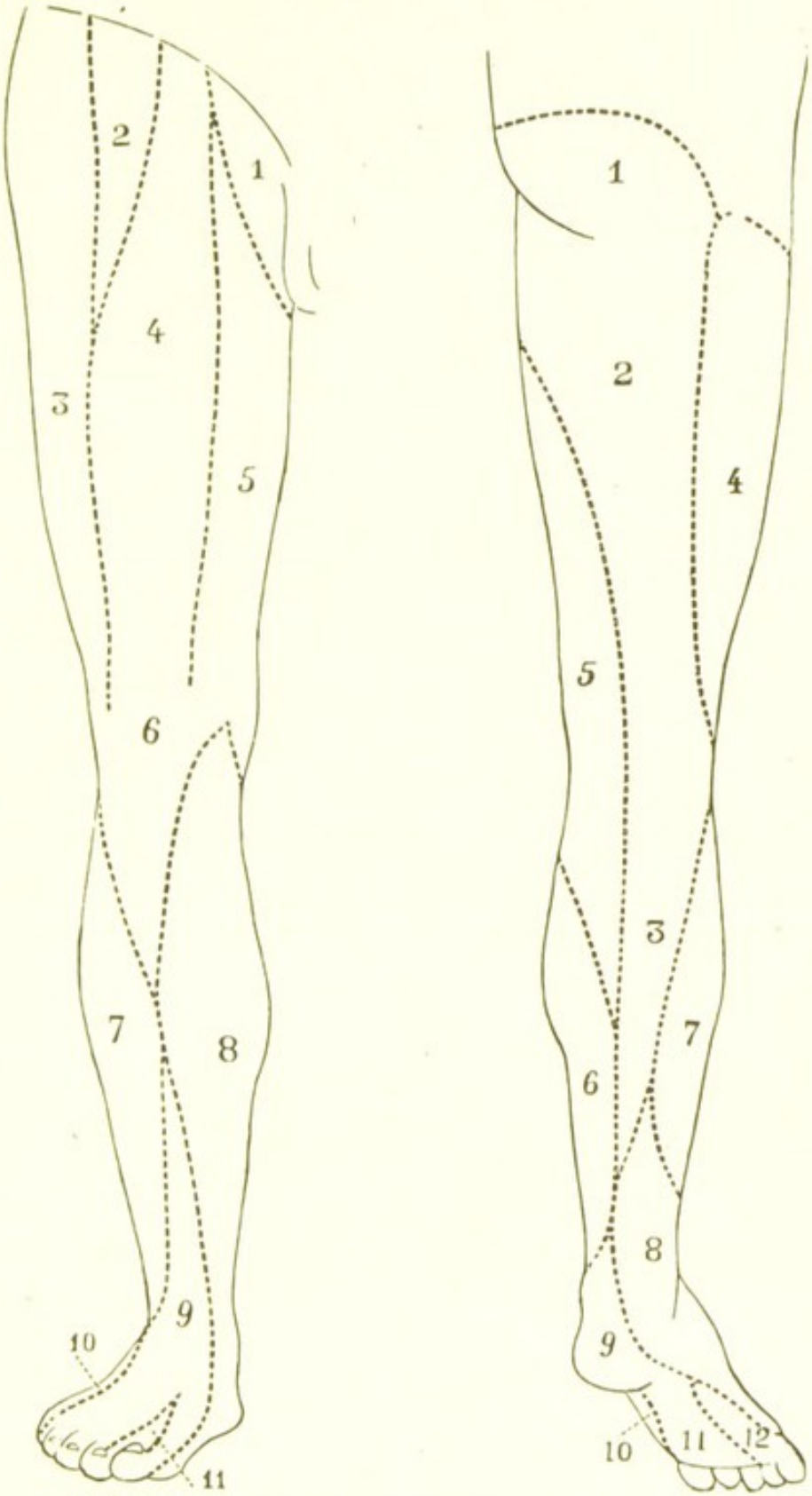


Fig. 116.—Cutaneous nerve supply of lower limb.

Anterior aspect: 1, ilio-inguinal; 2, genito-crural; 3, ext. cutaneous; 4, middle cutaneous; 5, int. cutaneous; 6, patellar plexus; 7, branches of ext. popliteal; 8, int. saphenous; 9, musc.-cutaneous; 10, ext. saphenous; 11, ant. tibial. *Posterior aspect:* 1, 2, 3, small sciatic; 4, ext. cutaneous; 5, int. cutaneous; 6, int. saphenous; 7, branches of ext. popliteal; 8, short saphenous; 9, post. tibial; 10, int. saphenous; 11, int. plantar; 12, ext. plantar.

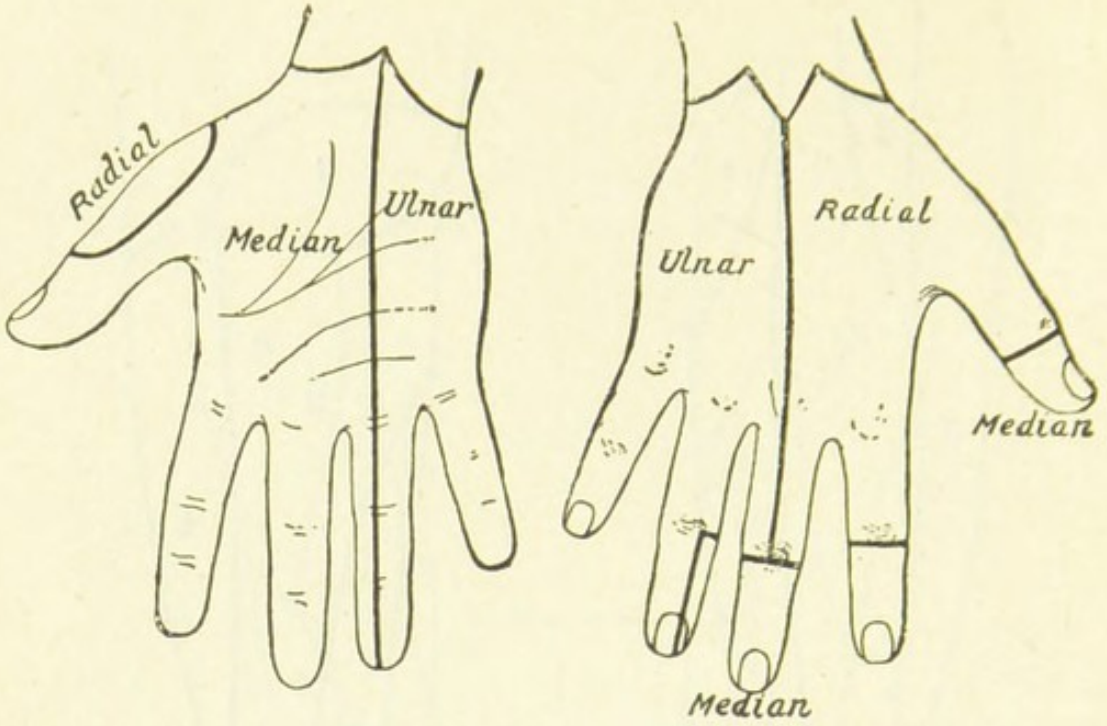


Fig. 117.—Distribution of the sensory nerves in the hand.
(*After Bernhardt.*)

The basilar artery supplies the upper surface of the cerebellum; the vertebrals supply its lower

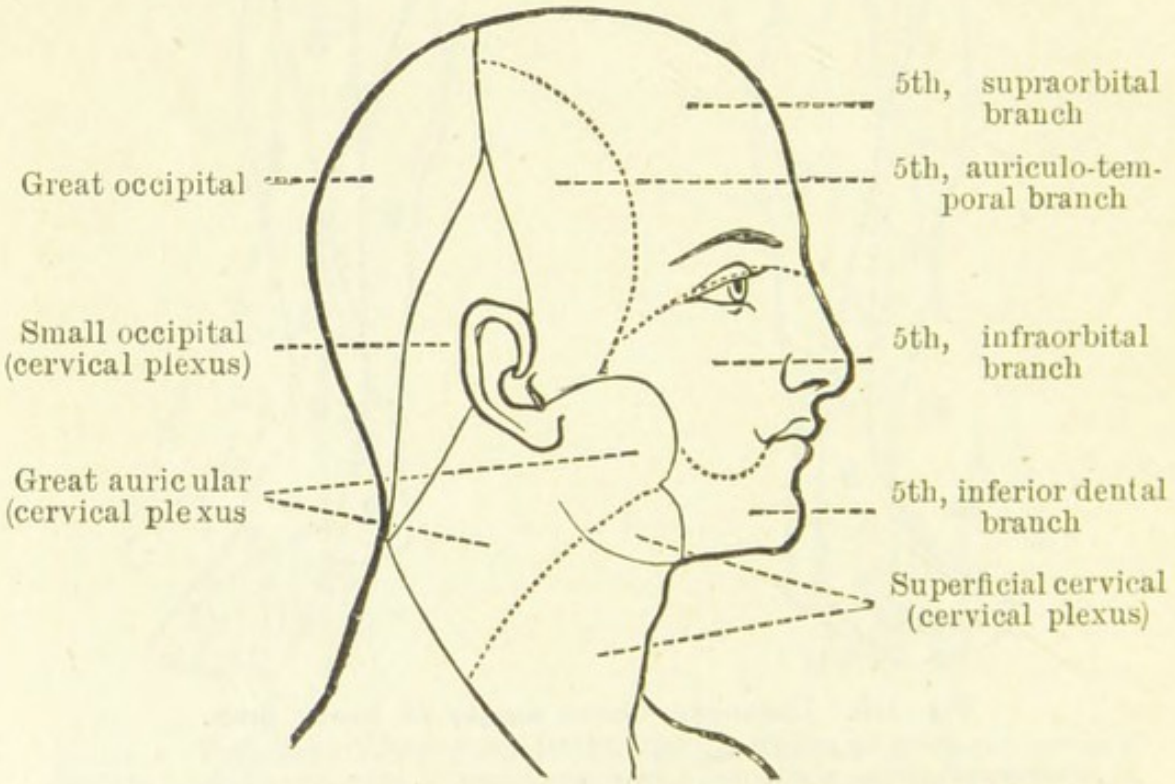


Fig. 118.—Distribution of the sensory nerves of the head. Compare with it the segmental distribution as shown in Fig. 111.

surface, as well as the greater part of the medulla oblongata.

The *internal carotid* gives off the *anterior cerebral* artery, which curves round the anterior end of the corpus callosum, and is chiefly distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure. It also supplies the superior frontal convolution.

The internal carotid is practically continued on to the brain as the *middle cerebral*, which lies in the Sylvian fissure. An embolus which has found its way into the internal carotid, therefore, usually ends in the middle cerebral or one of its branches. The middle cerebral gives off *cortical branches*, which supply the motor area and the upper part of the temporo-sphenoidal lobe. These branches anastomose freely with those of adjoining arteries, hence blocking of one of them may be largely compensated by the establishment of a collateral circulation. It also gives off *central branches*, which run more or less vertically upwards, penetrating into the brain substance and supplying the basal ganglia. There are two chief groups of these central arteries—an anterior group called the *lenticulo-striate*, and a posterior group, the *lenticulo-optic*. These arteries are very commonly the seat of miliary aneurysms, and as the lenticulo-striate are more directly exposed to the force of the wave of arterial blood they are more frequently ruptured than are the lenticulo-optic. These central arteries do not anastomose with one another. They are, therefore, to be regarded as end-arteries. Hence it is that a lesion of one of them is much less likely to be compensated than is a lesion of a cortical branch.

The venous blood from the brain is poured into the *venous sinuses*. Owing to the slow current in these, thrombosis readily occurs in them. The blood from

the interior of the lateral ventricles is chiefly returned by the veins of Galen, which end in the straight sinus. Owing to their long course, these veins are frequently exposed to pressure by tumours, etc. This is apt to lead to increased exudation of fluid into the lateral ventricles.

The **arteries which supply the spinal cord** have a long and tortuous course. This renders them liable to thrombosis, but makes embolism of them almost impossible. The lower end of the cord is far removed from the point of origin of the vessels which supply it. Hence it is, perhaps, that this part is more liable to suffer damage from nutritional changes than are the higher regions.

The student may now pass to the method of examining a patient with nervous disease, as described in the subsequent sections. We would recommend him to begin by ascertaining the state of the intellectual faculties of the patient, including speech (Section II.). He should then rapidly test the condition of the cranial nerves in their order. How this is to be done is described in Section III. (p. 439). By proceeding thus, valuable information is gained at the outset, which may guide one in the subsequent investigations. The motor, sensory, reflex, and trophic functions should then be examined in order, following the methods described in Sections IV. (p. 478), V. (p. 491), VI. (p. 497), VII. (p. 509). Lastly, the electrical reactions of the muscles and nerves should be tested in those cases in which it may seem necessary (Section VIII., p. 509).

II. INTELLECTUAL FUNCTIONS

It is important to arrive at some idea of the patient's intellectual state early in the taking of a nervous case, as it affords indications that are of help

in the subsequent investigation of his symptoms. For example, if one finds that his memory is deficient, one attaches only a limited value to the account that he gives of the onset of his illness or the state of his previous health. Or if one discovers that he is comatose, or unable to understand speech, it is evident that one cannot expect to make much of any attempt to investigate the state of his sensory functions. This section will, therefore, be devoted to methods of investigating a patient's mental condition, including the functions concerned in producing and interpreting speech.

The first thing to be determined is whether we are dealing with **a right-handed or a left-handed patient.** The importance of this depends upon the fact that right-handed people are left-brained, and vice versa. Ask the patient, if a male, which hand he uses to throw a stone or to pull a cork; if a female, which hand is employed in combing the hair. It is of comparatively little use to ask which hand he writes with, as all children are taught to write with the right hand.

The state of the **memory** next calls for investigation. It may be tested by asking the patient what day of the week it is, what he ate at breakfast, and so on. It is important to distinguish between (*a*) the memory of recent events and (*b*) the memory of older occurrences. Both should be tested in every case, as one or the other may be lost alone; in the psychosis which often accompanies alcoholic neuritis, for instance, the patient may retain his memory of events of his childhood, but may remember nothing that has happened during his illness. Inquire as to his **sleep,** and whether or not he is troubled with dreams.

Note whether or not he is more **emotional** than is normal. An abnormal emotional state may be

evidenced by the patient's bursting into laughter or into tears on very slight provocation, or by his giving way easily to fits of anger.

In the course of taking his case, one will already have arrived at a general notion of the degree of the patient's intelligence. Sometimes it is necessary to ascertain whether he is the subject of **hallucinations** or **delusions.** An hallucination consists essentially in an imaginary sense-impression. A delusion is an erroneous idea which would be incredible to the patient's equals, and which is unshaken by facts. If the patient says he hears voices when no one is present, or if he sees persons or forms which do not exist, he is the subject of an hallucination—in the former case auditory, in the latter visual. If he declares that he is the Emperor of Russia, he is the victim of a delusion. The existence of hallucinations and delusions is often difficult to ascertain. Sometimes they are discovered by chance ; in other cases they can be elicited by skilful questioning ; often they are reported by the friends.

Delirium or **coma** may be present ; in such a case the investigation of the intellectual faculties already described is futile.

One should next proceed to the investigation of the **speech functions.*** In considering speech it is essential to distinguish between defects of articulation and enunciation, and those disturbances of speech, due to disease of its cerebral mechanism, which we speak of as aphasia.

Supposing that the patient is able to speak, one should note whether there is any peculiarity in his

* In our description of the methods of clinically investigating the speech functions we have followed very closely the teaching of Prof. Wyllie (*see* his valuable work on "Disorders of Speech").

articulation. The following are the chief abnormalities which may be present :—

1. **Stammering.**—This requires no special description.

2. **Lalling, or baby speech.**—Ask the patient to read something aloud. If he lalls, one will recognize that all the difficult consonants are dropped ; he speaks like a baby, and, if a child, may perhaps make use of words of his own invention. P, B and M, T, D and N are the easiest consonants ; K, G, S, Sh, and Ch are more difficult ; C and L are the most difficult of all. Thus, such a patient has no difficulty in saying "papa," "mamma" ; but if asked to say "British Constitution," he will probably pronounce it "Bitte tontitu." *

3. **Scanning, or staccato speech.**—The patient speaks slowly and deliberately, syllable by syllable, as if scanning a line of poetry. Ask him to say "artillery" ; he will pronounce it "ar-til-ler-y." This is the kind of speech found in cases of multiple cerebro-spinal sclerosis.

4. **Slurring speech.**—The syllables are slurred together as in a state of intoxication. Thus, "British Constitution" becomes "Brizh Conshishushon." This kind of speech is met with very typically in general paralysis of the insane.

5. **Syllable-stumbling.**—In this condition the patient misplaces some of the letters in a word, and reiterates some particular syllables. For instance, "West Register Street" becomes "West Regigistreter Street."

6. **Dysarthria** is due to paresis of the peripheral mechanism of speech, either of the larynx, tongue, or lips, though often all three are affected together.

* An aggravated form of lalling in which the patient seems to speak a language of his own is described as *idioglossia*.

When it is severe (**anarthria**), sounds can be no longer emitted, as is the case in advanced bulbar or pseudo-bulbar paralysis.

If the patient's defect consists not in any perversion of articulation, but in an inability to produce speech at all, or to understand it when spoken or when written, then his condition is described as one of **aphasia.**

In order to understand the method of investigating a case of aphasia, it must be remembered that for purposes of speech we have (1) a producing mechanism. This consists of two parts—one concerned in the production of spoken speech, the other in the production of written speech. (2) A receiving mechanism. This also consists of two parts—one for the reception of spoken speech, the other for the reception of written speech.

We may thus classify cases of aphasia as follows :—

1. Lesions of productive mechanism (motor aphasia).	Motor aphasia (loss of power of talking). Agraphia (loss of power of writing).
2. Lesions of receptive mechanism (sensory aphasia).	Auditory (word-deafness). Visual (word-blindness).

It must be borne in mind, however, that it is the exception to meet with a case of aphasia of a pure type. Thus, a patient may have both motor aphasia and also word-deafness; he may be unable to read as well as unable to write; and so on.

The *cortical centres* for the production and reception of speech are situated in the left cerebral hemisphere in right-handed persons, in the right hemisphere in the case of those who are left-handed. Hence the importance of ascertaining early in the investigation of a nervous case whether the patient is right- or left-handed.

The centre for spoken speech occupies the posterior extremity of the 3rd frontal convolution (Broca's convolution), and the lower end of the ascending frontal.

The centre for the production of written speech is believed to be in the posterior end of the 2nd frontal convolution.

The centre for the reception of spoken speech is in the posterior half of the superior temporo-sphenoidal convolution, and that for the reception of written speech (visual speech centre) in the angular gyrus (*see* Plate 10, facing p. 410).

The visual speech centre is connected by special fibres with the primary visual centre in each occipital lobe. Hence, a lesion in the left occipital lobe does not produce word-blindness unless it be so situated as to cut off also the fibres which connect the visual centre in the right occipital lobe with the left angular gyrus (Fig. 119).

For practical purposes it is best to proceed with the investigation of any case of aphasia in the following order :—

I. Broken Speech

1. *How is it received and interpreted?*—Find out, first, whether the patient's hearing is good. If so, ask him to put out his tongue, shut his eyes, etc. If he does so, test him as to his understanding of nouns by asking him to touch his nose, ear, chin, forehead, etc., in turn. Then test his verbs by asking him to smile, to whistle, etc. Finally, put to him longer questions, or give him more complicated orders, as when the disturbance of speech is only slight he may be able to understand simple questions and commands, but not more complicated ones. If the patient responds satisfactorily to these tests, he has evidently no difficulty in interpreting the meaning of words heard—i.e. there is no *word-deafness*.

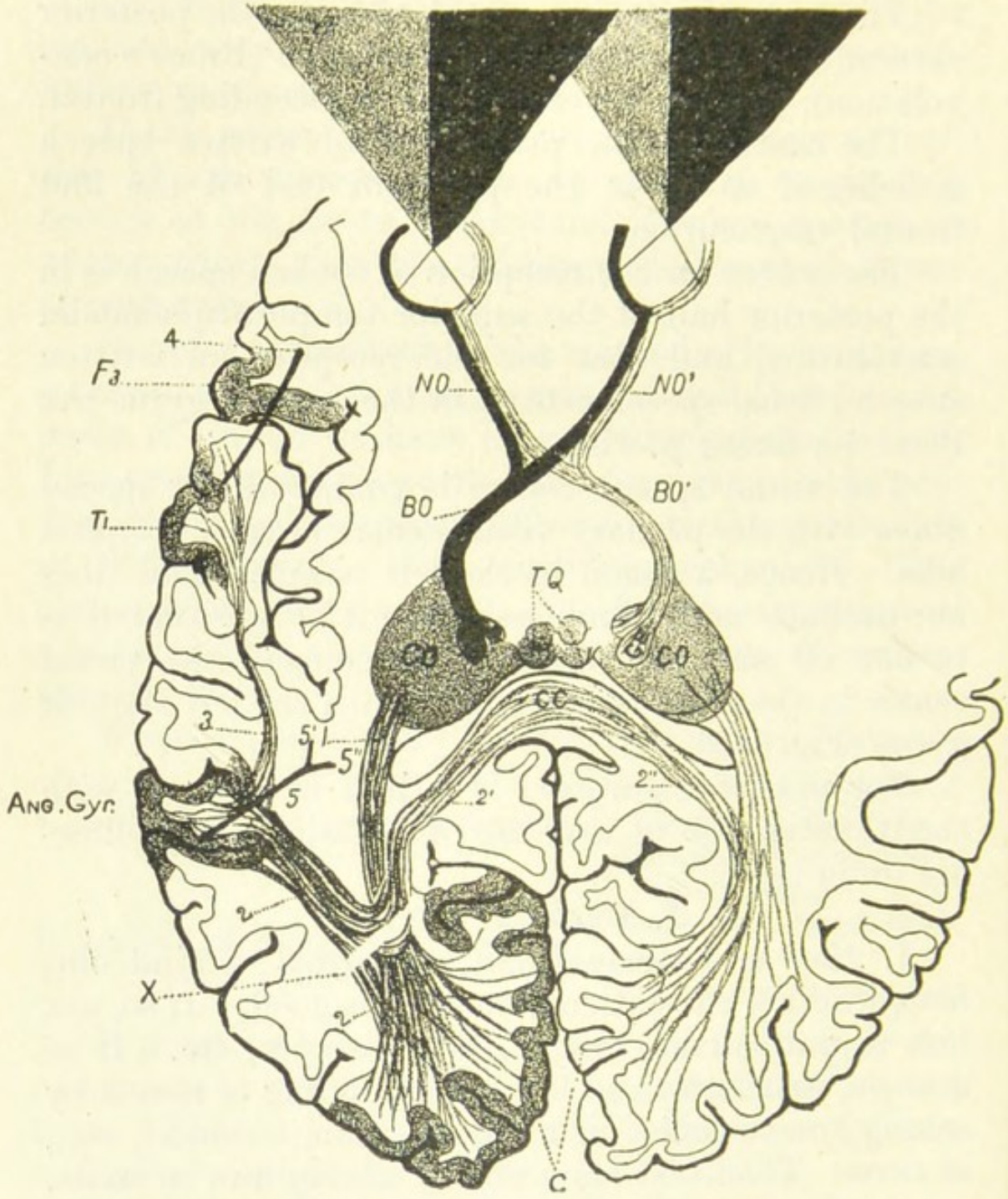


Fig. 119.—Schematic figure, showing the course of the optic fibres.
(From Wyllie, after Déjerine.)

C, cuneus; CC, posterior extremity of corpus callosum; F 3, Broca's convolution (speech centre); T 1, superior temporo-sphenoidal, or Wernicke's convolution (auditory word centre); 1, left optic radiation; 2, fibres connecting left angular gyrus with left cuneus, and through the corpus callosum (2′ and 2″) with the right cuneus; a lesion at x cuts these fibres as well as the optic radiation, and therefore causes right lateral homonymous hemianopsia—word-blindness—but no agraphia; 3, fibres connecting angular gyrus with Wernicke's convolution. The straight black line (4) represents the connection between Broca's and Wernicke's convolutions; the black line bifurcating in front (5) represents the connections of the angular gyrus with the motor region of the left (5′) and right (5″) hemispheres.

2. *How is it produced?*

i. If the patient can only use a few words, make a note of what these are. If he repeats any word or phrase again and again (**"recurring utterance"**), note what it is.

ii. If he has a considerable vocabulary, (*a*) make a note of any examples of lalling, slurring, etc., as described on p. 433. This affords an indication of his *power of articulation.*

Test him with such words and phrases as "British Constitution," "West Register Street," "Biblical criticism," "artillery."

(*b*) Show him common objects—a knife, a pen, a matchbox, etc.—and ask him to name them; or, if he is dumb, to indicate with his fingers the number of syllables in the name of each. If he is unable to fulfil these tests, he has evidently got some forgetfulness of words (**amnesia verbalis**). Sometimes the patient has a general idea of the word he wants to use, but forgets exactly how to pronounce it; he omits some syllables, or substitutes others for them, so that the listener may hardly be able to make out what word it is he wishes to use. This has been termed by Wyllie *articulative amnesia.*

(*c*) If he makes mistakes in his use of words, calling the knife a pen, or vice versa, he is suffering from **paraphasia.** In that case, one should note whether or not the patient shows that he is aware of his error by trying to correct himself, or whether he goes on talking gibberish.

3. *How is it repeated or echoed?*—Ask him to repeat words after you. If he is word-deaf, try to make clear your request by the aid of pantomime, repeating the word or phrase over and over again. If he is able to repeat what you say, endeavour to find out whether or not he understands what he is saying.

II. Written Speech

1. *How is it received or interpreted?*—Ascertain whether or not his sight is good. If so, write on a piece of paper such questions or commands as, How old are you? Put out your tongue; etc. If he does not respond satisfactorily, there is some word-blindness present—i.e. the patient has **visual aphasia.**

2. *How is it produced?*—Ask him to write his name. (This can often be done when all other power of writing is lost.) If he is able to do so, ask him some simple question—e.g. How many do two and two make?—and get him to write a reply. If he has word-deafness, put your question in writing. If his right hand is paralysed, make him write or print with his left. If he writes pretty well, get him to write an account of his illness, and note whether he makes use of the wrong word at times (**paragraphia**), or whether there is repeated use of any particular word.

3. *Can he write to dictation or copy?*—Try, using some simple book. If he succeeds, endeavour to ascertain whether or not he understands the meaning of what he writes.

III. Phenomena associated with Speech

1. *Does he understand pantomime?*—Does he nod his head for "yes," shake it for "no," and can he indicate numbers with his fingers? Loss of gesture language is termed **amimia.** Mistakes in the use of gestures—e.g. nodding for "no," or shaking the head for "yes"—is termed **paramimia.**

2. *Does he understand symbols*—e.g. numerals?—Thus, one may write down—

2	2	2
2	2	2
—	—	—
4	5	6

and ask him to point out which is right. If he can read music, test him with musical notes.

3. *Can he recognize common objects ?*—Place beside him a pencil, a coin, and a match. Ask him to strike a light, or to write something down. If he is unable to select the proper article for the purpose, he is suffering from **mind-blindness.** Inability to recognize his friends is another proof of the same condition.

It occasionally happens that a patient who has neither motor nor sensory paralysis, nor ataxia, cannot perform certain acts, though he can easily execute their component movements. He is consequently unable to make use of objects though he can recognize their use. This condition is known as **apraxia.** It results from destruction of the left frontal lobe, or of its connections, through the corpus callosum, with the right hemisphere. Sometimes it affects only the left limbs, but it is usually bilateral. It may be tested for by asking him to use certain objects, or make or imitate certain movements. For instance, he may be given a box of matches and a candle, and asked to light the latter. If there is apraxia he may fail to open the box, or to take a match from it, or to strike the match, or even to light the candle with the match if he has succeeded in striking it. It is, of course, important to make sure that the patient understands the order.

III. CRANIAL NERVE FUNCTIONS

In this section we propose to give a brief résumé of the essential points in the anatomy of each cranial nerve, to indicate its functions, and, in some cases, the chief symptoms which result from its paralysis, and then to describe the method in which one investigates the state of the nerve at the bedside.

First or Olfactory Nerve

Anatomy.—The nerve fibres which arise from the olfactory bulb are distributed to the Schneiderian membrane, at the upper part of the nasal fossæ. The cortical centre for smell is believed to lie in the uncinate gyrus. The exact course of the fibres between the cortex and the bulb is unknown, but it is probable that some of them do not decussate.

Test.—Have three small bottles containing some oil of cloves, some oil of peppermint, and some tincture of asafœtida. Apply these to each nostril separately, and ask the patient if he recognizes them. In testing, avoid the use of such irritating substances as ammonia, for these act, partly at least, through the 5th nerve. The sense of smell may be abolished. This is known as **anosmia.** Before concluding that the nerve is at fault, take care to exclude local changes in the nose itself—e.g. catarrh. **Parosmia** is the name applied to a condition in which the sense of smell is perverted, so that, for instance, offensive substances seem to have a pleasant odour, and vice versa.

Inquire also regarding **hallucinations of smell.** These sometimes constitute the aura of an epileptic fit.

Second or Optic Nerve

Anatomy.—From the retina, which is the end-organ of the sense of sight, the fibres of the optic nerve pass back to the optic chiasma. Here the fibres from the inner half of each retina decussate, whilst those from the outer half remain on the same side. Each optic tract, therefore, consists of fibres from the outer half of the retina on the same side and the inner half of the retina on the opposite side. Each tract passes back to the corpora quadrigemina and to the external geniculate body and the pulvinar of the optic thalamus of the same side. In these, which are known as the primary optic centres, all the fibres of the optic tracts terminate. But another system of fibres, which is known as the optic radiations, takes origin in them and passes through the posterior limb of the internal capsule and then backwards to the cortex around the calcarine fissure (*see* Fig. 119, p. 436). This, therefore, consti-

tutes the chief visual centre, and represents the opposite half of the field of vision, the left half of the field of vision being represented in the cortex of the right hemisphere, and vice versa.

Test.—In testing the optic nerve, one has to investigate three functions : (1) Acuity of vision ; (2) extent of field of vision ; (3) colour sense. We shall consider the methods of testing these *seriatim.*

Certain preliminaries must always be attended to. One of these is to see that any error of refraction in the patient's eyes is first corrected, and that there is no opacity of his media ; another is to take care to examine each eye separately.

1. **Acuity of vision.**—If this be very much diminished, it may be doubtful whether the patient is able to tell light from darkness. To investigate this, place the patient in a darkened room opposite to a lamp, alternately cover and uncover his eye, or, what is perhaps a better plan, concentrate the light upon his eye by means of a mirror or lens, and ask him to say when it is light and when it is dark.

In lesser degrees of impairment, ask the patient to count fingers. This is done by placing him with his back to the light while the observer, standing facing the patient, holds up a varying number of fingers of one hand, and asks the patient to say how many there are. The test should be applied at varying distances.

For the detection of slight degrees of impairment of visual acuity Snellen's types will be found useful. These consist of letters of different sizes, each of which should be capable of being read at a definite distance—the largest at 60 metres, the smallest at 6. In using the types, the patient is placed with his back to the light, while the types are placed level with the eye at a distance of 6 metres (about 20 ft.). He is then asked to read the letters from above downwards.

For the purpose of recording the result, the following symbols are employed :—

V = visual acuity.
d = distance of eye from type (i.e. 6 metres).
D = distance at which type should be capable of being read.

Suppose that at 6 metres the patient is able to read the smallest type—that is to say, that which should be readable at 6 metres off. Then his visual acuity (V) $= \frac{d \text{ (i.e. 6 metres)}}{\text{D (i.e. 6 metres)}}$ or normal.

But if at that distance he can only read the largest size of type—that which one should be able to read at 60 metres—then $V = \frac{6}{60}$.

The term **amblyopia** (literally "blunt-eyedness") is often used to mean defective vision. By **crossed amblyopia** one means dimness of vision in one eye, there being a lesion in the opposite half of the brain. It also occurs, not infrequently, in hysterical hemi-anæsthesia, on the same side as the loss of sensation. The term **amaurosis** (literally "darkness") is sometimes used to signify complete blindness.

2. **Extent of field of vision.**—For ordinary clinical purposes the extent of the field of vision can be tested with sufficient accuracy in the following way :—

Seat yourself opposite to the patient and at a distance of about half a yard from him. If his right eye is to be tested, ask him to place his hand upon his left, and to look steadily at your own *left* eye. Look steadily yourself at the patient's right eye, your own right being closed, and hold up your left hand in a plane midway between his face and your own, and at first at almost full arm's length off. Keep moving the fingers of the hand, and bring it nearer until you

can just yourself "with the tail of your eye" catch the movement of the fingers. Then ask the patient whether he sees them, telling him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he sees them. Test the field in this fashion in every direction —upwards, downwards, to right, and to left—using

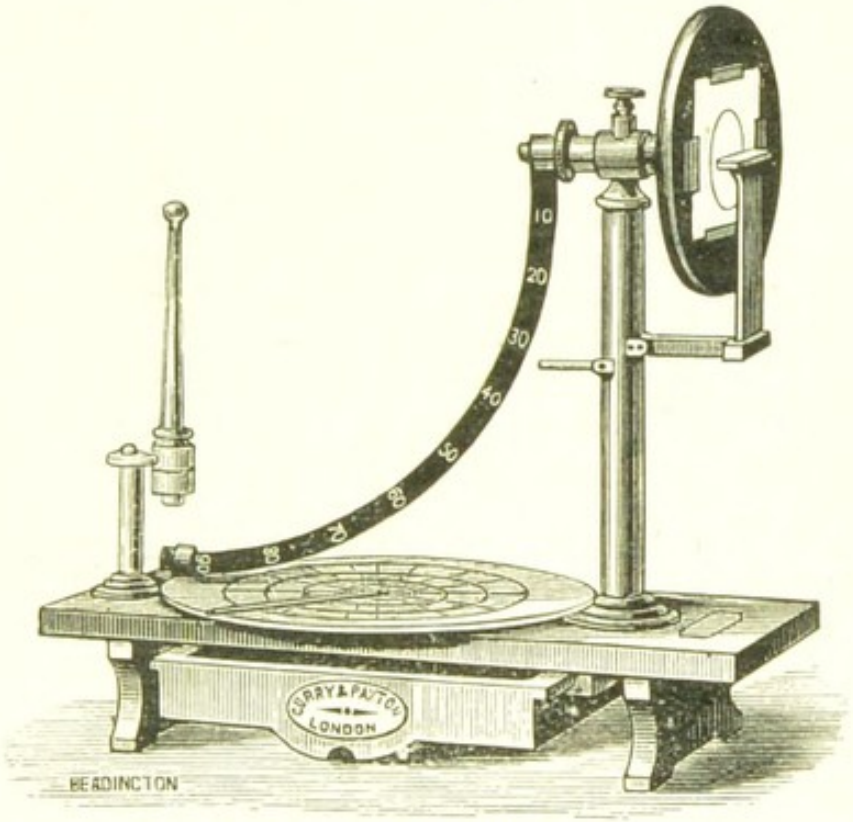


Fig. 120.—Priestley Smith's perimeter.

the extent of your own field always for purpose of comparison.

For more accurate delimitation of the field of vision one must make use of a **perimeter.** Priestley Smith's is a good form, and is shown in Fig. 120. It is used as follows :—

i. The patient rests his cheek against the wooden pillar, so that the eye is about 1½ in. above the knob and vertically over it. The height of the instrument is regulated by movable blocks.

ii. The quadrant, which is a flat strip of metal engraved upon its two sides, is rotated by a wooden hand-wheel attached

to the axis ; it is balanced by a weight upon the hand-wheel, so that it will stand in any position without being fixed.

iii. The test object is a square of paper gummed upon a light vulcanite wand which the operator holds in the left hand. With the right hand he rotates the hand-wheel and pricks the chart.

iv. The chart is placed upon the hinder surface of the hand-wheel, and rotates with it. There is a mark on the hand-wheel to show which way the chart is to be placed. This mark is brought to the top, and the chart is then slipped in from above downwards and in the upright position.

v. Immediately behind the hand-wheel is fixed a hozizontal scale, the divisions of which correspond with the circles on the chart. As the quadrant rotates the chart rotates with it, and, in whatever position the quadrant stands, the corresponding meridian of the chart stands against the scale. This arrangement enables the operator to prick off his observations with the greatest ease, and has the further advantage that the chart is constantly under inspection, so that any portion of the field can be immediately brought under examination at any time.

vi. The charts are of two kinds, A and B. The A charts correspond to the entire field and are divided by circles from 0° to 90°, the limits of the average normal field being shown by a dotted line. The B charts are for mapping the central part of the field on a larger scale, and are divided from 0° to 45°. The scale of the perimeter is graduated accordingly on its two sides ; the A side is to be used with the A charts, the B side with the B charts.

vii. There are many cases in which it is better to sweep the field, or parts of it, in circles rather than in meridians, e.g. hemiopic and sector-like defects in which the boundary line of the field runs in a meridianal direction. In cases of this kind the test objects may be placed in the clip upon the quadrant, and carried round the field in successive circles.

McHardy's instrument is shown in Fig. 121. The following points should be attended to in using it :—

The chin rest (E) is adjustable to suit each patient; the level of the eye is then gauged by the button (I) on the sliding stem (H) ; next, " zero " is raised, and fixed by the milled head (*b*) to the level of eye, by aid of scales on *a* and K. The biting fixation attachment (L, M) is detachable ; it is only to be put in place when required in mapping the " field of fixation," as

distinguished from the "field of vision." Registration is accomplished by simply pressing the chart box (*e*) into an upright position. The quadrant (*h h*) rotates smoothly, and remains rigid in any position. The large vulcanite disc (*n*) readily slides into position, so as to screen the operator's

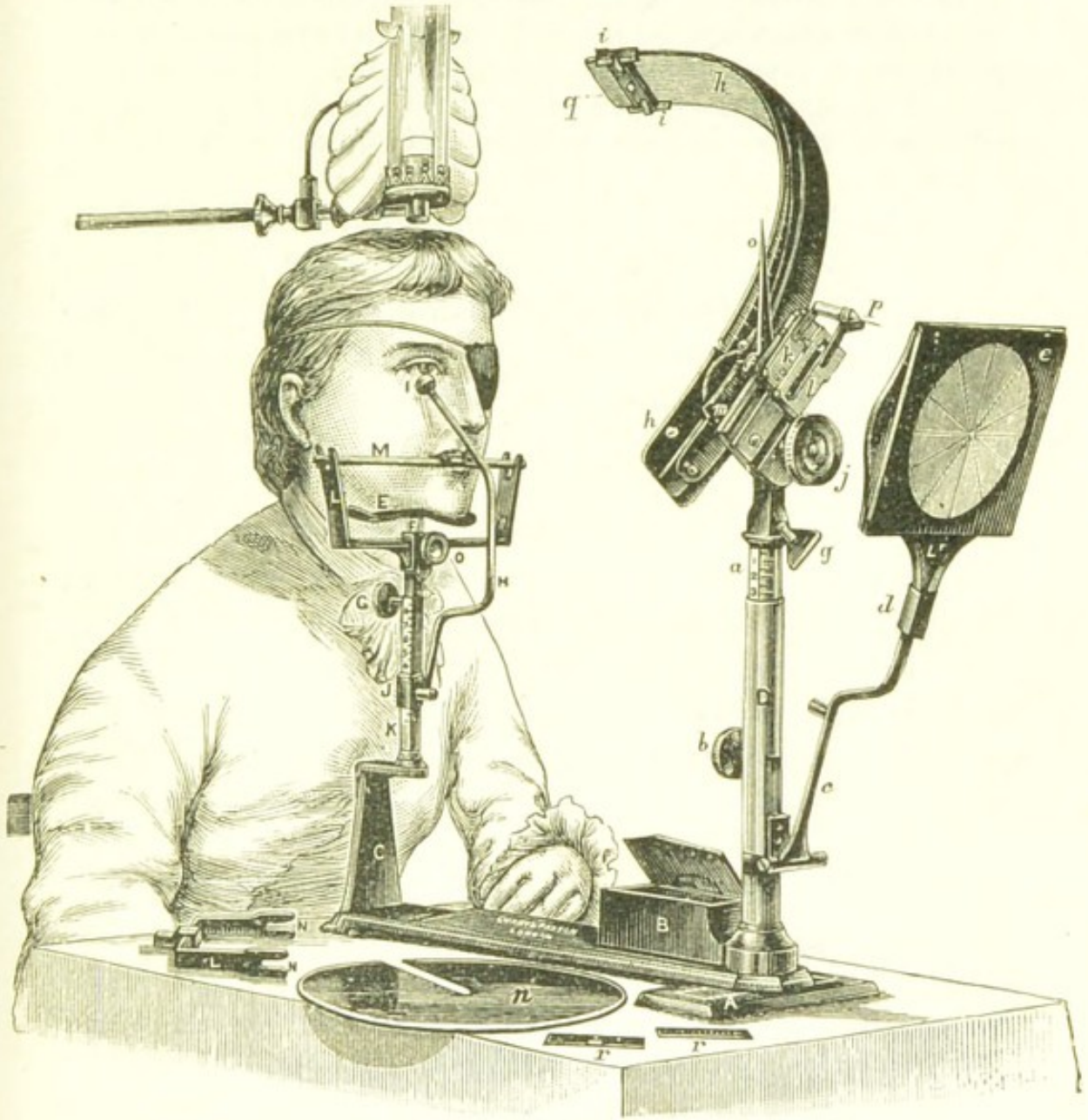


Fig. 121.—McHardy's perimeter. (For details, *see* text.)

cuff while he turns the driving milled head (*j*), which simultaneously manipulates the travelling carriage (*i*) and the pointer (*p*) of the automatic registration. The skeleton charts, which are printed red, correspond to the entire field, and are divided by circles from 0° to 90°, the limits of the average normal field being shown by a faint line. Any portion of the field

can be immediately brought under examination, while the records are not liable to be biased as when under inspection. The field or parts of it may be as readily swept in circles as in meridians, in those cases in which the former method is preferable; e.g. for hemiopic and sector-like defects.

The test objects, circular carriers of coloured paper, are placed in the carriage (*i*) behind various diaphragms (*r r*) with apertures of given diameter. Degrees and half-degrees are marked on the back of both edges of the quadrant, so that the instrument will determine the angle of squint or the angle *a* accurately.

Ascher's perimeter, which is constructed mainly of celluloid, is a light and portable form for private use.

Changes in the field of vision.—It may be contracted all round its periphery. This is spoken of as **concentric diminution** of the field of vision. It occurs in hysteria, optic atrophy, and various affections of the retina.

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a **central scotoma** or as **central amblyopia.** It is frequently due to toxic causes (e.g. excess in tobacco or alcohol), and is then generally bilateral. Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula. In that case it may affect only one eye.

The term **hemianopsia** (also written hemianopia and hemiopia) means loss of sight in one-half of the field of vision in both eyes from causes other than disease in the retina. Right lateral hemianopsia means abolition of the right half of the field of vision; left lateral hemianopsia, abolition of the left half. These forms of hemianopsia are also spoken of as *homonymous.*

Superior and *inferior hemianopsia* mean loss of the upper and lower halves of the visual field respectively. They are rarer than the lateral variety, and are sometimes spoken of as *altitudinal hemianopsia.*

Temporal hemianopsia means loss of vision in the temporal or outer halves of both fields, and is due, therefore, to loss of visual power in the nasal half of each retina. It can only be produced by a lesion of the optic chiasma, involving those fibres of the optic nerves which decussate, and is accordingly rare. It occurs in acromegaly, or with tumours of the pituitary body, or may result from a syphilitic meningitis compressing the chiasma.

Nasal hemianopsia signifies a loss of the nasal or inner half of each field, and indicates a diminution of visual power in the temporal half of each retina. It can only be produced by a bilateral lesion confined to the uncrossed optic fibres on each side of the chiasma. It occurs with excessive rarity.

Temporal and nasal hemianopsia are sometimes spoken of as *heteronymous*, in contradistinction to the *homonymous* variety.

3. **Colour sense.**—This is tested by means of Holmgren's wools. Throw all the skeins together on a table in good daylight, keeping the test skein separate. Explain to the patient that he is to match the colour, not to name it, and that he is to select all those skeins which are *like* it, whether they are of a darker or lighter shade or not. Show him first a pure pale-green skein, and ask him to match it. If he does so correctly, his colour vision is normal. If, on the other hand, he selects one of the "confusion colours" (grey, straw colour, etc.), he is to be regarded as colour-blind.

Total colour-blindness is rare. Red-green blindness is the commonest form. Yellow-blue blindness is not nearly so common. If the patient is totally colour-blind he confuses with the test skein all those of equal brightness, no matter what their tint may be. If red-green blindness is suspected, show him a

purple skein, and he will select blue as a match for it—indicating that he fails to see the red element in the purple. If he be blue-blind, he will select red or orange.

Colour field.—In a normal eye the field for blue is largest, then comes yellow, then red, and lastly green. Concentric diminution of the colour field occurs in hysterical amblyopia. In some toxic conditions, especially tobacco poisoning, one finds a central scotoma for colour. Its existence can easily be determined by placing the patient with his back to the light, and asking him to look steadily at any spot. A small piece of green or red paper is then placed over the spot which he fixes, and he is asked to distinguish the colour. If a central colour scotoma is present he cannot distinguish the colour when it is on the fixation point, but may be able to do so when it is moved some distance from it. The extent of the scotoma may be measured by the perimeter. Colour-blindness, of course, interferes with the use of the test in some cases.

The exact extent of the field for each colour is best tested by means of the perimeter.

Subjective visual sensations may be present. Amongst the commonest of these for which one may have to inquire is the occurrence of what are known as *muscæ volitantes*—little specks or motes seen floating before the eyes, especially on looking at a white surface or up to the sky. They are not infrequent in anæmic and debilitated persons. In migraine, peculiar zigzag lines, known as "fortification figures," are often seen at the beginning of the attack, and in the investigation of such a case should always be inquired for. The term *teichopsia* is applied to this condition. Hallucinations of sight occur in some cases, notably in delirium tremens; they may also form part of the aura in epilepsy.

Third, Fourth, and Sixth Nerves

It is convenient to take these together, as conjointly they serve to innervate the muscles which move the eyeball.

Anatomy.—The fibres of these nerves take their origin from a series of nuclei which begin in the floor of the aqueduct of Sylvius below the anterior corpora quadrigemina, and extend down as far as the eminentia teres in the floor of the 4th ventricle. The nucleus for the 3rd nerve is highest up; its most anterior cells supply the ciliary muscle and iris, those for the ocular muscles being farther back. Behind that comes the nucleus of the 4th, and most posteriorly of all that of the 6th. The 3rd nerve emerges on the inner aspect of the crus, and is therefore apt to be involved in lesions implicating that part of the brain.

The 4th pair emerge on the anterior part of the roof of the 4th ventricle. They are peculiar in that they are the only cranial nerves which decussate between their nuclei and their point of emergence.

The 6th emerges between the medulla and pons, and runs forward beneath the latter for a considerable distance before leaving the skull. This long course renders it particularly liable to the effects of pressure.

Functions.—The 6th nerve supplies the external rectus, the 4th supplies the superior oblique. All the other ocular muscles, along with the sphincter pupillæ, the muscle of accommodation, and the levator palpebræ superioris, are supplied by the 3rd.

Symptoms of paralysis. *Sixth nerve.*—Inability to move the eye outwards, and diplopia on looking in that direction. Possibly internal squint. In nuclear lesions there is also loss of the power of conjugate deviation of both eyes in the direction of the affected muscle.

Fourth nerve.—Impaired power of downward movement, and on the attempt to look downward the eyeball is rotated outwards by the inferior rectus. Diplopia only below the horizontal plane, with the

images uncrossed, but the false one tilted. There is rarely a visible squint.

Third nerve.—Ptosis; the eye can only be moved outwards and a little downwards and inwards; pupil dilated and unable to contract; loss of power of accommodation.

Paralyses of the 3rd nerve are not infrequently partial—only one or a few of these functions being lost.

Thus the levator palpebræ superioris is often alone affected, producing **ptosis,** while the other muscles retain their normal power. In order to estimate the degree of ptosis, one must eliminate the action of the occipito-frontalis. This is done by pushing down upon the latter muscle so that the eyebrows are kept level, and then asking the patient to look up. The extent to which the lids are raised indicates the strength of the levator.

Any **retraction of the upper lid,** from over-action of the levator, is to be noted by observing the relation of the edge of the lid to the upper margin of the cornea when the patient is looking straight forward.

How to test these nerves.—As will be gathered from the above résumé, the signs of a lesion involving any of these nerves may be—1, the presence of a squint; 2, defective power of movement of the eye; 3, the presence of diplopia. Of these signs the last is really the most trustworthy of all, for paralysis of the muscles supplied by the nerve may be so slight as to lead to no appreciable squint and to no visible defect in mobility.

We shall consider the question of squint first.

1. By **squint** or **strabismus** is meant a want of parallelism in the two visual axes. It may be due either to paralysis of a muscle or to over-action—i.e. spasm—of its opponent. The former constitutes

paralytic strabismus, the other brings about what is called **concomitant** (or *spasmodic*) **strabismus.** The first point, therefore, to be decided about any squint is this—Is it paralytic or is it concomitant? The chief points of distinction between the two are these :—

(*a*) Spasmodic squint is always present ; on asking the patient to look straight forward, which is the position of rest of all the ocular muscles, the squint is seen at once. Paralytic strabismus, on the other hand, may only be visible when the patient happens to look in a direction requiring the action of the paralysed muscle.

(*b*) In spasmodic strabismus the affected eye follows the sound eye equally in all its movements. It is for this reason that it is termed " concomitant." The visual axes are not parallel even in the position of rest, and the defect of parallelism remains the same in whatever direction the patient turns his eyes. In paralytic squint the visual axes may appear parallel in the position of rest, but, even if they do not, the want of parallelism becomes more and more evident the farther the patient tries to turn his eyes in the direction of action of the paralysed muscle.

(*c*) Diplopia is usually present in paralytic squints, absent in those which are spasmodic.

The commonest form of concomitant squint is the internal strabismus, which occurs in children, and is associated with hypermetropia.

2. **Defective power of movement of the eye.**—In order to elicit this symptom, place the patient with his face to the light ; stand in front of him, and, holding up one finger, ask him to follow its movements with his eyes. It will easily be noticed whether or not a squint is brought out in either eye when he attempts to do so. The examination may be

repeated in each eye separately, its mobility being tested in each direction.

The power of convergence of the eyes should always be tested in addition to the mobility of each eye separately. In order to do this, hold up your finger directly in front of the patient at a distance of about 18 in. from the tip of his nose. Tell him to keep looking at the finger, which is then gradually brought nearer to the nose. Note to what extent convergence occurs, and whether it is well maintained—i.e. whether the eyes remain directed towards each other, or whether they diverge again after their first convergence.

In some cases the defective mobility is so slight as to elude detection. This is especially apt to be the case in paralysis of the oblique muscles. In such a case one falls back upon the diplopia as an indication of the affected muscle.

3. **Diplopia.**—In order to elicit this symptom, hold your finger straight up in front of the patient, and ask him how many there are. Then repeat the question with the finger held at each side of the visual field, then high up, and then low down. Make sure that the patient's head is not moved during the investigation. If in each position he sees one finger only, there is no diplopia. If at any part of the field two fingers are seen—one distinct, the other somewhat hazy—one may be sure that diplopia is present.

In that case one has next to ascertain (1) which is the affected eye ; (2) which is the affected muscle in that eye.

In order to determine these points, proceed as follows :—

Place over one of the patient's eyes a red glass—preferably over the stronger eye, if he has better vision in one than the other. Then hold up in front

of him a tall lighted candle. Move it about until he sees two candles—a red and a yellow. One of these is the *true* image—i.e. that of the sound eye; the other is the *false* image—i.e. that of the affected eye. Which is which? In order to answer this question the following rule is given:—

The affected eye is that in the direction of the image of which the diplopia increases.

The application of this rule will be made plain by an example. Suppose the red glass is opposite the patient's left eye, and the patient says that the red image is to the right of the yellow. On moving the candle farther to the right the distance between the images increases—i.e. the diplopia becomes greater; that is to say, it becomes greater on moving the candle in the direction of the red image. But that is the image belonging to the left eye; therefore, applying the rule, the left eye is the one that is affected.

The affected eye having been thus discovered, one wishes to know which is the affected muscle. To help in this a second rule has been given:—

The paralysed muscle is that which would have turned the eye in the position and direction of the false image.

In the above example it was found that the left eye was the one affected, and that its image was to the right of the true image. Applying the above rule, one asks, Which muscle moves the left eye to the right? and the reply is, The internal rectus. The left internal rectus, therefore, is the affected muscle.

When, as in this case, the false image is on the opposite side to the affected eye, the diplopia is said to be *crossed;* when the false image is on the same side as the affected eye, the diplopia is said to be *direct.* Paralysis of an internal rectus always produces a crossed diplopia; paralysis of an external

rectus, a direct diplopia. The rule is, that when the optic axes would intersect if prolonged the diplopia is not crossed, and vice versa.

The detection of the affected muscle in cases of **vertical diplopia** is somewhat more difficult than in cases where the diplopia is lateral. The same rules, however, apply. Suppose, again, that the red glass is over the left eye, and that the two images are seen one above the other, the yellow being higher up. On looking upwards the distance between the images becomes greater; therefore, according to the first rule, the right eye is the one affected. *Which is the paralysed muscle?* In order to apply the second rule, it is necessary to remember the action of each muscle, and, from the position of the false image as described by the patient, deduce which muscle it is that would have turned the eye in that position and direction. This muscle will be the one affected. Werner's diagrams (Figs. 122, 123) facilitate the recollection of this greatly. The continuous lines on the diagrams represent the positions of the true images, the dotted lines those of the false images. Thus in paralysis of the left inferior rectus the false image is at a lower level than the true, it is to the right side of the true image (crossed diplopia), and the upper end is inclined towards the true image (Fig. 122). In paralysis of

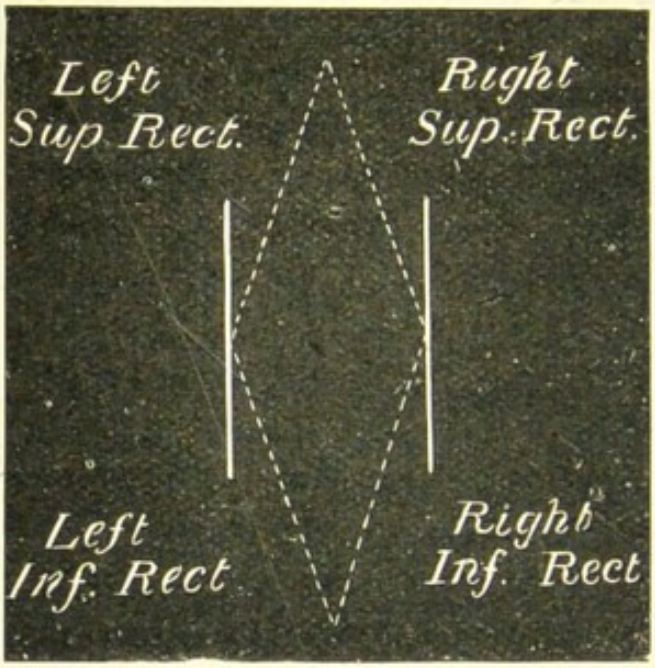


Fig. 122.—Werner's diagram.

the left inferior oblique, the false image is higher up than the true image and to its left side (direct diplopia), and its upper end is inclined away from the true image (Fig. 123).

To return, then, to our supposed case. One has proved that the right eye was the one affected, and that its image was higher up than the true image. The paralysis must therefore affect either the right superior rectus or the right inferior oblique. Ask the patient whether the upper end of the false image is inclined towards or away from the true, and whether it is to the right or the left of the latter. Supposing he says that the false image (yellow candle) is to the left of the true, and with its upper end inclined away from the latter: then one knows at once, by referring to the diagram (Fig. 122), that the right superior rectus is the muscle affected.

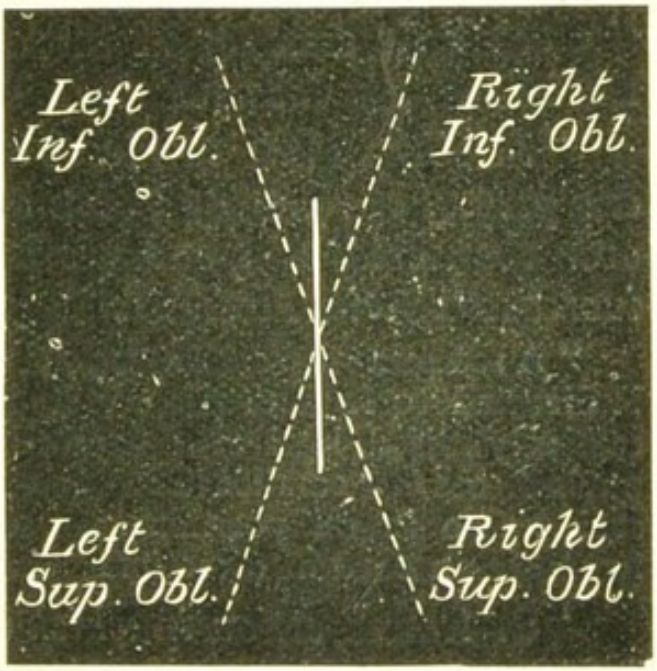


Fig. 123.—Werner's diagram.

If, in the above example, the patient be unable to state clearly whether the false image is to the right or left of the true, and what is the exact nature of its inclination, one can have recourse to another method. Ask in what direction the difference in height of the two images increases. If towards the temple, the rectus is the affected muscle; if towards the nose, it is the oblique.

In applying the above tests it is more convenient to move the patient's head in order to change the

direction of his eyes than to move the candle. Thus if one turns the head to the left and asks him to look at the candle the eyes are turned to the right. Hence, moving the head to the left comes to the same thing as moving the candle to the right, and vice versa. To lower the eyes, hold the head back; to elevate the eyes, depress the chin. These movements are effected by the observer, who places himself at the patient's side, the lighted candle being placed on a table some distance off.

An alternative method of diagnosis in paralyses of the extrinsic muscles of the eye is recommended by MacNab. He describes it as follows * :—

"1. *A muscle is paralysed when a diplopia occurs in one part of the field of vision or when there is diplopia over the whole field and the separation of the images increases in one direction.*

"A red glass is placed before the right eye, and the patient is directed to look towards a lighted candle, which is successively raised, lowered, moved to the right and left. If the patient sees two candles in some positions but not in others, or, seeing two in all positions, the distance between the two increases in certain directions, there is a muscle paresis.

"2. *The direction in which the diplopia is greatest* † *indicates the direction of action of the affected muscle.*

"In testing for the direction of the greatest diplopia the horizontal and the vertical components must be considered separately. The candle is held straight in front of the eyes, and the patient is directed to note how much one image is to the right of the other, and for the present to neglect entirely any difference in level; he should be asked to estimate in inches the distance between them and to hold up his two index fingers in their respective positions. The patient's head being then kept steady, the candle is moved to the right and to the left. If any difference is seen it should be noted as horizontal separation increasing to the right or to the left. The

* *Lancet*, June 19th, 1909.

† The expression "diplopia is greatest" is used to mean the angular separation of the two images is greater.

patient's attention is then directed to the difference in level —i.e. the vertical element of the diplopia—and in the same way on raising the candle or lowering it the patient will readily estimate any difference in the level of the two images ; a note can then be made that the vertical separation increases upwards or downwards. In determining the variations in the two components of a diplopia it is imperative that the patient's attention be kept to the point ; when testing the horizontal separation no remarks about level should be listened to ; to the question, ' How far is the one candle to the right of the other ? ' the answer, ' The red candle is now sloping,' only confuses the observer.

" If the vertical component is very small relatively to the horizontal the diplopia can be spoken of as a horizontal one ; the action of the affected muscle will be horizontal, and to the right or left according as the diplopia increases to the right or to the left. If the vertical element is greater than the horizontal the diplopia can be spoken of as a vertical one, and the muscle affected will be an elevator or a depressor according as the vertical element of the separation increases upwards or downwards.

" 3. *The image projected further in the direction of greatest diplopia is formed by the affected eye.* When the candle is moved in the direction in which the diplopia increases, the image which moves most rapidly in that direction is the one formed by the affected eye.

" Having determined the direction of action of the affected muscle we now find out which eye is affected. The diplopia is due to the lagging of a muscle, and is greatest when the muscle is called on to make its greatest effort. If the candle be placed in that part of the field where the diplopia is greatest the normal eye will be able to follow it and receive its image on the macula, but the affected eye will lag behind and receive an image of the light at a point on its retina remote from the macula in the opposite direction to that of the object. Taking into consideration the relative positions of these two retinal images, it will be apparent that the one in the affected eye cannot be superimposed on the other, but will always be at a point removed from it in the opposite direction to that of the source of light. If now we consider the mental projection of these two images in space, that one due to the eye which

lags will always be further in the direction of the object fixed. But this is the direction of greatest diplopia, therefore the image which is further out in the direction of the greatest diplopia is always formed by the affected eye. Having determined the direction of the greatest diplopia, we have merely to ask the patient which candle is further in this direction—up, down, right, or left—and his answer will indicate the eye which is affected.

"If the diplopia be a horizontal one the diagnosis will be complete, for there is only one muscle which moves each eye to the right or to the left. If, however, the diplopia be vertical, we have still to decide between two muscles, for each eye can be moved up by the superior rectus or the inferior oblique and downwards by the inferior rectus or the superior oblique.

"4. *The separation of the images is greatest when the eye is looking along the line of the tendon of the affected muscle* or when the object is opposite the end of the affected tendon.

"Bearing this rule in mind, we have only to recall the direction of the tendons to decide the muscle. We have already determined the action of the muscle and the eye which is affected. If the candle be placed in the two positions in which it would be intercepted by the lines of the two possible tendons, then the vertical separation will be greater when it is on the line of the affected one.

"Suppose that the vertical separation increases downwards and the image from the right eye is lower, we have a depressor of the right eye affected. The line of the one depressor tendon —the superior oblique—passes forwards and to the left, and the tendon of the other—the inferior rectus—passes forwards and to the right. The candle is therefore placed in the left lower part of the field, in line with the oblique, and then in the right lower part of the field, in line with the rectus, and the patient is asked in which position is the difference in level greater. His answer will determine the muscle affected.

"If a further control of these vertically acting muscles be wished, and the patient appreciates that one candle is inclined towards the other, then the angle of inclination of one image to the other is always greatest when the line from the object to the eye is at right angles to the tendon of the affected muscle.

"In the case of a depressor we have to distinguish between the superior oblique and the inferior rectus; and similarly for an elevator between the superior rectus and inferior oblique.

For the former pair, hold the candle in the lower part of the field, first to the right and then to the left, and ask the patient to say when the inclination is greater. Then, placing the candle in the position where the inclination is greatest, take a line from it to the eye, and that one of these two muscles whose tendon is at right angles to this line is the one affected. Similarly for the elevators. The line of the superior oblique tendon, of course, is taken as from the pulley to the insertion in the globe."

The application of these rules to the various muscle paralyses taken in detail is as follows :—

Given a diplopia which increases in one direction we have—

A. Horizontal Diplopia

1. Increasing towards the *right* :—
   (*a*) Image formed by the right eye further to the right : *Right external rectus.*
   (*b*) Image formed by the left eye to the right : *Left internal rectus.*
2. Increasing to the *left* :—
   (*c*) Image formed by the left eye to the left : *Left external rectus.*
   (*d*) Image formed by the right eye to the left : *Right internal rectus.*

B. Vertical Diplopias

Separation increasing *upwards.*

1. Right eye's image above :—
   An elevator of the right eye affected.
   (*e*) Vertical separation greater up and to the right than up and to the left : *Right superior rectus.*
   (*f*) Separation greater up and to the left than up and to the right : *Right inferior oblique.*
2. Left eye's image above :—
   An elevator of the left eye affected.
   (*g*) Separation greater up and to the left than up and to the right : *Left superior rectus.*
   (*h*) Separation greater up and to the right than up and to the left : *Left inferior oblique.*

Separation increasing *downwards.*

1. Right eye's image below :—
   A depressor of the right eye affected.
   (*i*) Separation greater down and to the right than down and to the left : *Right inferior rectus.*

(*j*) Separation greater down and to the left than down and to the right : *Right superior oblique.*

2. Left eye's image below :—

A depressor of the left eye affected.

(*k*) Separation greater down and to the left than down and to the right : *Left inferior rectus.*

(*l*) Separation greater down and to the right than down and to the left : *Left superior oblique.*

The **position of the patient's head** is also of considerable help in detecting the paralysed muscle. He tries, by turning his head, to place the affected eye in the position in which it would be independent of the action of the paralysed muscle. Thus, if the face is turned to the left, it indicates a paralysis of the left external rectus or right internal rectus, and vice versa. If it be directed upwards, an elevator is paralysed ; if downwards, a depressor.

Abnormal movements of the eye.—Involuntary rhythmical contractions of the muscles of the eyeball not infrequently occur. The movements are usually symmetrical, occurring equally in both eyes. To these movements the term **nystagmus** is applied.

It may exist during rest, but is usually more pronounced on voluntary movement, and is then greatest in the plane of the movement that is made, that is, it is horizontal on lateral movement of the eyes, and chiefly vertical on upward or downward movement.

In examining for nystagmus, ask the patient to look straight in front of him, and observe whether the eyes remain steady. Then ask him to look to his extreme right, then to the left, and then upwards and downwards. Observe the rate, amplitude, and rhythm of the nystagmoid movements in each direction.

Conjugate ocular palsies.—In addition to the defects of movement due to paralysis of the individual ocular muscles, weakness or paralysis of the

movement of both eyes in one direction frequently occurs. Thus the patient may be unable to look to either side, or upwards or downwards; or the power of convergence alone may be lost. Palsy of lateral conjugate movement indicates most probably a lesion of the sixth nucleus of the side to which the movement is weak. The conjugate vertical palsies are always associated with disease of the corpora quadrigemina, or in the neighbourhood of the oculo-motor nuclei.

If both eyes are kept persistently turned in one direction, the condition is spoken of as **conjugate deviation** of the eyes. It is usually either to the right or to the left. Conjugate deviation of the eyes may be brought about either by a lesion which produces paralysis or by one which causes irritation or spasm. In the former case the eyes (and usually, also, the head) are turned towards the side of the lesion, provided the latter be in the cerebral hemisphere. The patient, in fact, is said "to look towards his lesion." An irritative lesion in a similar situation causes the deviation to be towards the healthy side. If, however, the lesion have its seat in the pons, these rules are just reversed, the deviation being towards the sound side in a paralytic lesion, and towards the affected side in one which is irritative.

Skew deviation of the eyes—in which, for example, one is directed upwards and the other downwards—occurs in certain lesions of the cerebellum.

EXAMINATION OF THE PUPILS

This important part of the investigation of a nervous case may be conveniently considered at this stage. The following points must be noted about the pupils in every case :—

1. **Size.**—Compare the size of the two pupils, first in a bright light and then in a dim light. Note

whether the pupils are large or small, and whether any irregularity is present. It must be remembered that the size of the pupil in health is subject to great variations. As a rule, the pupils are larger in dark eyes than in light. A much dilated pupil is often a sign of nervous exhaustion or instability. Slight inequality of the pupils may also be present in perfectly healthy subjects. We are inclined to think that in such cases the left pupil is usually the larger.

If one pupil is larger than the other, the question arises, Which is the normal? This question is not always very easily answered, but, as a rule, the pupil which exhibits the less mobility is to be regarded as the abnormal one.

2. **Shape.**—Note whether the pupil is circular in outline, as it should be, or whether its contour is irregular. Such irregularities may be due to adhesion of the iris to the lens or to the effects of an old iritis (*see* p. 526). Irregularity in shape of the pupil is often an early symptom in general paralysis of the insane.

3. **Mobility.** (*a*) **Reaction to light.**—This is a reflex action. The afferent fibres involved are contained in the optic nerve, the intermediate station is in the corpora quadrigemina, and the efferent fibres pass by the 3rd nerve, through the ciliary ganglion, to the pupil-sphincter.

Test.—Examine each eye separately. Place the patient opposite a bright light, be sure his accommodation is relaxed, and cover the eye with the hand. Leave it covered for about half a minute, then withdraw the hand and watch the pupil. It should contract almost immediately, then dilate again a little, and, after undergoing slight oscillations, settle down to its normal size.

The test may also be carried out by concentrating

light upon the pupil by means of a mirror or lens, just as one does in testing the light perception.*

Owing to the decussation of some of the fibres of the optic nerves at the chiasma, light acting upon one eye affects the centre for pupil contraction of the other eye as well as that on its own side. It is probable that fibres pass directly between the centres for the two 3rd nerves which aid in bringing about this result. As a consequence, one finds that if light be shut off from one eye both pupils dilate, and if bright light be made to enter one eye both pupils contract. This is known as the *consensual reaction* of the pupils. It should be tested by keeping one eye in the shade while light is thrown into the other. The effect on the pupil of the shaded eye is then observed.

Lesions of the optic nerve of the mid-brain, or of the oculo-motor nerve or its nucleus, interfere with this reflex contraction of the pupil to light.

Wernicke's hemiopic pupil reaction may be mentioned here. Hemianopia, as we have seen, may be due to a lesion of the optic tract between the chiasma and the corpora quadrigemina, or it may be produced by destruction of the optic fibres between the corpora quadrigemina and the occipital cortex, or it may be due to lesion in the cortical visual centres themselves.

If the lesion be in front of the corpora quadrigemina—i.e. in front of the pupil centre—the reflex contraction of the pupil to light coming from the blind portion of the visual field is lost, whereas, if it be at any point behind that, the contraction of the pupil to light is retained. It is upon this fact that Wernicke's reaction is based. In carrying out the test the light must, of course, be concentrated on the blind halves of the retinæ. Proceed as follows: Place the patient in a dark room with a light beside his head. Hold a large plane mirror in the left hand, and by means of it illuminate both pupils and observe their size. Then take an ordinary ophthalmoscopic mirror in the right hand and direct a strong beam of light on to the blind side

* A convenient method is to throw the light on the pupil by the mirror of an ophthalmoscope with a + 8 lens behind. Looking through the lens one gets a magnified view of the pupil, and small changes in it can be more easily observed.

of the retinæ. If the lesion be in front of the corpora quadrigemina no contraction of the pupils should result; if behind that, they become smaller. Lately, considerable doubt has been thrown on the occurrence of Wernicke's reaction under any circumstances.

(*b*) **Reaction to accommodation.** — As is well known, the pupils become smaller on accommodating for a near object. It is really more correct to speak of reaction on *convergence*, as it is found that it is the convergence of the eyes, not the mere effort of accommodation, which causes the pupil to become smaller.

Test.—Hold up one finger close to the patient's nose. Ask him to look away at a distant object. Then suddenly tell him to look at your finger. As the eyes converge to accomplish this the pupils should become decidedly smaller.

If the patient be unable to see, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to direct his eyes to it.

Argyll-Robertson pupil.—This is the term applied to the condition of pupil usually observed in locomotor ataxy, but also found sometimes in general paralysis and other degenerative diseases of the nervous system, especially syphilitic. It reacts to accommodation, but not to light. Sometimes the reaction to light is not entirely absent, but takes place in a very sluggish fashion.

(*c*) **Cilio-spinal reflex.**—Dilatation of the pupil can often be observed to follow irritation of the skin of the neck either by pinching or by the action of a faradic current. It is due to stimulation of the pupil-dilating fibres in the cervical sympathetic (p. 477), and is abolished in lesions of that nerve.

Abnormal movements of the pupil.—The term *hippus* is applied to the alternate contraction and dila-

tation of the pupil, which can sometimes be observed going on rhythmically (*see* p. 526).

Fifth Nerve

Anatomy.—1. The **sensory root** takes origin from the cells of the Gasserian ganglion and enters the lateral surface of the pons at about its middle. Its fibres terminate in a large nucleus in the pons, situated in the floor of the 4th ventricle and lying externally to the motor nucleus, and partly also from the "descending" or bulbo-spinal root, which begins as low down as the 2nd cervical segment of the cord. Immediately beyond the Gasserian ganglion the nerve separates into its three divisions.

The **first or ophthalmic division** supplies the eyeball, conjunctiva (except that of the lower lid), and lachrymal gland, the mesial part of the skin of the nose as far as the tip, the upper eyelids, the forehead, and the scalp as far as the vertex.

Paralysis of this division results in loss of sensibility in the area of skin and mucous membrane supplied, and in trophic changes in the eyeball. The conjunctival reflex is abolished.

The **second or superior maxillary division** supplies the cheek, the front of the temple, the lower eyelid and its conjunctiva, the side of the nose, the upper lip, the upper teeth, the lining membrane of the nose, the upper part of the pharynx, the roof of the mouth, the soft palate, and the tonsils.

Paralysis of this division leads to abolition of sensibility in the above area, and loss of the palate reflex.

The **third or inferior maxillary division** is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the side of the head, the ear, the tongue, and the lower teeth. It supplies also the salivary glands and, through the motor division, the muscles of mastication, the tensor tympani, and also, perhaps, the tensor palati, although many believe that this muscle is innervated by the spinal accessory.

2. **Motor root.**—This takes origin in a small nucleus lying internally to the chief sensory nucleus, and partly also from the mesencephalic root, which arises in nerve cells scattered around the aqueduct of Sylvius. It emerges at the

side of the pons, just in front of the sensory division, passes underneath the Gasserian ganglion, and joins the inferior maxillary division, to which it gives its motor fibres.

Paralysis of the whole 5th nerve leads to loss of sensation in the areas of skin and mucous membrane above mentioned, and to defective power of chewing. (Fig. 124.) Trophic lesions may be present, and the salivary, buccal, and lachrymal secretions much diminished; and the sense of taste is occasionally abolished on the anterior two-thirds of the same side of the tongue.

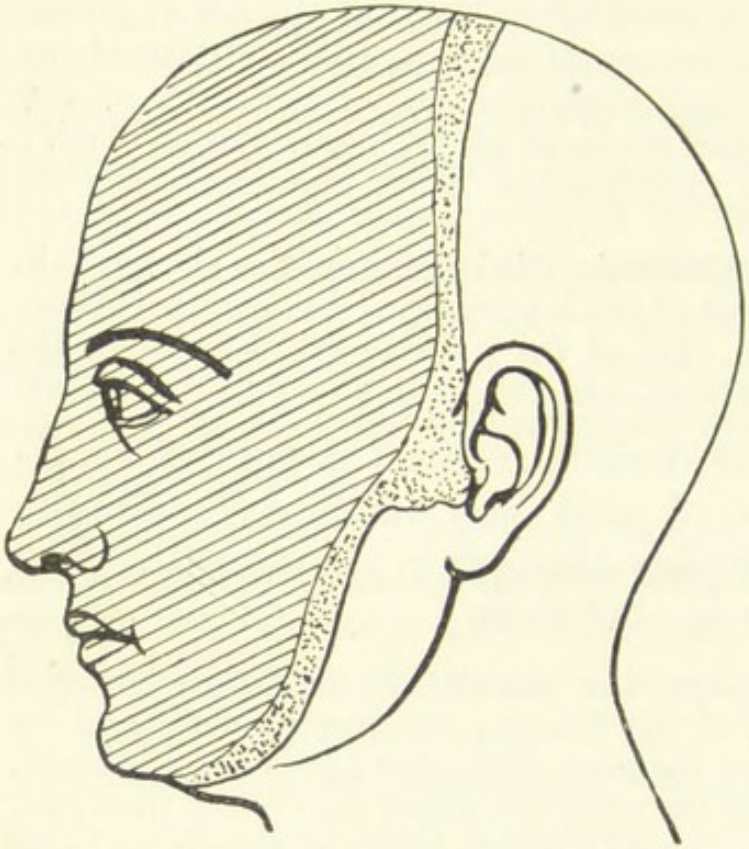


Fig. 124.—Distribution of sensory loss in complete paralysis of the 5th nerve. The shaded area represents complete anæsthesia, the dotted area partial, and chiefly loss to light touch.

One curious result of the sensory paralysis is that the patient, when drinking, imagines that the cup is broken, as he only feels it on one side of his mouth.

How to test the fifth nerve. 1. **Motor functions.**—Ask the patient to clench his teeth while the observer keeps his hands on the temporal and then on the masseter muscles. These should stand out with equal prominence on each side. If there be paralysis on one side, the muscles on that side will fail to become prominent. On opening the mouth the jaw

deviates towards the paralysed side, being pushed over by the healthy external pterygoid muscles. The condition of the tensor tympani muscle cannot be satisfactorily examined except by noting whether there is any difficulty in hearing notes of a particular pitch—i.e. a diminution in the "field of hearing."

2. **Sensory functions.**—The common sensibility of the area supplied is tested in the usual way (p. 491).

Taste.—In suspected lesions of the 5th nerve the sense of taste should be always examined, as it seems probable that in certain cases at least taste fibres from the anterior two-thirds of the tongue reach the brain through the 5th nerve. As a rule, however, they pass from the lingual nerve into the chorda tympani, and thence through the geniculate ganglion and the nervus intermedius of Wrisberg into the medulla oblongata. The taste fibres from the posterior third of the tongue enter by the glosso-pharyngeal nerve, and probably terminate in the same centre in the medulla as those that enter by the nervus intermedius.

The higher connections of this primary gustatory centre are not accurately known in man, but it is probable that a cortical centre exists in the tip of the temporo-sphenoidal lobe.

How to test the sense of taste.—Have some sugar, some quinine, and some salt, all in powder. Ask the patient to put out his tongue and to keep it out until the conclusion of the test. Many men, especially smokers, are unable to taste on the protruded tongue; the tongue should be then drawn in, but the mouth kept open in order to avoid spread of the test substances. Place some sugar on the tongue, rub it gently in, and ask him, "Is that salt?" If taste is normal he will shake his head. In this way all the substances are tried, first on the anterior part of the tongue and then at the back. A weak galvanic current is also a useful test. It should produce a sort of metallic taste.

Loss of taste may, of course, arise from lesions of the taste fibres in any part of their course above stated.

In addition to loss of taste, one should always ask the patient whether he has any abnormal taste sensations.

Seventh Nerve

Anatomy.—The course of the fibres from the cortical centre to the nucleus of this nerve has already been described (p. 411). The nucleus is situated in the pons external to that of the 6th nerve. On leaving the nucleus the fibres wind round the nucleus of the 6th, and finally emerge mesially to the 8th nerve, between the olive and restiform bodies.

The nerve lies in close contact with the 8th, so that a lesion of the one at this part can hardly avoid injuring the other, and enters the internal auditory meatus along with it. During its course in the aqueduct of Fallopius it gives off a branch to the stapedius muscle, and is joined by the chorda tympani, which contains taste fibres from the anterior two-thirds of the tongue. In this part of its course the nerve is exposed to the effects of pressure, owing to its being enclosed in a bony tube. It emerges at a point opposite the junction of the anterior border of the mastoid with the ear, and spreads out on the side of the face to supply its muscles. In this part of its course it seems to be peculiarly liable to the effects of exposure to cold.

Functions.—The 7th is a purely motor nerve. It supplies all the muscles of the face and scalp, except the levator palpebræ superioris. It also supplies the platysma.

Effects of paralysis.—These are usually at once seen on looking at the patient. The affected side of the face has lost its expression. The naso-labial fold is less pronounced than on the other side. The furrows of the brow are smoothed out, the eye is more widely open than the other, and the mouth is somewhat drawn to the healthy side. The patient is unable to whistle, food is apt to collect between his teeth and his gums, and saliva and any fluid he drinks may escape from the affected angle of the mouth.

How to test the seventh nerve.—1. Ask the patient to shut his eyes as tightly as ever he can.

Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend—or, if the eye is closed, the eyelashes are not so far rolled in as on the healthy side. Try also forcibly to open the eyes while the patient attempts to keep them closed. If the orbicularis is acting normally, it should be almost impossible to open the eye against the patient's wish. If the muscle be partially paralysed, however, the exertion of very little force may suffice to open it.

The effort made in screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards. In paralysis of the lower part of the face the corner on the affected side is either not drawn up at all, or at all events not so much as on the healthy side.

2. Ask the patient to whistle. He is unable to do so.

3. Ask him to smile or show his upper teeth. The mouth is then drawn to the healthy side.

Signs of paralysis of the facial nerve in different parts of its course.—Paralysis of the face presents different symptoms according as the lesion is situated above the nucleus, or either at the nucleus or below it. The former constitutes what is known as cerebral or supranuclear facial paralysis, the latter produces peripheral or infranuclear paralysis.

The chief difference between the two forms is that in *supranuclear paralysis* the lower part of the face is chiefly affected; in *infranuclear paralysis* both the upper and lower parts are equally involved. The probable explanation of this is that the two orbicularis palpebrarum muscles are so often required to act together that each is supplied from both sides of the brain, and consequently a unilateral lesion only partially cuts off the nerve impulses to one side. We have already described what is meant by "crossed paralysis," and the part which the facial plays in it.

Infranuclear facial paralysis may be produced by a lesion of the nucleus itself, of the nerve trunk inside the aqueduct, or of the nerve trunk either after its emergence from the aqueduct or before it has entered it.

A lesion inside the aqueduct—unless it be towards the outer end of the latter—involves the fibres of the chorda tympani, and therefore produces a paralysis of taste sensation in the anterior two-thirds of the tongue. A lesion in any of the other situations produces a typical complete facial paralysis (Bell's paralysis).

A lesion of the nerve before it has entered the aqueduct can be distinguished from a lesion below the stylo-mastoid foramen by the fact that in the former condition the stapedius muscle is paralysed (causing excessive sensitiveness to loud sounds, or "hyperacusis"), while in the latter it escapes. Never omit, therefore, in a case of facial paralysis to inquire regarding the patient's sensitiveness to loud sounds.

Lesions of the nucleus or the nerve below it will result in atrophy of the facial muscles and the appearance in them of the reaction of degeneration (p. 520). Supranuclear lesions do not produce this effect.

Abnormal facial movements.—The muscles supplied by the facial nerve are frequently affected by spasm or spasmodic movements. These may involve all the facial muscles, or groups of them only. The spasm may be of either the clonic or tonic variety (p. 486). The nature of the movements, if present, their extent and the muscles affected by them, should always be carefully noted.

Eighth Nerve (Auditory)

Anatomy.—This nerve consists of two sets of fibres. One set supplies the cochlea, and subserves the function of hearing; the other part supplies the vestibule and semicircular canal, and is the nerve of equilibration. The auditory fibres, which

arise from the cochlear ganglion, enter the brain laterally to the corpus restiforme and form the dorsal root of the 8th nerve. They terminate in the ventral cochlear nucleus and in the tuberculum acusticum. The vestibular fibres take origin from the vestibular ganglion, and terminate in a nucleus placed laterally in the floor of the 4th ventricle. They enter the medulla mesially to the corpus restiforme, and therefore form the ventral root of the 8th nerve.

The secondary auditory tracts, after partial decussation, terminate in the posterior corpora quadrigemina and the median geniculate bodies, and another system that takes origin from these passes through the internal capsule to the cortical centre for hearing, in the 1st and 2nd temporo-sphenoidal convolutions. Sounds received in one ear probably reach the opposite hemisphere of the brain predominantly, but owing to the partial decussation of the secondary auditory tracts a unilateral cerebral lesion cannot produce deafness in one ear.

The vestibular nerve is closely connected with the cerebellum. Nothing is known of its cerebral connections.

Test. 1. **Hearing.**—Before testing a patient's power of hearing it is well to exclude the presence of wax in the ear (*see* p. 541). This being disposed of, one can test the hearing power by means of a watch. Stand behind the patient and ask him to shut his eyes. Begin outside the probable range of hearing power, and bring the watch gradually nearer the ear, asking the patient to speak whenever he hears the tick. One requires, of course, to know at what distance the tick should be audible to a healthy ear. It is necessary to test each ear separately, one being closed whilst the other is being examined.

If impairment of hearing be detected, one must next try to ascertain whether it is really due to disease of the auditory nerve or merely to some affection of the middle ear. In order to settle this point, the **tuning-fork test** may be employed. When the fork is beating strongly, hold it opposite the ear; if it can be heard, then place its base on the mastoid process in order to determine if its vibrations can be heard when

conducted through bone. If the patient succeeds in this, ask him to compare the relative loudness of the fork when heard through air and through bone, or to determine which can be heard the longer as the vibrations die out. This is **Rinne's test.** Normally, aerially conducted sounds are louder to the patient than when conducted through bone. In middle-ear disease, aerial conduction is diminished or lost, while bone conduction remains more or less normal. When the auditory nerve is affected, both air and bone conduction are diminished or lost.

Weber's test, though less reliable than Rinne's, should be also used. Strike a tuning-fork and place the end of it against the centre of the patient's forehead. If the deafness discovered by the watch be due to an affection of the middle ear, the patient will hear the tuning-fork *louder* on that side than on the healthy one. On the other hand, if the deafness be due to disease of the auditory nerve, the tuning-fork will only be heard on the healthy side. The test may also be carried out by means of the watch. In affections of the nerve, the watch is not heard even when pressed against the ear; in disease of the middle ear, it is heard even more loudly than when similarly applied to the healthy side. The explanation of these facts is not yet clearly made out, nor are they invariably trustworthy. They hold good, however, for a majority of cases. Other points in favour of the deafness being due to the nerve and not to the middle ear are, (*a*) if the hearing is better in a quiet place, (*b*) if conversation is heard better than the watch, (*c*) if inflation of the middle ear renders the hearing worse.

Abnormal auditory sensations.—The patient may complain of "ringing in the ears," or **tinnitus.** The precise character of the sound varies in different cases. It may be of a humming, buzzing, hammering, or

whistling character. The presence or absence of this symptom should always be inquired for, and whether it is constantly present or in what circumstances it comes on.

Hyperæsthesia of the auditory nerve (**hyperacusis**), by which even slight sounds are heard with painful intensity, sometimes occurs, especially in hysteria and in lesions of the facial nerve above or in the aqueduct, owing to paralysis of the stapedius muscle.

Hallucinations of sound may also be present, the patient fancying that he hears voices, bells, etc. These occur chiefly in states of mental disturbance, but they have been occasionally observed with lesions of the cerebral auditory centre.

2. **Vertigo.**—The patient will describe this as giddiness or dizziness. In order to constitute true vertigo, external objects should seem to move round him. Ask if this is so, and, if it is, in what direction the objects seem to move. Ask also whether the vertigo ever causes him to fall to the ground.

Vertigo may be due to paralysis of one of the ocular muscles, to an affection of the ear or vestibular nerve, to loss of muscular or common sensibility, especially in the lower extremities, or to states of intoxication. When a patient complains of vertigo as his chief symptom, one should therefore examine carefully for squint, for disease of the outer and middle ear, and for signs of disease of the 8th nerve. One should also investigate the condition of muscular and common sensibility, and inquire for symptoms pointing to gastric disturbance.

Ninth (Glosso-Pharyngeal), Tenth (Vagus), and Eleventh (Spinal Accessory) Nerves

Anatomy.—These arise in order from above downwards from an elongated nucleus in the floor of the 4th ventricle.

They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies. The spinal part of the 11th emerges from the lateral column of the cord, beginning as low as the 6th cervical nerve; it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen. After its emergence the two divisions of it again part company, the medullary or accessory portion joining the vagus.

The **ninth** (glosso-pharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus. In contains the taste fibres for the posterior part of the tongue (*see* p. 467).

How to test the glosso-pharyngeal.—The 9th nerve is rarely paralysed alone. Paralysis of it can only be diagnosed by examining its sensory functions. Examine the power of taste in the posterior part of the tongue (p. 467). Loss of it *may* mean paralysis of the trunk of the glosso-pharyngeal nerve.

Tickle the back of the pharynx, and note if the reflex is present.

The **tenth** (vagus) is motor for the soft palate (with the exception of the tensor palati), pharynx, and larynx. It is also sensory and motor for the respiratory passages, the heart, and (through the sympathetic ganglia) for most of the abdominal viscera.

The fibres for the soft palate and larynx take origin in the nucleus ambiguus, emerge in the upper roots of the 11th, reach the pharyngeal plexus, and thence pass to the muscles of the palate, the constrictors of the pharynx, and to the larynx.

The viscero-motor and the cardio-inhibitory fibres are derived from the dorsal vagus nucleus in the floor of the 4th ventricle.

How to test the vagus.—Paralysis of the vagus

is chiefly evidenced in its palatine and laryngeal branches.

1. **The palate.**—Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow. This is a common occurrence in total paralysis of the soft palate, owing to defective elevation of it during swallowing. For a similar reason the patient is unable to pronounce words which require complete closure of the naso-pharynx. Thus "egg" is sounded as "eng," "rub" becomes "rum," and so on. In unilateral paralysis these symptoms are not observed.

For direct examination of the soft palate, place the patient facing the light with his mouth open, and introduce a tongue depressor. The position of the uvula is quite unreliable as a guide to the state of the soft palate, as deviation of it is not uncommon even in health. One must watch the movements of the palate during phonation. Ask the patient, therefore, to say *Ah*, and observe whether both sides of the palate arch upwards; in health a sort of depression appears in the centre of the palate when the patient says *Ah*. If one side be paralysed, that side will remain flat and immobile, and the median raphé will be pulled towards the other side. The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken. In bilateral paralysis the whole palate remains motionless.

2. **The larynx.**—The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true cords, and is motor for the crico-thyroid muscle. Unilateral paralysis of the nerve does not produce any symptoms. Bilateral paralysis causes the vocal cords to be relaxed. The voice is therefore hoarse and deep, and the utterance of high notes impossible.

The recurrent laryngeal branch supplies sensation to the larynx below the level of the cords, and motor fibres to all the laryngeal muscles except the crico-thyroid. Paralysis of it leads to appearances which are recognized by the laryngoscope, and are described at p. 548.

The eleventh nerve.—*Anatomy.*—The accessory part of this nerve gives to the vagus motor fibres for the larynx and pharynx. The spinal part of the nerve dips beneath the sterno-mastoid muscle about one inch below the tip of the mastoid process, and emerges from underneath that muscle again at about the middle of its posterior border. It supplies the sterno-mastoid and trapezius, which are also supplied by twigs from the cervical plexus. Lesions of the 11th nerve, therefore, lead to paralysis of these muscles.

How to test the spinal accessory.—Paralysis of the upper part of the trapezius is evinced by asking the patient to shrug his shoulders while the observer offers passive resistance by pressing on the shoulders from behind. Paralysis of the sterno-mastoid causes difficulty in rotation of the chin towards the opposite side.

Twelfth or Hypoglossal Nerve

Anatomy.—The 12th nerve arises from a nucleus in the lower part of the floor of the 4th ventricle, close to the middle line. It emerges between the anterior pyramid and the olive. It is a purely motor nerve, supplying the tongue and the depressors of the hyoid bone. Its cortical centre is in the lower part of the ascending frontal convolution.

Test.—Ask the patient to put out his tongue as far as possible. If the hypoglossal be paralysed, the tongue, instead of being protruded straight, is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue, really due to the mouth being twisted to one side, for a real deviation of it. Such an apparent deviation occurs in facial paralysis. Ask him also to move his tongue from side to side, and to lick each cheek with it; observe

whether he can do so freely. Note whether there is any wasting of the tongue, and whether there is any tremor or fibrillary twitching in it. The presence of wasting indicates that the lesion is either nuclear or infranuclear.

Paralysis of the cervical sympathetic may be conveniently considered here. A complete description of the functions and distribution of the nerve, however, is not necessary in such a work as this. For purposes of diagnosis the fibres supplied to the eyeball are alone of importance. These take origin in the lower cervical and upper dorsal regions of the spinal cord (cilio-spinal centre), from which the fibres emerge in the last cervical and first dorsal nerve roots and pass to the sympathetic cord by the rami communicantes. From the cervical sympathetic cord the fibres pass along the internal carotid to the cavernous plexus, and thence via the ophthalmic division of the 5th to the eyeball. They convey the impulses which cause dilatation of the pupil, and supply also the unstriped muscle in the insertion of the levator palpebræ into the upper lid. Paralysis of the cervical sympathetic is recognized by the following signs: Some recession of the eyeball, so that the eye looks smaller than its fellow; slight drooping of the upper lid, due to paralysis of the unstriped muscle fibres contained in it; contraction of the pupil with absence of dilatation on shading the eye or on instillation of cocaine; abolition of the cilio-spinal reflex; less commonly, absence of sweating, even after the use of pilocarpin, on the corresponding half of the head and neck both in front and behind extending as low as the 3rd rib and 3rd dorsal spine, and over the whole of the upper limb on the same side.*

* Sweating of the face can best be induced by making the patient smell mustard.

IV. MOTOR FUNCTIONS

In investigating the motor functions of a patient, one has to satisfy oneself on four separate points :—

1. Is there any muscular paralysis or weakness ?
2. Can the patient co-ordinate his actions normally ?
3. What is the state of nutrition of his muscles ?
4. Is there any abnormal muscular movement ?

1. Investigation of Motor Power

The first thing to be noted as regards the patient's voluntary power is whether or not he is capable of performing gross muscular movements. Can he walk ? Can he sit up in bed ? Can he move each of his limbs as a whole ?

These main points having been determined, it may be necessary to investigate the range of the movements that the patient can make, and the strength of the principal muscles and groups of muscles separately.

The general rule for one's guidance in this investigation is to ask the patient to throw into action the particular muscle or group of muscles which one wishes to test, whilst the observer offers to that action a greater or less degree of passive resistance. The following is the method of procedure :—

i. **Upper limb.** *Flexors of fingers.*—Ask the patient to squeeze your hand. If a record of the power of grasp be desired, that can be compared with the result yielded in similar circumstances on another occasion, one should make use of the dynamometer.

Interossei and lumbricales.—Paralysis of these muscles gives rise in cases of some standing to a peculiar position of the hand known as **"main en griffe"**

or claw-hand. The above-mentioned muscles produce flexion of the first phalanges on the metacarpals and extension of the other two phalanges. Paralysis of them produces, by over-action of the long flexors and extensors of the fingers, over-extension of the first phalanges and flexion of the other two. The fingers are also slightly separated from one another. Claw-hand occurs in some cases of progressive muscular atrophy; and, in a partial form, in paralysis of the ulnar nerve. Claw-foot is an analogous condition.

Opponens pollicis.—Ask the patient to touch the tip of his little finger with the point of his thumb.

Adductor of thumb.—Ask the patient to grasp a book between the forefinger and thumb, keeping the thumb and fingers in the same plane.

Flexors of wrist.—The hand being held with the palm upwards, ask him to bring the points of his fingers towards the front of the forearm.

Extensors of wrist.—The hand being held with the palm downwards, the observer grasps the patient's wrist and asks him to bend the hand up backwards as far as possible. The fingers should be at the same time held flexed, as the wrist can be extended by contraction of the long extensors of the fingers. If he be unable to produce dorsiflexion of the wrist, some weakness or paralysis of the extensors is present.

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand. If the extensors be weak the wrist becomes flexed as he does so, owing to the flexor muscles getting the better of the extensors.

Weakness or paralysis of the extensors of the wrist leads to the condition known as **wrist-drop.**

Supinator longus.—Place the arm midway between the prone and supine positions; then ask the patient to bend up the forearm whilst the observer offers

opposition to the act by grasping the hand. If the muscle be healthy, it will be seen and felt to stand out prominently at its upper part.

Biceps.—The patient's elbow being held against his side, ask him to bend up the forearm while opposition is offered by grasping the hand or wrist. If the biceps be healthy, it will be observed to stand out prominently as it contracts.

The *triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed by means of passive resistance.

Deltoid.—Ask the patient to lift his arms straight out at right angles to the trunk. In paralysis of the deltoid he is unable to do so.

Pectorals.—Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart. Note whether both heads of the muscle are thrown into contraction or not.

Serratus magnus.—Ask the patient to push against resistance. In a healthy condition of the muscle its various digitations will be seen to stand out in contraction, whilst the scapula will remain in close apposition to the chest wall. If the muscle be paralysed, the posterior border and inferior angle of the scapula will come to project more or less when the patient pushes.

Latissimus dorsi.—Ask the patient to clasp his hands behind his back while the observer, standing behind the patient, offers passive resistance to the downward and backward movement; or grasp the two posterior axillary folds and ask the patient to cough. In health the latissimus can be felt to contract.

ii. **Trunk muscles.**—Weakness of the muscles of the abdomen is indicated by the patient being unable to raise himself in bed without the aid of his

arms.* To test the *erector spinæ* and muscles of the back, make the patient lie on his face and try to raise his head from the bed by extending the neck and back. If the back muscles are healthy, they will be seen to stand out prominently during this effort.

The method of detecting paralysis of the *diaphragm* has already been described (pp. 259, 263).

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind. In its lower part it can be tested by asking him to approximate the shoulder-blades.

iii. **The head muscles.**—For the methods of detecting weakness or paralysis in the muscles of the head, the reader is referred to the section dealing with the investigation of the Cranial Nerves (p. 439).

iv. **The lower limb.**—The muscles of the foot are tested on the same lines as the corresponding muscles of the hand—passive resistance being offered to their action in each case.

Extensors of knee.—Bend up the patient's knee, and then, pressing with your hand on the sole of his foot, ask him to try to straighten it out again.

Flexors of knee.—Turn the patient on his face, and then ask him to bend up the knee whilst the observer endeavours to hold it down by pressing upon the back of the ankle.

Extensors of thigh.—The knee being extended, lift the patient's foot off the bed, and ask him to depress it against resistance. If the extensors of the hip are paralysed he will be unable to do so.

* Babinski's "rising-up sign" consists in making the patient lie on his back with the legs extended and rise up without using his hands. In *organic spastic* paralysis of a leg the affected limb will rise first, owing to the rigidity; but in *functional* paralysis this does not occur.

Flexors of thigh.—The knee being extended, ask the patient to raise his leg off the bed.

The *adductors of the thigh* are tested by abducting the limb and then asking the patient to bring it back to the middle line while passive opposition is offered to the act. In a similar way the *abductors* are tested by bringing the limb across the middle line and then asking the patient to move it outwards again.

Rotators of the thigh.—Turn the patient on his face, and bend the knee to a right angle. Then ask him to roll the leg outwards or inwards, whilst passive resistance is offered by grasping the foot.

If, on carrying out any of these tests, a muscle or group of muscles be found to have only a feeble power of contraction, **paresis** of it is said to be present. If no contraction be elicited at all, the condition is one of **paralysis.**

The term **hemiplegia** is applied to a condition in which there is paralysis of one side of the face, and of the arm and leg on the same side. If the paralysis of the arm and leg be on one side, and that of the face on the other, the condition is one of **crossed paralysis.** The term **paraplegia** is applied to a paralysis of the lower part of the body; the term **monoplegia** to a paralysis of one arm (which is therefore characterized as a *brachial* monoplegia), one leg (*crural* monoplegia), or one side of the face (*facial* monoplegia).

The detection of paralysis, and still more of paresis, in a patient who is comatose is often a very difficult matter. It is to be observed, however, that if the paralysis is of recent onset, in such a patient one can usually detect a greater degree of *limpness* in the paralysed limb. If the arm, for example, be raised from the patient's side and allowed to drop, it falls, if it be paralysed, just as if it did not belong to him; the

sound arm also falls, but not in such an utterly limp fashion. The distinction, however, is often by no means easy.

2. Investigation of Muscular Co-ordination

By muscular co-ordination is meant the co-operation of separate muscles, or groups of muscles, in order to accomplish a definite act. If such co-operation be absent or imperfect, the performance of certain acts becomes difficult or impossible, and the condition is then said to be one of **inco-ordination.** The term **ataxia** or **ataxy** has a similar meaning.

The co-ordination or harmonious action of groups of muscles is the product of various factors, among the chief of which are the afferent impulses coming from the muscles that never reach consciousness and those on which the sense of position of the limbs depends; the state of tone of the muscles, and in some acts, perhaps, cutaneous sensibility. When inco-ordination is present it is not always easy to say which of these factors is at fault. The movements that constitute an act can be controlled and directed by vision, but sight itself is not concerned in the co-ordination of movements. When, however, there is loss of the sense of position, the sensory defect may be compensated by vision, and the disturbance of movement may become apparent only when the eyes are closed or bandaged, or in the dark. This disturbance, which occurs in tabes dorsalis and other conditions, is sometimes spoken of as inco-ordination, but it is not properly included in this term.

How to test co-ordination. 1. *In the upper limbs.*—Ask the patient to touch the point of his nose first with one forefinger and then with the other; or ask him to bring the points of the two forefingers together. If he is able to succeed in these tests

naturally and without making random shots, no inco-ordination is present. He may then be asked to perform the same actions with his eyes closed; any additional irregularity of the movements can be due only to disturbance of the sense of position.

Another good test of co-ordination in the upper limb is to ask the patient to thread a needle. In this case, of course, the eyes must be left uncovered.

2. *In the lower limbs.*—If the patient is able to walk, a good test for co-ordination in the lower limbs consists in asking him to walk along a straight line—e.g. a crack between two boards of the floor, or the edge of a carpet. If inco-ordination be present he will soon deviate to one side or the other.

If he cannot walk, proceed as follows: The eyes being covered, ask the patient, as he lies in bed, to touch the dorsum of one foot with the great toe of the other.

Another method is to leave the eyes open, and then ask him to follow with his toe one's forefinger, with which one describes circles in the air. If he is able to describe the circles accurately his power of co-ordination is good.

Romberg's sign is often regarded as a special test for the co-ordination of the lower limbs, but its presence is only evidence that the sense of position in these limbs is defective. The patient is made to stand with his feet close together, and if he can do so he then closes his eyes. If the sign be present he begins at once to sway about or may even fall. To elicit slight degrees of the phenomenon, it may be necessary to make the patient stand on tiptoe with his knees bent. The essential feature of the sign is that the patient is more unsteady standing with his eyes closed than when they are open. This is due to the fact that, owing to deficient sensory impressions from the lower

extremities, the patient is unable to maintain his attitude without the aid of vision.

Babinski has described a special sign for cerebellar ataxia under the name **adiadokokinesia;** it consists in inability to execute rapidly repeated movements. In order to test for it the patient is asked to flex his elbows to a right angle and then supinate and pronate his forearms as rapidly as possible. All normal persons can do this at approximately the same rate, but, as a rule, slightly less rapidly with the left than with the right arm. When, however, adiadokokinesia is present the movements are slow, awkward, and incomplete, and often become impossible after a few attempts.

3. State of Nutrition of the Muscles

This is gauged roughly by palpating the muscles and noting whether they are firm, as in health, or wasted and flabby. In spastic paralysis the muscles are usually more firm than normal. In the disease known as pseudo-hypertrophic paralysis some of the muscles are abnormally firm and large. This is especially apt to be the case with the calf muscles and the infraspinati. Such increase in size and firmness must not be mistaken for a mere increased muscular development; it is due to an overgrowth of the interstitial tissue of the muscle at the expense of the muscle fibres, and is really a sign of disease. The finer indications regarding muscular nutrition which are afforded by the use of electrical stimulation are described in the section on the Electrical Examination of Muscles and Nerves (p. 509).

4. Abnormal Muscular Movements

These consist of involuntary muscular contractions of various sorts. The first thing to note is whether the movements are widespread or localized.

If they be confined to one part of the body, note the joints at which the movements occur, and the muscles or groups of muscles involved. The term **spasm** is often applied to any exaggerated and involuntary muscular contraction. The contraction may either be continuous, in which case it is said to be **tonic**; or there may be a series of short contractions with complete or partial relaxation of the muscle in the intervals, and in that case they are spoken of as **clonic.**

A general increase of "tone" in the muscles is spoken of as **hypertonia**; a general diminution as **hypotonia**. **Kernig's sign** is a phenomenon depending upon the existence of an increased tonus. In order to elicit it as originally described, place the patient on his back with the legs relaxed and extended at the knees. On raising him to a sitting posture the knees become flexed, and cannot be straightened when he is again laid upon his back, owing to a strong contraction of the hamstrings. The sign is more commonly elicited now by flexing the thigh to almost a right angle and then trying to extend the knee (Fig. 125), whilst the other leg is kept flat upon the bed. If a positive result is obtained the hamstrings are thrown into contraction. Another plan is first to extend the knee fully, then flex the thigh on the pelvis, and measure the angle at the hip. (Rudolf.)

Kernig's sign is present in about 85 per cent. of all cases of meningitis, but has also been found occasionally in cerebellar hæmorrhage and other conditions at the base of the brain; also in diseases of the upper motor neurones, after disuse of the lower limbs for some days, as in recumbency; and in local conditions such as sciatica. It commonly coexists with cervical opisthotonos.

The term **contracture** is usually applied to that

permanent shortening of groups of muscles, often observed in cases of paralysis due to cerebral lesions, which cannot be overcome by passive movement. It is due to secondary fibrous changes in muscles which are weak or paralysed.

Tetanic spasm is observed in its completest form in tetanus, strychnine poisoning, hydrophobia, and some kinds of hysterical fits. It may lead to a bend-

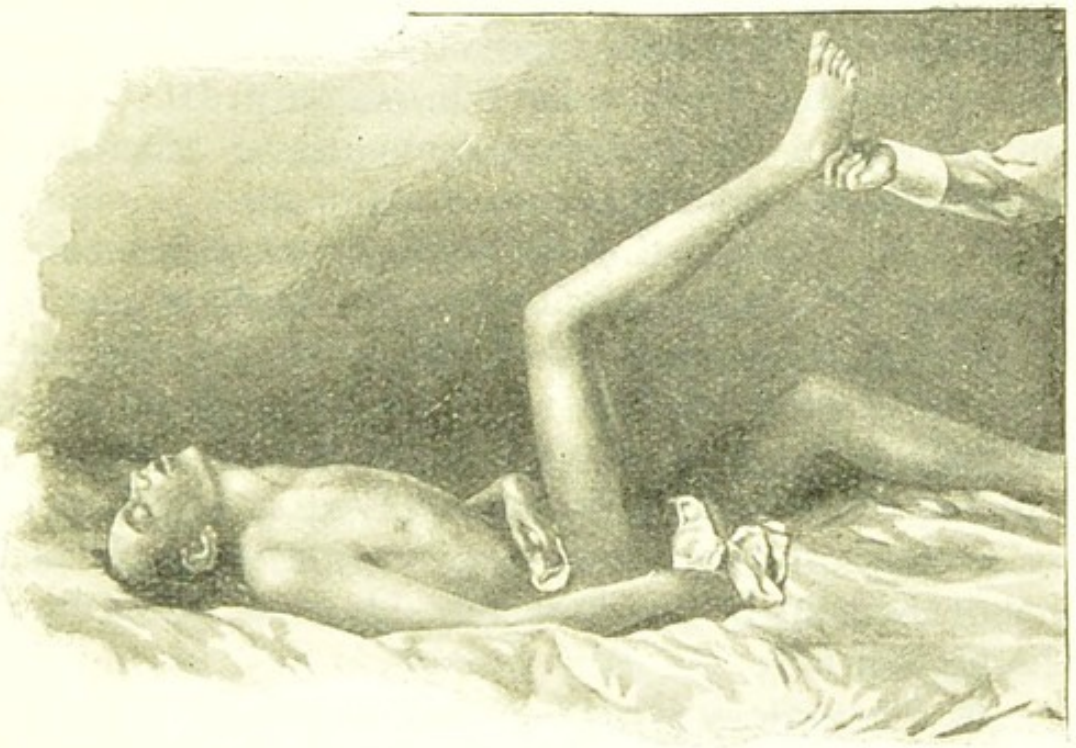


Fig. 125.—Head retraction and Kernig's sign.
(After a photograph by Dr. W. Thyne.)

ing of the whole body backwards (*opisthotonos*), or sideways (*pleurosthotonos*), or forwards (*emprosthotonos*).

The term **tetany** is applied to a peculiar form of tonic spasm affecting the hands and feet. The fingers are pressed together, flexed at the metacarpo-phalangeal joints, but extended at their phalangeal articulations. The thumb is tucked in under cover of the fingers. The hand therefore assumes a conical shape, and is sometimes said to be in the "obstetric position," that being the position in which the accoucheur holds

his hand when introducing it into the vagina. The wrist is also flexed. In the foot one finds marked flexion of the toes and anterior part of the foot, sometimes also of the ankle. Tetany is chiefly a disease of infancy, but is sometimes met with in adults who have dilatation of the stomach and in other conditions.

Clonic spasms are of various degrees of severity. If very widespread they are spoken of as **convulsions**, and are seen typically in epilepsy. If the patient gives a history of fits, their character should be inquired into, following the lines laid down on p. 10. Should the observer be fortunate enough to witness an attack, he should note—

i. *The nature and distribution of the movements.*—Are they general, or confined to one limb or part of a limb? What part is first and what last affected? Are the convulsions tonic or clonic? Is there any struggling, arching of the back, or attitudinizing? Are the abdominal muscles involved or not?

ii. Is there any *involuntary evacuation* of the bladder or rectum? Is there any blood or froth about the mouth?

iii. *The state of the eyes.*—Is the conjunctival reflex present or abolished? Do the pupils react to light? Is there any inco-ordinate movement of the eyeballs?

iv. How does the patient behave *after the fit?*

If one group of muscles be first affected, the spasm spreading to others by degrees, it indicates a spread of the irritation along the surface of the cortex cerebri This occurs typically in Jacksonian epilepsy.

Tremor consists of more or less rhythmical oscillations of any part or parts of a limb, and is due to the alternate contractions of a group of muscles and its antagonists. Tremor may be either *fine* or *coarse*. Fine tremor is usually more easily felt than seen. It occurs in exophthalmic goitre, alcoholism, and in some

forms of metallic poisoning. All forms of tremor are most easily seen by increasing the leverage at which the affected muscles act. Thus, tremor of the upper limbs is often brought out by getting the patient to extend his arms in front of him. In describing tremor, always note whether it is constantly present, or if it is affected in any way by voluntary muscular action. Also observe its rate, the amplitude of the movements, and whether they are regular or irregular. Ask the patient to lift a glass of water to his lips, or to attempt to follow the movements of your finger, and note whether the tremor is increased thereby (as it is, for example, in cases of disseminated sclerosis), or whether it is diminished or altogether abolished.

Tremor which only comes on when the patient attempts to use the affected muscles is described as *intention tremor*.

Clonic contraction of individual fibres or bundles of fibres in a muscle is termed **fibrillary twitching**. It is seen in many cases of progressive muscular atrophy, and indicates an abnormal state of nutrition in the spinal cells connected with the affected fibres.

To the transient flickering of a few muscle fibres (commonly known as "live-flesh" or "live-blood") the term **myokymia** is applied. It is most often seen in the orbiculares palpebrarum, and is usually an indication of fatigue or debility. It also occurs as an independent condition, and is then more or less general.

The term **choreic** is applied to involuntary movements of a purpose-like character occurring in individual muscles or groups of muscles. Such movements are seen most typically in chorea minor or St. Vitus's dance. They consist of abrupt involuntary twitchings or contractions which cause the patient (usually a child) to seem fidgety and unsettled. They are increased by mental agitation, but are often

diminished by voluntary muscular effort. If the movements are limited to one side of the body the term *hemichorea* is applied.

Choreic movements, if slight, can be elicited in two ways. First, one may ask the patient to hold both hands straight up above the head; or, second, one may ask him to spread out his hands, palms downwards, on the extended hands of the observer. In the former case it may be observed that the patient is unable to hold up his hands steadily for any length of time; in the latter, one may notice that little twitchy movements soon become evident in the patient's fingers.

If the patient be able to write at all, one may get him to scrawl his name with the affected hand, and keep the result for purposes of comparison later. In this way one is able to gauge any increase or diminution in the choreic movements.

Tics are co-ordinated purposive acts which are started in the first place by some external cause, or by an idea. By repetition they become habitual and finally involuntary, without any relation to the cause that first excited them. They may assume various forms; perhaps the most common are blinking of the eyes, smacking the lips, or rotation or nodding of the head. They can be distinguished from other involuntary movements by their complexity, and by the fact that they always retain their purposive character.

The term **athetosis** is used to describe a slow muscular contraction which leads to continuous and deliberate twisting movements specially affecting the hands and feet.

The last point to be noted regarding any abnormal muscular movement is whether or not it persists during sleep.

V. SENSORY FUNCTIONS

In investigating the sensory functions of a patient, we have to test the acuteness of the following forms of sensibility :—

1. Tactile sensibility. This includes the powers of appreciating touch and pressure. The ability to localize the stimulus may be observed at the same time.
2. Sensibility to pain.
3. Thermal sensibility.
4. The sense of position and the appreciation of passive movement.
5. The power of recognizing the size, shape, and form of objects.
6. The power of appreciating weight.

Other sensory faculties, as the appreciation of vibration and Weber's compass test, cannot be dealt with here.

In addition, one has to note the presence or absence of any abnormal sensations.

At the outset it is well to explain to the patient the nature of the tests to be performed, so as to secure, as far as possible, his intelligent co-operation. The eyes should then be bandaged, or the part under examination screened from sight, and the different forms of sensibility tested as follows :—

1. **Tactile sensibility.**—The feather end of a quill pen may be used as a stimulus, but for carefully mapping out areas of altered sensibility a small cone of cotton-wool is best.* It is so light that the element of pressure is entirely eliminated. A fine camel-hair brush also answers the purpose very well. If it be desired to test the sensibility of the skin to light touch over a hairy part, it is essential to shave it

* The untreated cotton-wool used by jewellers is the most suitable.

first, as the sensibility of the hairs themselves is so acute.

Tell the patient to say "Now" as soon as he feels a touch. Compare corresponding points on opposite sides of the body, and employ every now and then a negative test, asking the patient if he feels you touch him, in order to prevent his making random replies. The appreciation of *pressure touch* should be then tested; this may be done by touching him with the point of a finger or any blunt object. It is important that its temperature should not differ much from that of the skin, and the pressure must not be so heavy as to give pain or discomfort. Ask him also to localize the stimulus by describing or in other way indicating the exact position of the spot touched. This is important, as a patient may be able to feel the stimulus and yet not be able to localize it.

Sensibility to touch may be altered in various ways. (1) It may be entirely abolished. This constitutes **anæsthesia.** If the abolition affects the whole of one side of the body, it is termed *hemianæsthesia.* If the existence of anæsthesia be discovered, one must at once proceed to mark out its exact extent and boundaries. (2) Sensibility may be so altered that what should in health be felt as a mere touch produces a painful impression resembling pricking or burning. This is generally called **hyperæsthesia.** If hyperæsthesia be discovered, its extent should be carefully mapped out. Hyperæsthetic spots are sometimes met with, especially in hysterical patients. The commonest sites for these are over the brim of the pelvis, in the inframammary region, along the vertebral column, and on the scalp. Pressure on such spots may sometimes induce hysterical fits. If that occurs, the spots are spoken of as "hysterogenetic." (3) Sensation may be appreciated well enough, but

there may be great delay in its conduction, an appreciable interval occurring between the application of the stimulus and the response of the patient. This **delayed conduction** exists not infrequently in cases of alcoholic neuritis and tabes. (4) The stimulus may be badly localized, the patient believing, for example, that the outer side of a limb was touched when the stimulus was really applied to its inner aspect. Sometimes a touch on one side of the body is referred to a corresponding point on the opposite side; this is termed **allocheiria.**

2. **Sensibility to pain.**—Pain may be evoked either by a cutaneous stimulus, as the prick of a pin, or by pressure on the deeper structures, as the muscles or bones. Sensibility to superficial and to pressure pain should be tested separately.

(*a*) **Superficial pain.**—The point of a steel pin or needle may be used as the stimulus. Care must be taken that the patient distinguishes between the sharpness of the point (that is, its relative size) and the pain which the prick evokes; it often happens that even when sensibility to pain is abolished he can recognize that the stimulus is pointed, and thus confuse the observer by calling it "sharp." The relative sensibility to pain may be measured by an algesimeter, by which the observer can determine the amount of pressure on a needle that is necessary to evoke pain.

The application of a faradic current is also an excellent method of testing sensibility to pain. It enables one to gauge the degree of sensibility by noting what strength of current is necessary to cause pain, and then comparing the result with the corresponding area on the opposite side.

(*b*) **Pressure pain** may be examined by pressing firmly on the part with a blunt object, as the end of a pencil, or by squeezing the muscles. The degree of

sensibility may be measured by such an instrument as Cattell's algometer, by which the amount of pressure necessary to evoke pain may be determined. Abolition of pressure pain is often the most prominent sensory disturbance in tabes dorsalis.

Absence of sensibility to pain is termed **analgesia**; and an exaggerated sensibility, so that even a mild stimulus causes an unnatural degree of suffering, is known as **hyperalgesia.**

3. **Thermal sensibility** is most conveniently examined by using test tubes containing hot and cold water. The part to be tested is touched with each in turn, and the patient says whether each tube feels hot or cold. It is often important to determine the thresholds for heat and cold, i.e. the lowest temperature that feels warm and the highest that is cold. This can be done by noting the temperatures of the water in the tubes on thermometers contained in them; but in attempting this it is desirable to use silver rather than glass test tubes, as glass conducts so badly that there may be a considerable difference between the temperatures of the surface of the tube and of the water it contains. Note also the reactions evoked by high and low temperatures and the sensations they produce in the patient. It frequently happens that such temperatures evoke only pain, and may be called indiscriminately hot or cold.

It should be noted that the different forms of sensibility already mentioned may require to be tested on the accessible mucous membranes as well as on the skin surfaces. The sensibility of some viscera is also of importance. Thus the absence of pain on squeezing the testicle may be an early sign of tabes.

4. **Sense of position.**—The patient's eyes being carefully shut, take hold of one of his limbs and move it about in various directions through the air, finally

leaving it in some definite position, say semiflexed and slightly elevated ; then ask him to put the corresponding limb in a similar position. If there be no paralysis of the latter, and yet the patient is unable to imitate with it the position of the other, then there is reason to believe that the sense of position is impaired.

In the case of the hand the patient may be told that the fingers of one hand will be moved, and that he must imitate with the other the position in which they have been placed. In the case of the foot he may be told that the great toe will be placed pointing upwards or downwards, and that he must try to tell which it is.

In testing a patient's sense of position in this manner, be careful not to allow the part tested to touch any other skin surface ; otherwise the patient will be able to appreciate its position by the information derived from his ordinary sense of touch.

A very delicate test for the sense of position in the upper limbs consists in shutting the patient's eyes and then making him hold his arms straight out in front of him with the fingers in a horizontal row. After a moment or two, if the muscular sense be defective, the fingers cease to remain in an even line. Some will rise a little, others fall, or even become twisted in below the rest.

The **appreciation of movement** is closely related to the sense of position, and should be tested at the same time. Grasp any segment of a limb firmly, and then move it gradually into another position ; ask the patient to say "Now" as soon as he recognizes the movement, and note the angle through which the limb was moved. If the appreciation of movement be diminished this angle is many times greater than that which is necessary in a normal limb, but if the defect be slight it may be necessary to measure the range of the movement accurately for comparison.

Movements of, at most, 3° can be appreciated at all normal joints. Finally, test if the patient can recognize the direction of the movement, that is, whether the joint is flexed or extended. It often happens that the patient can recognize the occurrence of a movement though he is ignorant of its direction.

5. The **recognition of size, shape, and form.**—These faculties can be tested most accurately in the hands. To test size, place in the patient's palm objects of the same shape, but of different sizes, as small rods or matches of different length. Two objects should be applied consecutively, and he is asked to say which is the larger.

To test the power of recognizing **form,** familiar objects, as coins, a pencil, a penknife, scissors, etc., are placed in the hand, and the patient is asked to identify them or to describe their form. Loss of this power is generally known as **astereognosis.**

6. **Appreciation of weight.**—Place in the patient's hand substances which resemble one another as far as possible in every respect except as regards weight. Metal balls covered with leather, some being solid and others hollow, are often used for the purpose. In their absence one may use two match-boxes, one full, the other empty, or some other extemporized device. A solid ball and a hollow one may be placed one in the patient's right hand, the other in his left, and he is then asked to state which is the heavier ; or one hand may be tested at a time, the balls being lifted one immediately after the other. If the leg is being investigated, the weights should be placed in a handkerchief and slung round the patient's ankle.

Are there any abnormal sensations present?—These are termed paræsthesiæ, and consist in various sensations experienced by the patient in the absence of any outward stimulus. The commonest

of these are a feeling of "needles and pins," of numbness, of heats or chills, of pressure or tightness (a good example of the latter being the "girdle pain" of tabes dorsalis), of itching—sometimes termed pruritus—or a feeling as if insects were crawling over the body (formication).

The term **aura** is applied to the curious paræsthesiæ which frequently precede an epileptic fit and serve as a warning of its approach.

VI. REFLEXES

There are three classes of reflexes which one has to test—

1. The superficial reflexes.
2. The deep or tendon reflexes.
3. The organic reflexes (including the action of the sphincters).

We shall consider these separately.

1. Superficial Reflexes

In these the simplest form of reflex action is concerned. On stimulation of a certain part of skin or mucous membrane, contraction of certain muscles results. The path of the impulse is by the sensory nerve fibres to the grey matter of the cord or to a higher centre in the brain-stem or forebrain, thence by motor nerve fibres to the muscle. A lesion in any part of this path causes the reflex to disappear. Thus, anæsthesia of the skin, disease of the sensory fibres or posterior nerve roots, changes in the grey matter of the cord, lesions of the motor nerve fibres or of the fibres of the muscles, may all cause abolition of the superficial reflexes. In addition to this, it must be borne in mind that the reflex excitability of some individuals is normally very much greater than that of others, and this makes it difficult for one to estimate the value of slight alteration in the reflexes unless the

lesion is unilateral, in which case the healthy side can be taken as a standard of comparison. The investigation of the superficial reflexes is of more value as affording information regarding the health or otherwise of the reflex arc concerned than as a guide to the presence or absence of disease elsewhere. In hemiplegia the superficial reflexes are usually diminished on the healthy side.

The chief superficial reflexes of spinal origin, their nature, the mode of obtaining them, and the level of the cord concerned in their production, are given in the table on p. 502.

The **plantar reflex** demands special consideration. In order to elicit it the patient should be in the supine position, with the lower limb semiflexed at the hip and knee and rotated outwards, the knee resting on a pillow. This induces muscular relaxation, which may be increased by distracting the patient's attention by conversation or by making him perform with his hands some act which requires attention. Care should be taken that the sole of the foot be warm and dry. The plantar region is stimulated by gentle scratching with the finger-nail or with a soft quill. In healthy adults a minimal stimulus produces a contraction of the tensor vaginæ femoris, often accompanied by a slighter contraction of the adductors of the thigh and sartorius. With a slightly stronger stimulus, flexion of the four outer toes appears, which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together, the ankle being dorsiflexed and inverted. With still stronger stimuli, violent regular movements of the limb occur, which spread to the lower part of the trunk and to the opposite side. The position of the foot at the height of a response to a moderate stimulus is shown in Fig. 126 ("flexor response").

It is doubtful whether the plantar reflex is ever constantly and completely absent in healthy subjects.

In infants up to the age of learning to walk, the reflex is very brisk, and differs markedly from that in adults. The earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes, with eversion of the foot or dorsiflexion of the ankle, and subequently by flexion of the hip and knee ("infantile response").

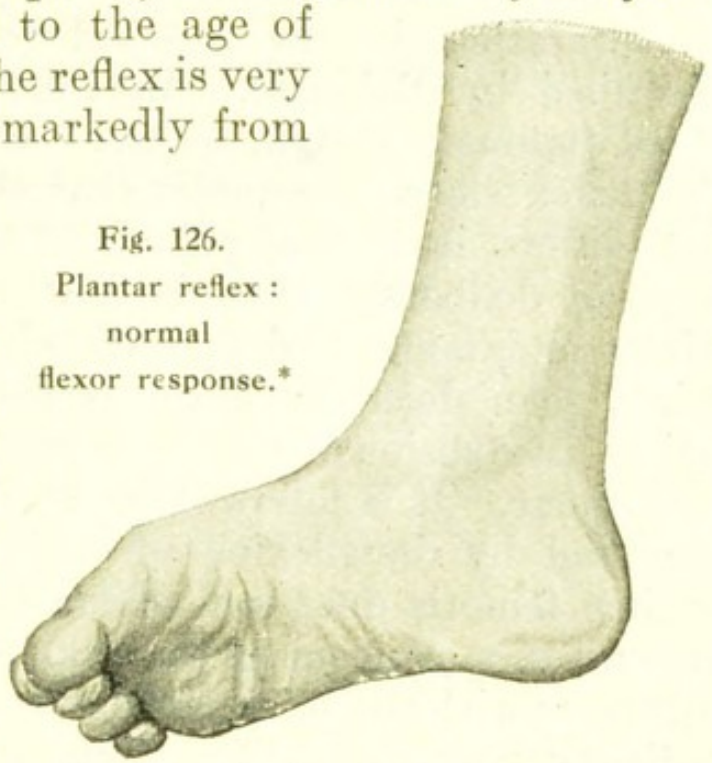


Fig. 126. Plantar reflex: normal flexor response.*

During sleep the plantar reflexes are diminished and the infantile and adult forms preserved, save in some children up to the age of 12 years, where in deep sleep the infantile form of reflex returns.† In pathological conditions the

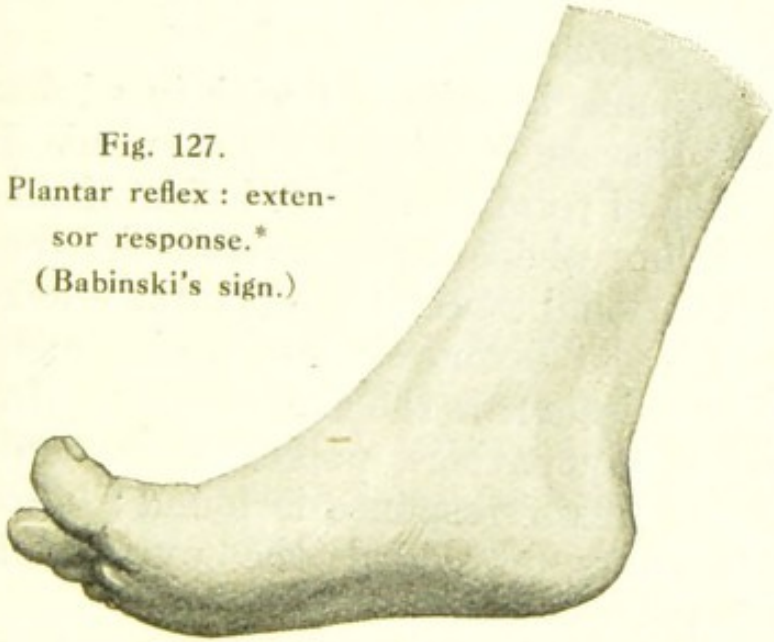


Fig. 127. Plantar reflex: extensor response.* (Babinski's sign.)

* After instantaneous photographs by Dr. James Collier.

† An extensor response is also obtained in some adults during deep sleep (Rudolf).

reflex varies, and may be of great diagnostic importance. In *lesions of the pyramidal systems* an alteration in the type of responses was first described by Babinski. In this, which is spoken of as **Babinski's sign,** or the "extensor response," the reflex closely resembles that obtained in infants, but differs in a few points. The whole response is more deliberate than is that obtained either in adults or infants, and appears with much more certainty than does the flexor response to each stimulation. Extension of the great toe precedes all other movement. It is followed by extension of the other toes and by dorsiflexion of the ankle. The tensor vaginæ femoris does not contract early. The small amount of movement at the ankle is conspicuous, and contrasts with the brisk movement at the ankle in the ordinary response. The extensor response is often more easily elicited by stimulation of the outer part of the sole, whereas the flexor response is usually more easily obtained by stimulating the inner part. Fig. 127 shows the position of the foot at the height of a moderate extensor response.

The extensor response is only met with in adults in cases of organic disease involving the pyramidal systems, and in cases of total transverse lesion of the cord is the only reflex phenomenon present in the lower limbs. In functional cases, if the plantar reflex can be elicited at all, the flexor response is obtained. In tabes and peripheral neuritis the same is true. In neurasthenia, chorea, myopathies, poliomyelitis, and paralysis agitans the flexor response is found; also in intracranial tumours, provided the pyramidal systems are not involved.

The **epigastric and abdominal reflexes** (*see* p. 502) are also extremely valuable signs, as they disappear when the pyramidal tract of the same side is

in any way affected. It is often impossible to obtain them in old or obese people, and in women who have borne many children.

The following superficial reflexes are dependent on cranial nerves :—

i. **Conjunctival.**—Elicited by touching the conjunctiva, resulting in contraction of the orbicularis palpebrarum. The nerves concerned are the 5th (sensory) and the 7th (motor).

ii. **Pupil reflexes.**—(*See* pp. 461 and 526.)

iii. **Palate reflex.**—Elevation of the palate on touching the mucous membrane covering it. The nerves concerned are the glosso-pharyngeal and the vagus (or spinal accessory ?).

2. Deep or Tendon Reflexes

If a muscle be put upon the stretch and its tendon be then sharply struck, the muscle immediately contracts. This is spoken of as a *deep* or tendon reflex. It is very doubtful, however, whether one is correct in speaking of it as a reflex at all. It would seem that the contraction of the muscle follows too rapidly on the blow upon the tendon to permit of there being time for a reflex arc being traversed. What really happens is probably this. The stretching of the muscle reflexly increases its "tone." When the tone is thus raised, any mechanical stimulus—in this case the blow upon the tendon—by suddenly increasing the tension of the muscle, excites in it a direct contraction. The only point where a reflex act really comes in, therefore, is in the preliminary raising of the muscle tone. Without this increase of tone, however, the subsequent direct contraction would be impossible.

We have already indicated (p. 414) that the "tone" of a muscle is dependent upon the cells in the anterior cornua of the spinal cord—i.e. the lower neurones.

Chief Superficial Reflexes of Spinal Origin

Reflex	How excited	Result	Level of Cord concerned
Plantar	Stroking sole of foot.	Movements of toes, of toes and foot, or leg.	Lower part of lumbar enlargement (5th lumbar and 1st sacral segments).
Gluteal	Stroking skin of buttock vertically.	Contraction of gluteal muscles.	4th and 5th lumbar segments.
Cremasteric	Stroking skin at upper and inner part of thigh.*	Drawing upwards of testicle.	1st and 2nd lumbar segments.
Abdominal	Stroking abdominal wall from costal margin to nipple line.	Contraction of abdominal muscles.	11th dorsal to 1st lumbar segment.
Epigastric	Stroking side of chest downwards from nipple.	Drawing in of epigastrium on same side.	7th to 9th dorsal segments.
Scapular	Stroking skin in interscapular region.	Contraction of scapular muscles.	5th cervical to 1st dorsal segment.
Bulbo-cavernosus	Pinching dorsum of glans penis.	Contraction of bulbocavernosus.	3rd and 4th sacral segments.

* The cremasteric reflex can often be most easily elicited by pressing over the sartorius in the lower third of Hunter's canal.

When the control of the upper neurones over the lower is cut off, the latter produce an increased "tone" in the muscles, with the result that the tendon reflexes are more easily excited than normally. In other words, their reflexes are increased or exaggerated. Exaggeration of tendon reflexes, therefore, is characteristic of lesions affecting the upper neurones—i.e. the cerebral cortex, or the fibres passing from it to the anterior horns of the cord. A similar exaggeration may be brought about by anything which stimulates the lower neurones, thus making them more able to resist the controlling influence of the upper. Strychnine and the toxin of tetanus are able to do this, and therefore produce an increase of the tendon reflexes.

On the other hand, anything which impairs the activity of the lower neurones will cause a diminution in the tone of the muscles, and will thus make the tendon reflexes correspondingly difficult to elicit. Diminution or abolition of the tendon reflexes is therefore characteristic of lesions affecting the lower or spinal neurones. Of course, any disease of the muscle fibres themselves, or of the sensory fibres which proceed from the muscle to the spinal cord, and along which the stimulus travels when the muscle is first stretched, which stimulus causes the reflex increase of tone, will also cause a diminution of the tendon reflexes by preventing the necessary increase of tone from taking place. Hence it is that in tabes dorsalis, in which the posterior roots are much involved, the deep reflexes are absent.

There is only one point more to be referred to, and it is rather a confusing one. In a lesion—e.g. a fracture dislocation—which produced complete transverse destruction of the cord at any level, one might expect that, owing to the cerebral influences being cut off, all the deep reflexes below that level would be exagger-

ated. This, however, is not the case. The reflexes in that condition are, as a rule, totally abolished. The explanation of this apparent anomaly is still a little doubtful. It has been supposed by some that it is to be attributed to the cutting off of a cerebellar influence which descends to the cells of the anterior cornua, and, so to speak, energizes them, so that they are able to maintain the tone of the muscles. It must be admitted, however, that experimental evidence is totally opposed to this conclusion, as lesions of the cerebellum are followed by exaggeration and not by abolition of the deep reflexes.

The **knee jerk** or **patellar tendon reflex** is the best known of the deep reflexes. It consists in a contraction of the quadriceps extensor when the patellar tendon is tapped. The spinal segments concerned are the 3rd and 4th lumbar.

How to elicit the knee jerk.—If the patient be able to sit up, get him to sit on a chair or on the edge of the bed and cross one knee over the other. If he be unable to effect the latter movement, pass your wrist under the knee to be tested, resting your hand on the opposite knee and allowing the patient's leg to swing suspended, as it were, on the back of your wrist. If he cannot sit up, bend the knee up a little as he lies on his back, and support it by allowing it to rest on your hand or the back of your wrist.

The result of this disposition of the limb is slightly to stretch the quadriceps extensor, and so reflexly to increase its tone. The next thing to do is to try to divert the patient's attention. This may be done by asking him to "let the leg hang as if it did not belong to him," or by engaging him in conversation. The patellar tendon must then be struck a sharp blow midway between the patella and its insertion. The edge of the hand may be used for the purpose, or the

edge of a thin book; or the ear-piece of a stethoscope, especially if it be surrounded by a rim of solid india-rubber; or, best of all, a percussion hammer.

Immediately after the blow the foot will be observed to be jerked up from the sudden contraction of the muscle.

The briskness of the knee jerk varies greatly in different individuals. In health, however, it is hardly ever entirely absent. Sometimes one is unable to elicit it without having recourse to what is known as **reinforcement** of the knee jerk. This consists in asking the patient to make some strong voluntary muscular effort with the upper limbs. One may ask him, for example, to hook the fingers of the two hands together and then to pull them against one another as hard as possible. Whilst he is doing so, one tries to elicit the knee jerk, and one usually gets it more readily than under ordinary conditions. How reinforcement acts it is a little difficult to say. According to some, it is by increasing the general muscular tone throughout the body; according to others, it acts by diminishing the inhibitory cerebral control.

The following tendon reflexes are similar in nature to the knee jerk, but—probably owing to mechanical difficulties in producing adequate stretching of the muscle—are not usually so easy to obtain in health, and may indeed be entirely absent.

Ankle jerk.—Grasp the dorsum of the foot with one hand and hold up the leg with it. Slightly dorsiflex the foot so as to put the tendo Achillis on the stretch, then with the other hand sharply flick the latter on its posterior surface. A sharp contraction of the calf muscles results. This reflex can also be conveniently elicited when the patient is kneeling on a chair.

Adductor jerk.—This is produced by abducting

the thigh and tapping the tendon of the adductor magnus. Contraction of the adductors results. Sometimes in patients who have very exaggerated reflexes one finds that on tapping the patellar tendon a sudden contraction occurs in the adductor muscles of the opposite thigh. This is termed the **crossed adductor jerk.** Its explanation is still rather obscure, but it is apparently a truly reflex phenomenon.

Triceps or elbow jerk.—Flex the elbow to more than a right angle, then tap just above the olecranon. The triceps contracts. The reflex depends upon the 8th cervical and 1st dorsal segments.

Biceps or flexor jerk.—Flex the elbow to a right angle and place the forearm in a semipronated position ; then strike the lower end of the radius, and the elbow flexes owing to contraction of the biceps and supinator longus. The 5th and 6th cervical segments of the cord are concerned.

Wrist jerk.—Produced by letting the hand hang down, and then striking the extensor tendons just above the wrist. The hand is jerked up. This reflex depends upon the 6th cervical segment.

Jaw jerk.—Ask the patient to open his mouth, but not too widely. Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion. A contraction of the muscles that close the jaw results. This jerk is rarely present in health. The motor nucleus of the 5th nerve is the centre involved.

The term *clonus* is applied to the following tendon reflexes :—

Ankle clonus.—To elicit this phenomenon, bend the patient's knee slightly and support it with one hand, grasp the fore part of the foot with the other hand, and suddenly dorsiflex the foot. The sudden strain put upon the soleus muscle causes it to contract. The pressure of the hand upon the sole of the foot

is meanwhile continued, and, when the contraction ceases, causes the muscle again to become tense, and so produces another contraction in the latter. In this way a whole series of contractions—i.e. a clonus—results.

The relative tendency to the development of ankle clonus on the two sides is best estimated by slowly dorsiflexing the foot and observing the exact point at which the movements first begin. The less the degree of dorsiflexion required to produce the clonus the greater is the tendency to the development of the latter.

In cases of functional paralysis a spurious clonus may be elicited. It is usually ill sustained and irregular in rhythm, and can be recognized by the feeling of voluntary contraction in the muscles, especially at the beginning of the clonus.

Ankle clonus is nearly always a sign of disease. The spinal segments concerned in it are the 1st to the 3rd sacral.

Knee clonus.—In cases where the knee jerk is exaggerated, one can sometimes elicit a knee clonus by extending the patient's leg and then suddenly pushing down the patella towards the foot. If the pressure on the latter be continued, a series of clonic contractions of the quadriceps can in many cases be produced.

3. Organic Reflexes and Sphincters

This term includes such processes as respiration, deglutition, micturition, and defæcation. They depend upon complex muscular movements excited either by stimulation of mucous membranes or, in the case of respiration, of a centre in the medulla.

One should always ascertain from the patient whether he has any difficulty in swallowing, noting specially whether there is any regurgitation of food

through the nose. The function of **deglutition** does not usually require to be specially tested beyond the examination necessary to exclude the existence of an obstruction (p. 52).

Defæcation.—The patient should be questioned as to any difficulty in the act, and as to the presence or absence of tenesmus. Note also the occurrence or not of incontinence of fæces.

The reflex action of the anal sphincter may be tested by introducing the oiled finger into the anus, and noting whether contraction of the sphincter occurs with normal force, whether it be weak or altogether inactive, or whether any spasm be excited.

The activity of the anal sphincter reflex may also be tested by pricking the skin in the neighbourhood of the anus. If the conditions be normal, a brisk contraction of the sphincter should immediately be visible. This depends upon the 5th sacral segment.

Micturition.—The patient should be questioned as to difficulty or pain in the act (*see* p. 9). One should then note whether there is either *retention* of urine or *incontinence* of it. If there be incontinence, ascertain by the use of the catheter whether it be due to the *overflow* from a distended bladder, or whether it be a *reflex incontinence*—i.e. whether the bladder merely fills up and then empties itself completely by reflex action. In another group of cases the patient feels the desire to micturate, and is unable to restrain the act, which takes place at once. This is spoken of as *precipitate micturition*.

The centres for the bladder and rectum used to be regarded as situated in the 3rd and 4th sacral segments of the cord. It is now believed, however, that they are situated in the hypogastric and hæmorrhoidal sympathetic plexuses.

VII. TROPHIC FUNCTIONS

In disease of the nervous system the nutrition of different tissues or organs may be impaired. The *bones* may become more brittle from interstitial absorption, or may exhibit spontaneous fracture (osteopathies), or the *joints* may be the seat of painless effusion with or without atrophy or enlargement of the articular ends of the bones (arthropathies). In other cases the bones and joints are involved together (osteo-arthropathies). More commonly the *skin* is the seat of change. It may exhibit an erythema, which may pass on to ulceration and the formation of bed-sores at points of pressure; pigmentary changes may develop in it, or it may be the seat of various eruptions—urticarial, vesicular, pemphigoid, or herpetic—or it may be simply glossy. Perforating ulcers may appear, usually on the toes or soles of the feet, as in tabes, or there may be actual gangrene, or the development of painless whitlows. In other cases it is the epidermic appendages which especially suffer change, the hair falling out, or the nails becoming dry and brittle. Atrophy of the *muscles* is a common phenomenon, and may be marked by the appearance of the reaction of degeneration (p. 520). More rarely, certain glandular organs, such as the testis, may be the seat of atrophic change.

In taking a nervous case it is necessary to be on the outlook for such changes, and to note their situation and extent.

VIII. ELECTRICAL EXAMINATION OF MUSCLES AND NERVES

1. **Apparatus.**—For purposes of diagnosis, as distinguished from therapeutics, electricity is applied in three forms—first as the continuous current, second as the faradic current, third by using the discharge

through Crookes's tubes, with high vacua and suitable internal arrangements, for the purposes of skiagraphy by the X-rays described by Prof. Röntgen. The details of this last application are beyond the scope of the present work.

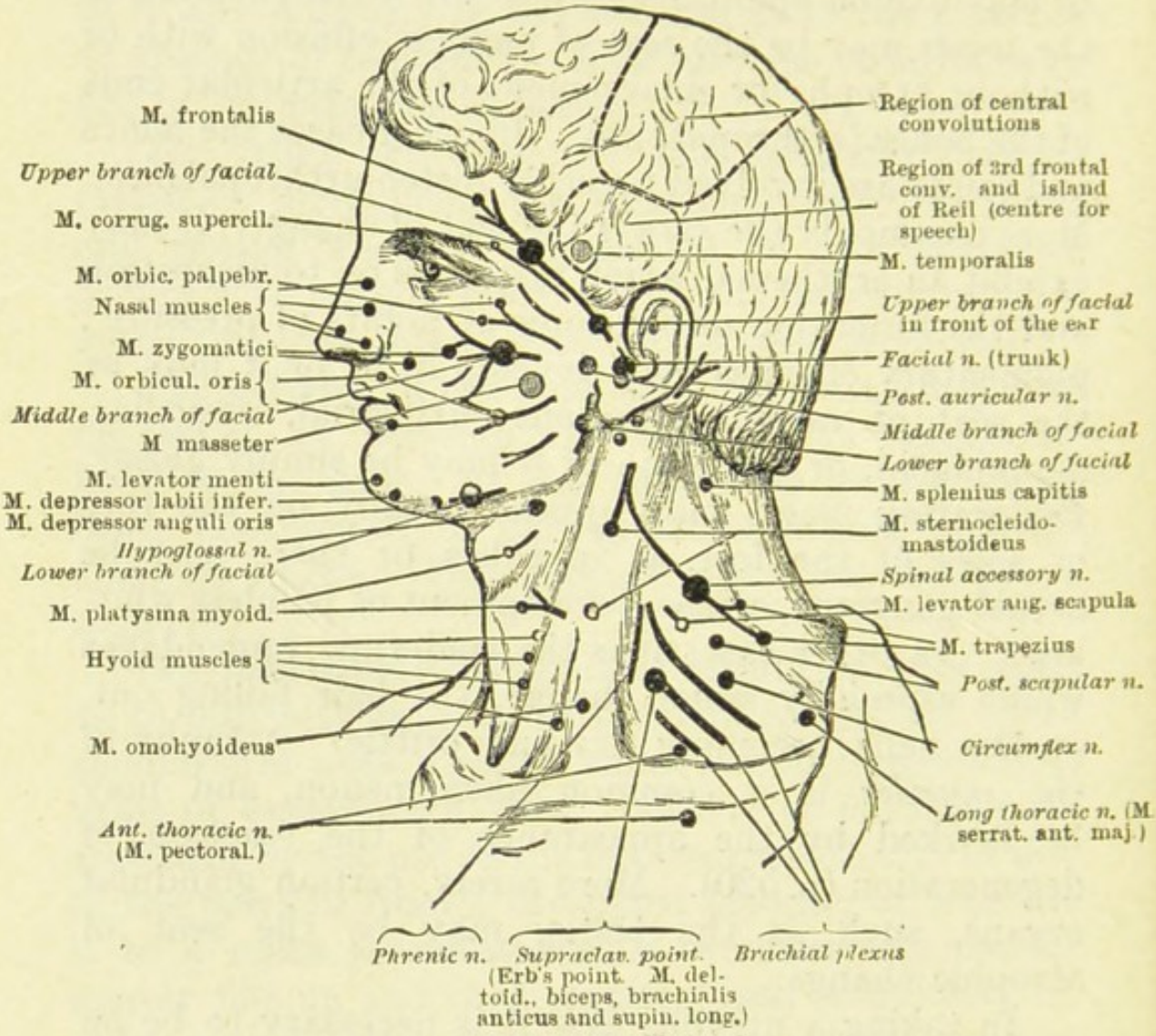


Fig. 128.—Motor points, face and neck.

The **continuous current** must be of sufficient intensity to overcome the resistance of the body and yet have enough strength left to stimulate the nerves and muscles. It must therefore have an available electromotive force of over 40 volts. A battery of thirty-two Leclanché dry cells is ample for ordinary use, as its electromotive force is decidedly higher than this to

start with, and will continue so for a considerable time if properly cared for and neither left too long

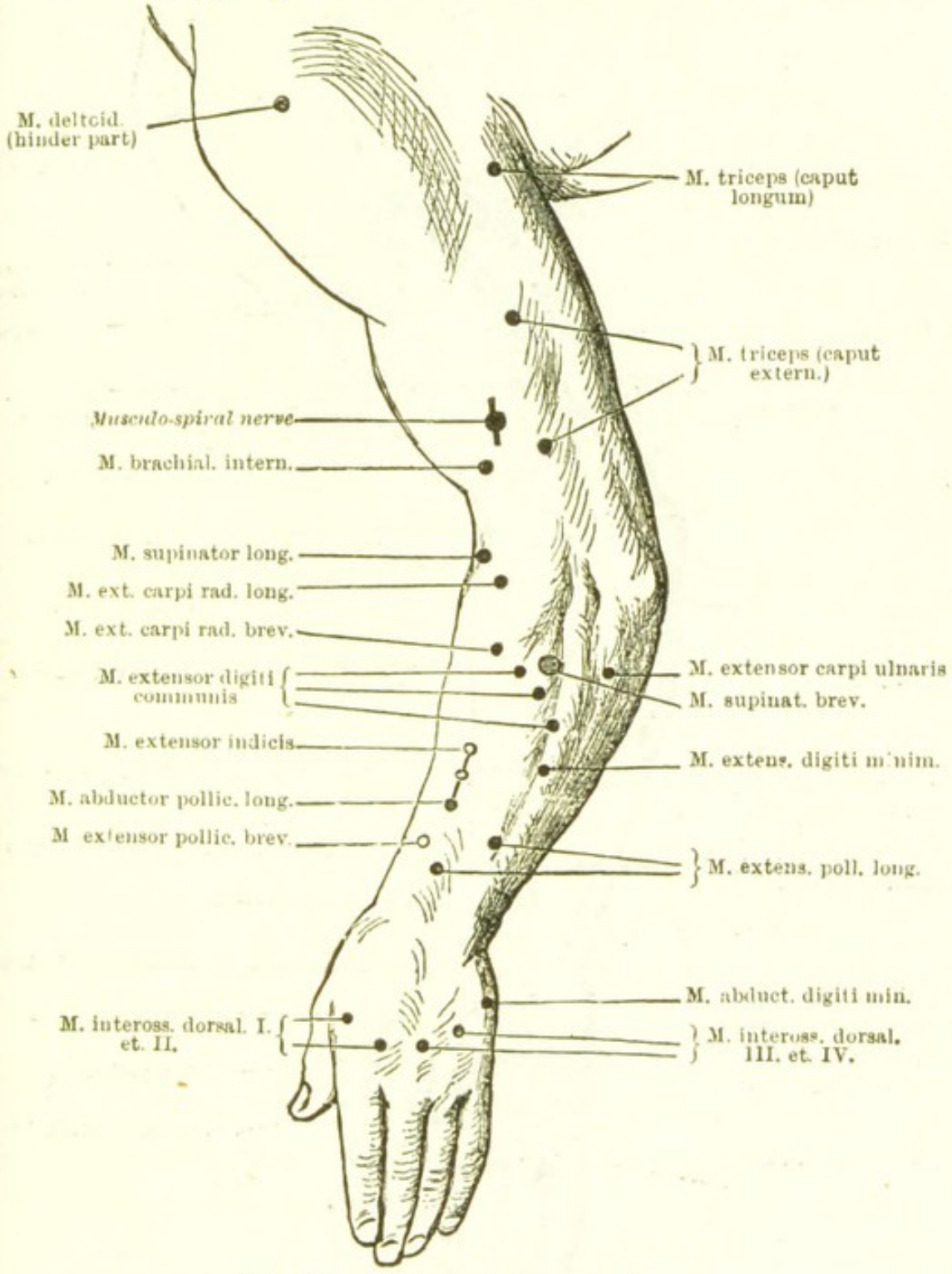


Fig. 129.—Motor points, back of arm.

absolutely idle nor employed to yield very heavy currents which would speedily exhaust the small cells. Where the continuous current from an electric light

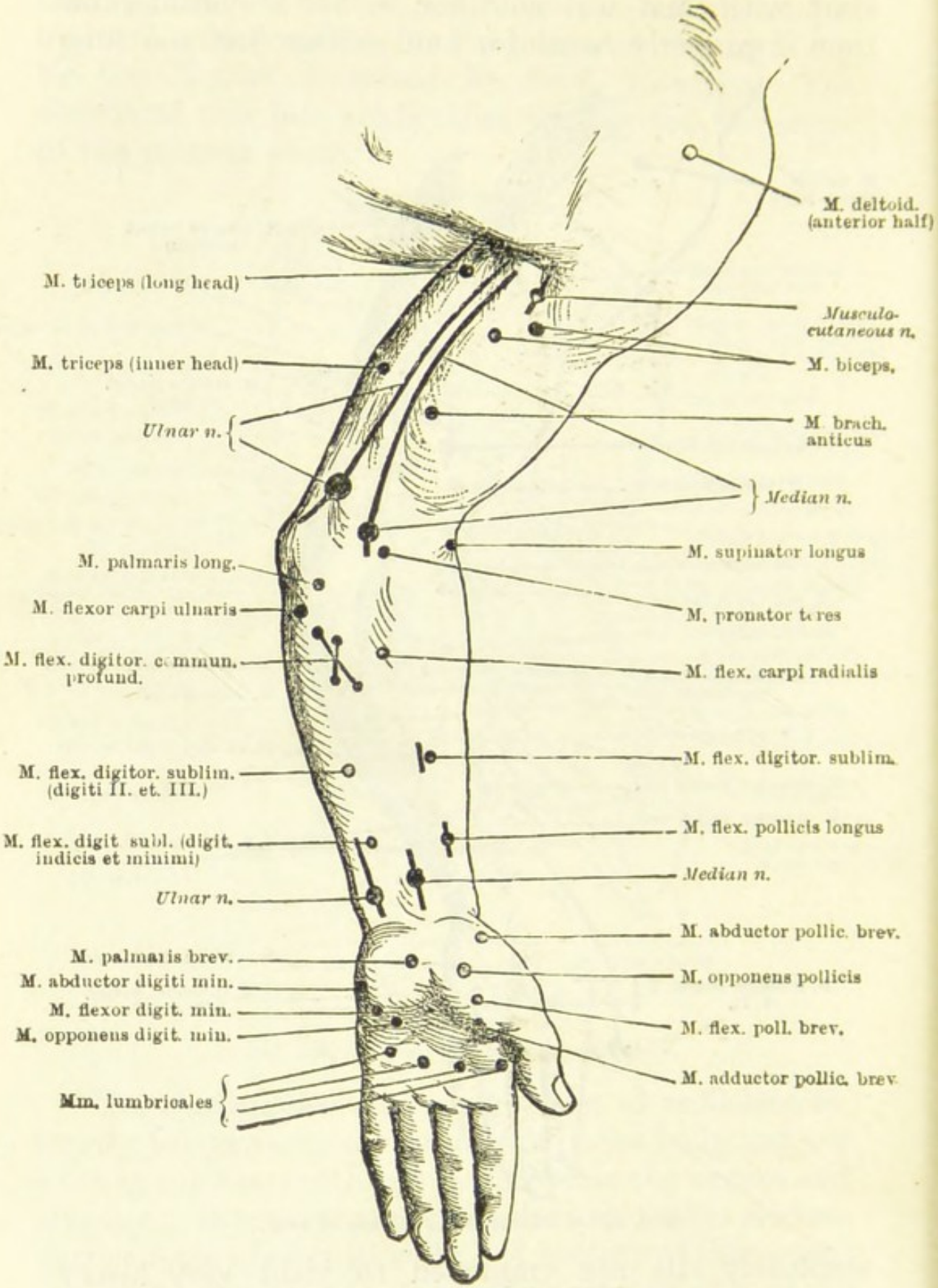


Fig. 130.—Motor points, front of arm.

installation is available, it may be used when precautions are taken for the safety of the patient by the employment of shunts to reduce the voltage to a suitable figure.

Whether battery or light installation with regulating board be used, it is of great importance to have

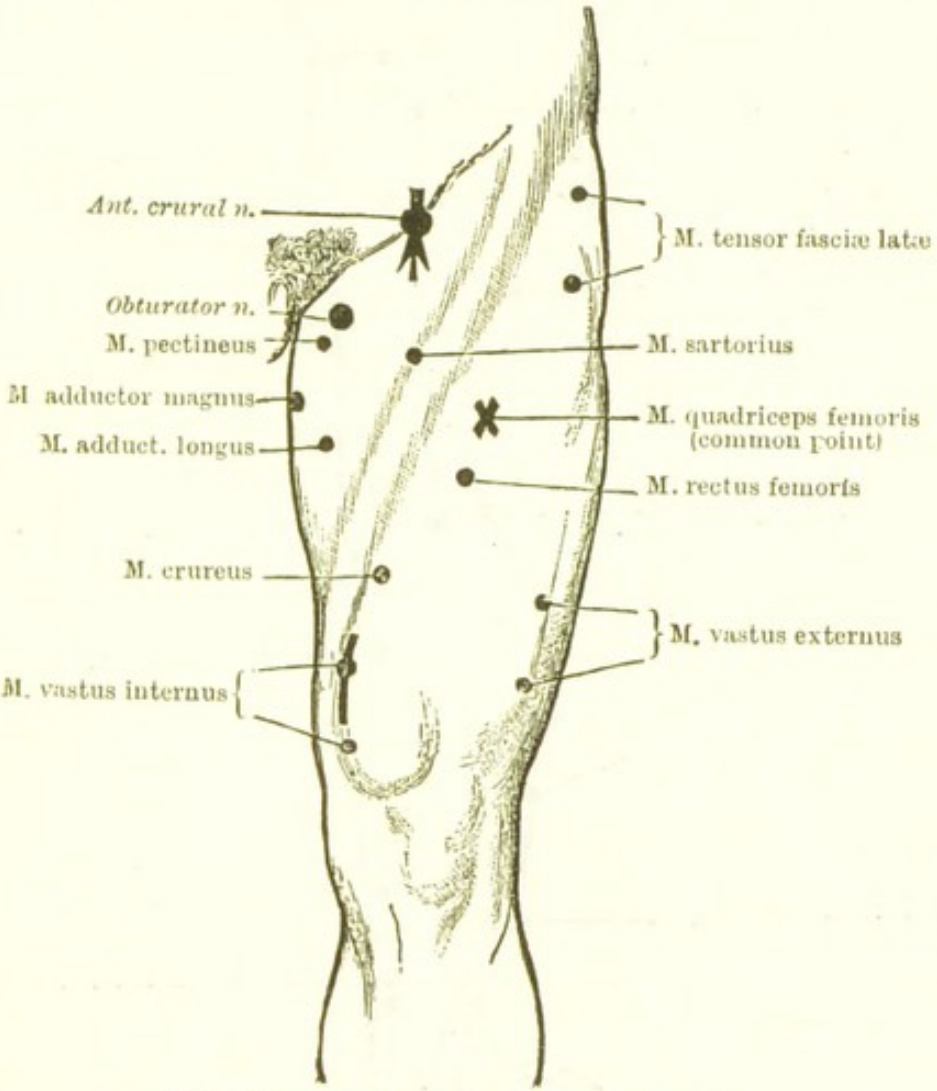


Fig. 131.—Motor points, front of thigh.

efficient accessory apparatus in the way of current reversers, galvanometers, connecting wires, electrodes. The galvanometers should give readings in milliamperes, not in unmeaning graduations, as too often is the case in those supplied with medical batteries.

For the **faradic current** the best form of apparatus is an induction coil of the sledge pattern with a

secondary coil of wire which should not be very fine, as very thin wire on the secondary coil produces too great an effect on the sensory, and too little on the

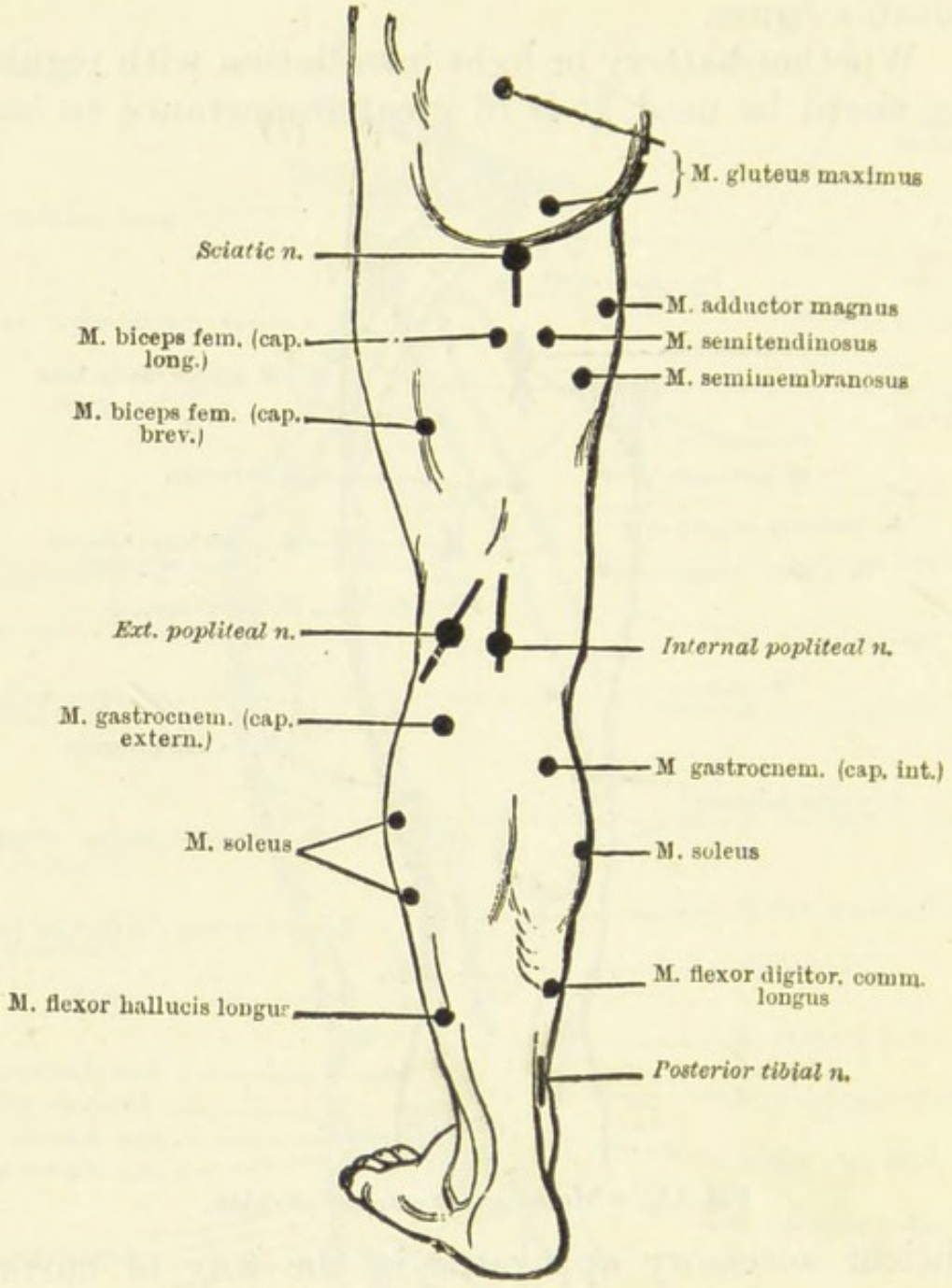


Fig. 132.—Motor points, back of thigh and leg.

motor nerves for most diagnostic purposes. To drive the coil one or two dry Leclanché cells are sufficient. It is often convenient to be able to pass from the continuous to the faradic current, and vice versa, without

changing the connections leading to the patient. This can be effected by a suitable switch.

One large flat electrode should be procured, and several small ones varying in size from a small spherical bulb to a disc of metal with a surface of about 60

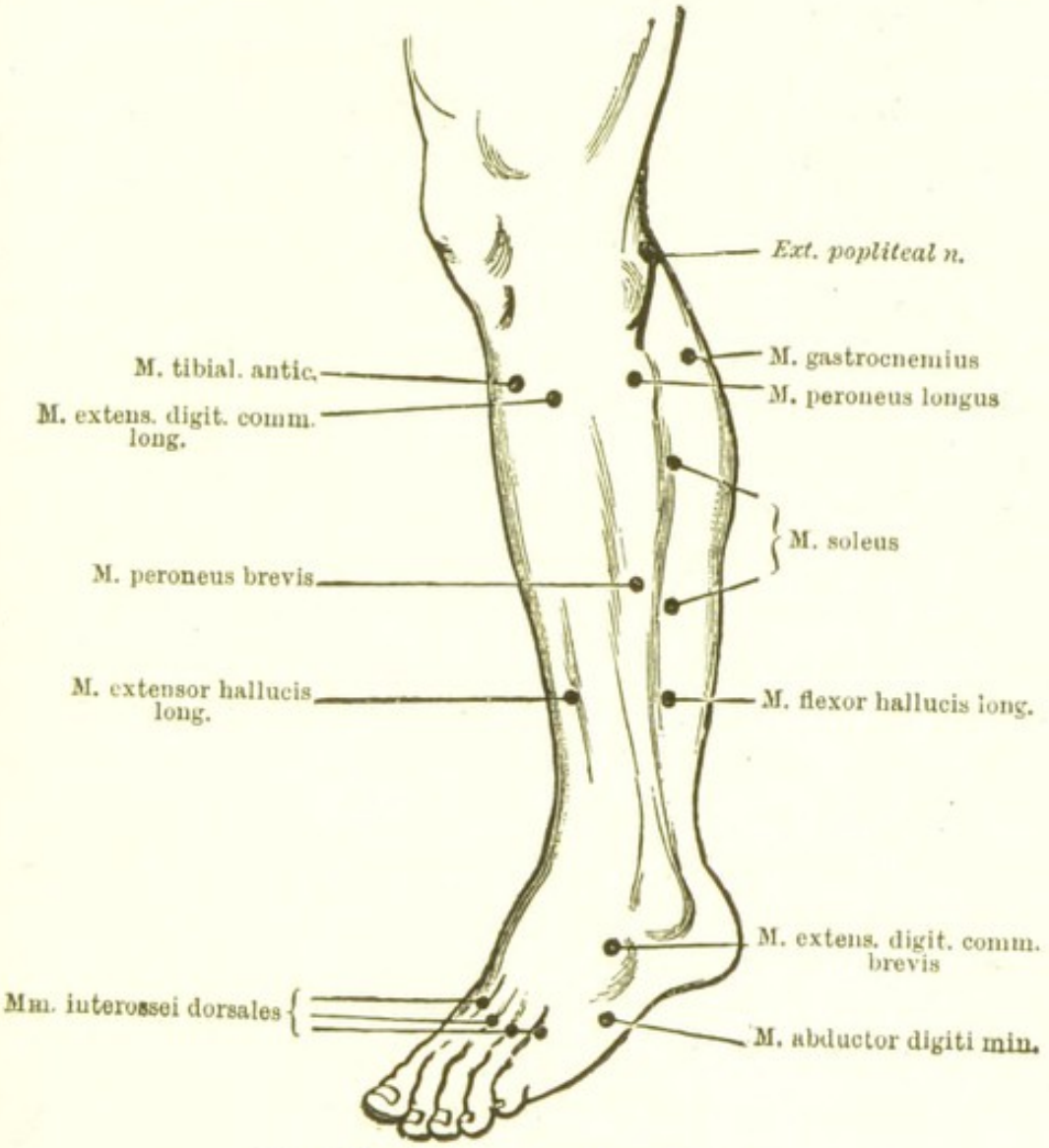


Fig. 133.—Motor points, side of leg.

or 100 square centimetres. These should be covered with wash-leather, which can be renewed at frequent intervals, and should be made to screw into a holder with a button by means of which the observer can interrupt the current.

2. **Electro-diagnosis** (Figs. 128–35).—**Begin**

with the faradic current.* It stimulates the nerves directly, the muscles only indirectly through their nerve supply. Examine systematically each nerve trunk in the area under consideration, and the motor point of every muscle supplied by each of these nerves. The motor points correspond for the most part with the points of entry of the motor nerves into the muscles which they supply, and are shown in Figs. 128–35. The observer should try the current upon himself before applying it to the patient, both to reassure the latter and to compare the effects it produces on himself with those elicited in the patient. The examination must be conducted in a good light, and the patient must be so placed that access to both sides is easily secured. Before examining any group of muscles, care must be taken that they are so far relaxed as to be able to respond readily to stimulation. A small electrode—either the button electrode or the disc of 10 to 20 square cm., according to circumstances—should be used for the part under examination, whilst the other (or "indifferent") electrode should be a larger plate placed either on the abdomen or between the shoulders. The electrodes and the skin where the tests are applied should be well soaked with a solution of common salt in warm water, but care must be taken to avoid the presence of any crystals of undissolved salt on the skin.

Note the intensity of the minimum current which produces contraction at each point, and compare the effects of a similar current on the corresponding point on the other side of the body.

The intensity of the current is recorded in terms of the distance of the secondary from the primary

* In dealing with children it is almost always necessary, where a careful electrical examination has to be made, to give an anæsthetic, although the anæsthesia need not be very deep.

coil—battery power, rate of interruption, and other adjustments being assumed to remain constant.

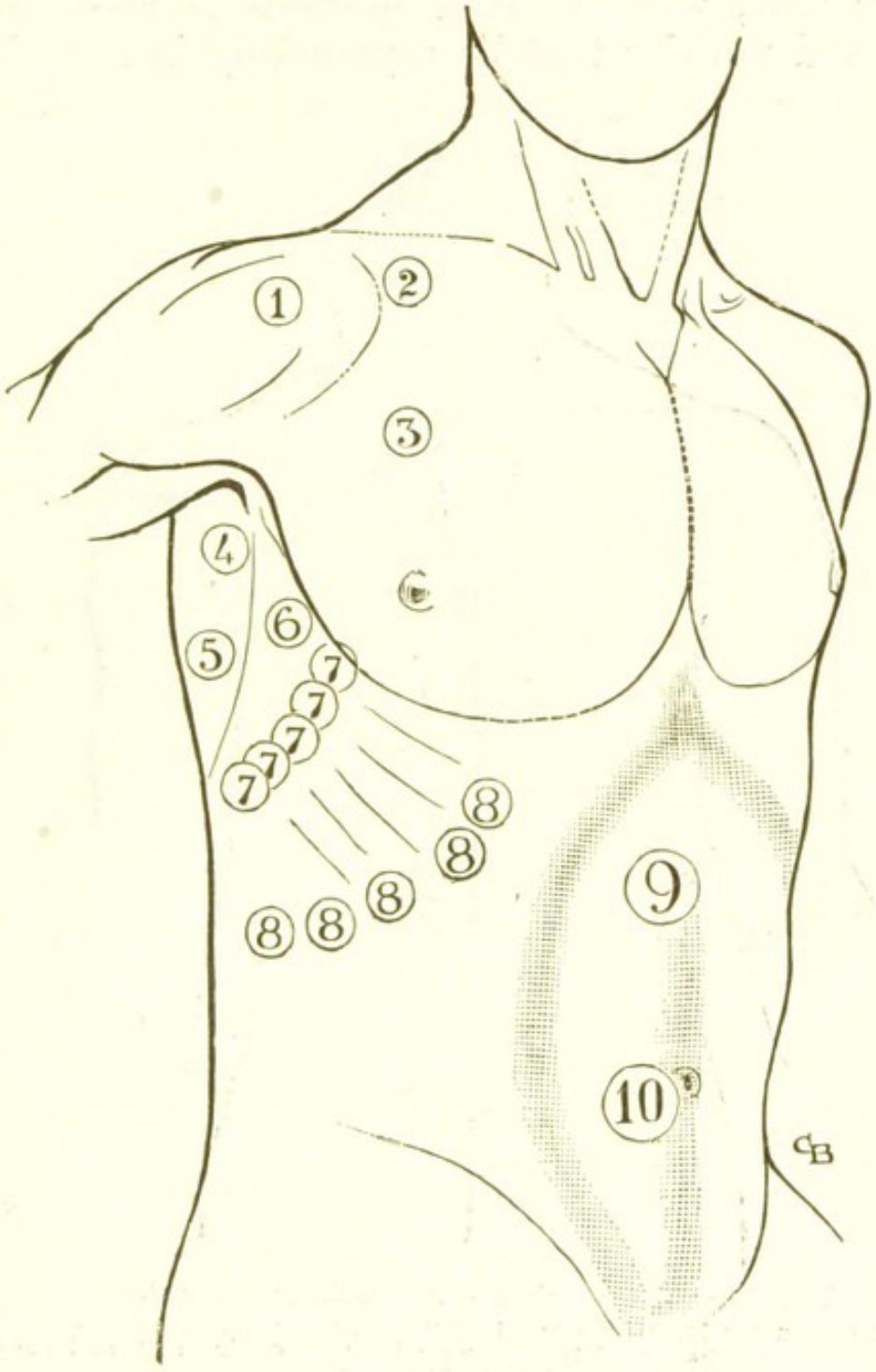


Fig. 134.—Motor points of the trunk, anteriorly.

1, deltoid (anterior part); 2, nerve to pectoralis major; 3, pectoralis major; 4, teres major; 5, latissimus dorsi; 6, nerve to serratus magnus; 7, serratus magnus; 8, obliquus externus; 9, rectus abdominis (upper part); 10, rectus abdominis (lower part).

When the faradic current has been employed, pass

to the **galvanic.** Use the electrodes in exactly the same manner as for the faradic examination, and remember that, when a small electrode is used, the nerves and muscles that lie superficially beneath it,

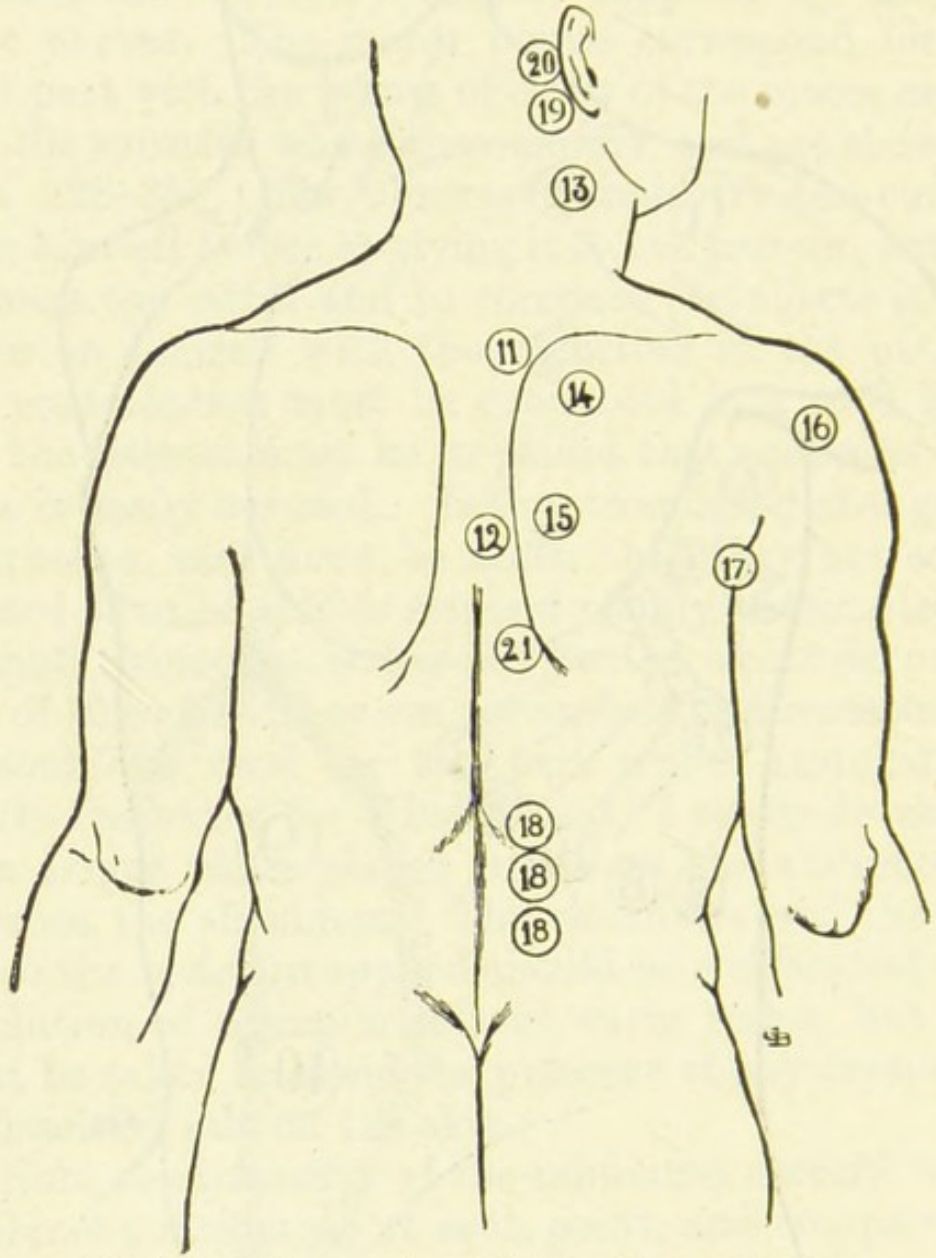


Fig. 135.—Motor points of the trunk, posteriorly.

11 and 12, trapezius; 13, splenius; 14, supraspinatus; 15, infraspinatus; 16, deltoid (posterior part); 17, teres minor; 18, erector spinæ; 19, facial nerve trunk; 20, posterior auricular nerve; 21, rhomboid.

and are therefore closer to it, are chiefly stimulated, since response to the stimulus depends on current density. Begin with a weak current, and gradually increase its strength till the muscle responds by slight

contraction. At each point employ first the cathode* and then the anode, and observe whether contraction occurs most readily when the circuit is completed or when it is interrupted. Record the strength of the current as indicated by the milliamperemeter, and compare it with that which produces similar effects at the corresponding point on the other side.

In **health** the cathodal closing contraction is the first to appear : in other words, the muscle responds more readily when the pole applied to it is the cathode, and when the electric circuit is completed ; a decidedly stronger current is required to elicit the anodal closing and opening contractions, and the cathodal opening contraction appears last of all. This sequence may be represented by the formula **C**.C.C. > **A**.C.C. > **A**.O.C. > **C**.O.C., or by the following expansion of it :—

Weak current	**C**.C.C.	—	—	—
Medium current	**C**.C.C.′	**A**.C.C.	—	—
Fairly strong current	**C**.C.C.″	**A**.C.C.′	**A**.O.C.	—
Very strong current	**C**.C. Tetanus	**A**.C.C″	**A**.O.C.′	**C**.O.C.

The contractions in health are abrupt and sharp. Except when the current is very strong, contractions only occur when the circuit is made or broken, not during the passage of a uniform current.

In **disease** the response to electrical stimulation may be altered either quantitatively or qualitatively. By "**quantitative alterations**" one understands that a given current produces a greater or less contraction than it could were the nerves and muscles in a normal state. "**Qualitative alterations**" involve either or both of two changes—namely, first, the character of the contraction, instead of being abrupt, becomes "sluggish"; second, the cathodal closing

* The cathode or negative electrode is attached to the zinc rod in the cell, the anode or positive electrode to the copper or carbon.

contraction is less readily elicited than the anodal contractions.

Such qualitative changes depend on the separation of the nerve and muscle from their nutritive centre. After a brief period the separation induces degeneration, and whilst the degeneration proceeds the nerve first fails to respond to electrical stimuli, and after a longer interval the muscle also becomes insusceptible.

It is during the time that elapses between the insensitiveness of the nerve and of the muscle that the most characteristic electrical changes are observed, and these are together known as **"the reaction of degeneration."** They may be summarized as follows :

(1) *Faradic current.*—No response can be elicited, even when a very strong current is employed.

The reason for this is that the faradic stimulus, in consequence of its brief duration, acts only on the nerve, and, since the degenerated nerve can no longer transmit stimuli to the muscle, the latter remains unaffected.

(2) *Galvanic current.* i. *Quantitative change.*—The muscular excitability is increased, contraction following the application of a weaker current than is necessary to produce it in health (the "irritability of weakness").

ii. *Qualitative changes.*—(*a*) The contraction is no longer sharp, but "sluggish."

(*b*) In most cases anodal closing contraction is elicited with a weaker current than cathodal closing contraction. This phenomenon is less constant and less important than the sluggish character of the contraction.

The quantitative change depends partly on alterations in the nutrition of the muscle, and partly on the removal of inhibitory influences ; the qualitative changes are produced partly because the nerve no longer regulates the character of the contraction, and partly also as a result of changes in the muscle itself.

The foregoing description applies to a fully developed reaction of degeneration. This is not manifested till more than a week after the trophic influence has been cut off. The nerves, however, begin to lose their sensitiveness about three days after the injury, and gradually become less and less responsive. The muscles behave as do the nerves to faradic stimulation. To the galvanic current they at first exhibit diminished excitability. After eight or ten days the excitability again increases, and eventually exceeds that found in health. At the same time as the increase appears, the sluggishness of contraction and the abnormal sensitiveness to anodal closure become manifest. When the cause which has led to the severance of the nerve and muscle from their trophic centre cannot be removed, the muscular response once more gradually diminishes, and after the lapse of a considerable period, which may extend to two years or even longer, disappears entirely. When the trophic influence is timeously re-established, the reactions of nerve and muscle progressively return to the normal. In such cases ordinary muscular power usually begins to return some days before the nerves show any response to electrical stimuli.

A clinically important form of reaction has been described by Ghilarducci, who has named it the reaction of degeneration at a distance (R.D.d.). In examining for this reaction neither pole is placed on the affected muscle, but the indifferent electrode is situated over the upper dorsal region, whilst the active one is applied at a point farther from the trunk than are the muscles to be tested. Thus, for the muscles of the forearm it is placed on the wrist, and for the tibialis anticus in front of the ankle. Under these conditions the response occurs almost exclusively at closure, and when the active electrode is negative; whilst the contraction thus elicited is stronger than that which is present in ordinary reaction of degeneration, and can be obtained with a weaker current. Its clinical importance is due to the fact that as it persists after all other electrical stimuli fail to produce any effect, its presence enables

one to determine that the muscle is not hopelessly destroyed, but may yet recover within certain limits. If it be absent the observer is justified in concluding that the muscle is wholly severed from its trophic centre.

Ghilarducci regards the phenomenon as due to the increased electrical capacity of the muscle when thus longitudinally stimulated, the increased capacity causing the electric wave to be less abrupt, and thereby prolonging the duration of efficient stimulation.

It will be readily perceived that, from the standpoint of diagnosis, electricity yields far more valuable information when the disease is situated in the lower motor neurone, thereby severing the nerve endings and muscles from their trophic centres, than when the morbid process occupies a more central position. Serious disorder may be present in the higher trophic realms without revealing itself by any change in the electrical reactions—at most there will only be a quantitative change whose detection is often difficult and whose value is uncertain.

In the sensory system, electro-diagnosis is of less value, and is chiefly of service in cases where an hysterical element is present.

For recording the results of an electro-diagnostic examination, one should employ a printed chart, in which the nerves and muscles are conveniently arranged with spaces opposite for the insertion of the observations. This both saves time and prevents the accidental omission of important muscles from examination.

CHAPTER X

EXAMINATION OF THE EYE, EAR, THROAT, AND NOSE*

I. THE EYE

NOTE first any obvious peculiarity about the eye. Observe whether there is any undue prominence of one or both eyes. Prominence of the eyes occurs in **exophthalmic goitre.** It is associated in that disease with the presence of what is known as *von Graefe's sign.* Ask the patient to look down. If von Graefe's sign is present, the upper lid seems to lag behind the eyeball in its descent, so that a large part of the upper portion of the sclerotic becomes visible. In paralysis of the cervical sympathetic the eyeball recedes so as to look more sunken than normal.

Note also whether the winking movements are increased or diminished in frequency. Infrequency of the movements along with an increased size of the palpebral fissure constitutes *Stellwag's sign* of exophthalmic goitre.

The occurrence of squint, ptosis, retraction of the upper lid, and alterations in the pupil has already been considered. The presence of any inflammation along the margins of the lids (marginal blepharitis) should always be noted. It is often an indication of a strumous constitution.

Next turn your attention to the **conjunctiva.** It may be necessary to examine the conjunctiva lining the

* In this chapter only those methods of investigation are described that are required in ordinary medical cases.

eyelids. In order to do this in the case of the lower lid, it is sufficient to depress the latter firmly with the thumb. To expose the inner surface of the upper lid, place the right thumb at the upper part of the upper lid and push upwards, so as to make the eyelashes stand out prominently. Grasp the lashes between the forefinger and thumb of the other hand, and evert the lid by rotating it round the thumb of the right hand.* Note the colour of the conjunctiva—whether it is pale, injected, or jaundiced. The method of detecting œdema of it has already been described.

Look next at the **cornea.** Note the presence of any ulceration or opacity of it. Small opacities are described as *nebulæ*, larger opacities are spoken of as *leucomata*. Try to make out whether the opacity is really on the surface of the cornea or deeper down in its substance. This can best be determined by looking along the surface of the cornea, as it were, and observing whether the light is reflected from it over the opaque spot, or whether it is dull. If the former be the case, the opacity is deep-seated; if the latter, it is superficial. Such superficial opacities point to

* The following simple method of everting the upper eyelid, so as to permit of an inspection of the conjunctiva, is described by Lang in his book on "The Methodical Examination of the Eye": "Whilst the patient, with head erect, turns his eyes down and looks towards the ground, the observer, who stands in front, places the end of his left forefinger on the right upper lid just above the tarsus, and his thumb on the lower lid just below the tarsus. By moving the forefinger a little upwards the margin of the upper lid is tilted forwards away from the globe; the thumb is now made to push the edge of the lower lid upwards into the space between the upper lid and the cornea. The lower lid thus acts the part of a wedge, and drives the upper lid forwards until its conjunctival surface is in contact with the thumb. When the whole thickness of the upper lid is between the finger and the thumb, the lower lid is released and the free edge of the tarsus is pushed upwards and backwards by the thumb whilst the forefinger presses its attached margin downwards and forwards; in this way the lid is everted. The right hand is employed to evert the left lid."

former strumous ulceration. Deep-seated opacities are often due to congenital syphilis.

The term *arcus senilis* is applied to a crescentic opacity which is sometimes observed towards the margin of the cornea. It usually appears at the upper part of the cornea first, and then gradually extends round. It occurs normally in old people, and is sometimes observed also in the eyes of younger persons who have sclerosed arterial walls and other signs of premature senility. True arcus senilis can be recognized by its leaving a small strip of clear cornea between the arc and the sclerotic; a crescentic opacity extending inwards from the sclerotic, which is sometimes met with, leaves no such clear strip. If arcus senilis be observed, its presence should always be noted.

It is often of importance in medical cases to be able to say whether a patient is suffering from **iritis** or merely from **conjunctivitis.** In each case the eye looks red and injected, but the characters of the injection are different in the two conditions. They are contrasted in the following table :—

Conjunctival Injection	*Ciliary Injection* (Iritis).
Colour, brick red.	Colour, pink.
Vessels very tortuous.	Vessels straight.
Vessels can be moved on sclerotic.	Vessels cannot be moved.
Injection greatest on lids and in cul-de-sac, diminishes round cornea.	Injection greatest round cornea, diminishes towards periphery.
Pressure on lid leaves no anæmia.	Pressure on lid leaves temporary anæmic spot.

In taking the case, note specially which of these sets of characters is present.

The **tension** of the eyeball should next be tested. This is done by placing the two forefingers on the upper part of the sclerotic outside the upper lid while the

patient looks downwards, the other fingers resting on his forehead. Then try for fluctuation. The normal tension must be learnt by practice, and any increase or diminution of it noted. An increased tension contra-indicates the use of atropine. Having observed these different points, one should next proceed to what is termed **oblique focal illumination** of the eyeball. For this purpose it is best to have the patient in a dark room, a lamp being placed in front of him. By means of a convex lens—the ophthalmoscope lens does very well—the light is focused on the surface of the eyes. If necessary, one may hold another lens in the left hand, and so magnify the view. Study in this way the surface of the cornea. The nature of the opacities already referred to can now be observed more minutely. Look at the iris. The outline of the pupil, its contractility to light, the existence of synechiæ and the presence or absence of hippus, can all be very well observed by this method. Note whether any opacity can be detected behind the pupil, and, if so, try to estimate the depth at which it is situated.

Then proceed to **retinoscopy.** Use for the purpose a plane or slightly concave mirror, with an aperture in the centre. An ordinary ophthalmoscope mirror does very well, but it is preferable to use one of larger size, say about 2 in. in diameter. The patient should be in a dark room with a light just above his head, and it is well to have his eyes atropinized. Sit fully a yard from him, and ask him to look far away over the top of your head. Then throw the light into his eye by means of the mirror. In a normal eye a red reflection from the retina will be seen through the pupil. If there be any opacity of the refractile media, the red reflection will be obscured. In this way cataract may be detected. When commencing, it usually takes the form of opaque bands passing in towards the

centre of the pupil like the spokes of a wheel. Now tilt the mirror, first upwards and downwards, then from side to side. As one does so, a black shadow is observed to flit across the pupil. Watch the edge of this shadow. From the direction in which it moves information is obtained as to the state of refraction of the eye. If the eye be emmetropic, or if it be hypermetropic, or has less than one dioptre of myopia, the edge of the shadow moves in the opposite direction to that in which the mirror is tilted if it be concave, but in the same direction to that in which it is tilted if the mirror be plane. In myopia of more than one dioptre the edge of the shadow moves in the same direction as a concave mirror, but in the opposite direction to one which is plane. In a normal eye the shadow moves very rapidly, and has a straight and sharply defined edge.* The more abnormal the patient's refraction, the more slowly does the shadow move, and the more crescentic and the less well defined is its margin.

If the edge of the shadow move differently in opposite meridians, the eye is **astigmatic.** If one meridian be normal the astigmatism is *simple,* and may either be of the myopic or of the hypermetropic variety, according to the nature of the refraction in the abnormal meridian. If both meridians be abnormal, the error of refraction being the same in kind in each, but unequal in degree, *compound astigmatism* is present. It may also be either of the myopic or of the hypermetropic variety, according to the nature of the refraction. If one meridian be myopic, the other hypermetropic, the condition is one of *mixed astigmatism.*

In *regular astigmatism* the directions of greatest

* The edge of the shadow is, of course, straight only when the source of light has a straight edge. This is so in an Argand burner, but not in an electric lamp.

and least refraction are at right angles to each other, and usually fall exactly in the vertical and horizontal meridians, the meridian of greatest curvature being most frequently the vertical. Sometimes, however, the directions of greatest and least refraction are in the oblique meridians.

In *irregular astigmatism* the directions of greatest and least refraction are not at right angles. This occurs comparatively rarely.

For the optical explanation of these facts, and for the more detailed description of their significance, the reader is referred to special works on ophthalmology. We would only remark here that retinoscopy affords the simplest and readiest method of arriving at an idea of the state of a patient's refraction.* In examining many medical cases such information is well worth obtaining, as errors of refraction have been found to be the reflex cause of many nervous symptoms, e.g. of headache, vomiting, etc.

The *fundus oculi* remains still to be examined, and for this one must have recourse to the use of the **ophthalmoscope.** Many patterns of this instrument are sold. The essential points are that there

* Sometimes a patient comes before one wearing glasses, and it may be important to know what their refraction is. In order to discover this, hold the glass in front of the eye and look at an object through it. Then move the glass from side to side, and watch the object. If the latter seems to move in the opposite direction to the glass, the latter is convex; if in the same direction, it is concave.

The strength of the glass may be approximately determined by bringing the small lenses of the ophthalmoscope behind it, until one finds that which abolishes the apparent movement of the object looked at.

In order to tell whether the glass is spherical or cylindrical (*see* foot-note on p. 530 for a definition of these terms), look at a straight object, e.g. a window bar, through the glass, and then slowly twist the latter round. If the glass is cylindrical the object looked at will appear to take up an oblique position. Patients who use cylindrical glasses are astigmatic.

should be two mirrors—a larger one for use in the indirect method of examination, and a smaller-angled one for direct examination of the fundus. It is also important to be able to bring a series of small lenses of different refraction behind the eye-hole in the mirror.

There are two methods of using the ophthalmoscope—the indirect and the direct. We shall describe the former first.

1. **Indirect ophthalmoscopy.**—It is best to have the room darkened; at any rate, bright sunlight should be excluded. If the patient be in bed, this may be effected by placing an umbrella over him. The patient (if he be able) sits in a chair with his head *slightly* inclined forwards, and the observer in another about 2 ft. from that of the patient and directly opposite to the latter. A light—an ordinary oil lamp, or, preferably, an Argand burner—is placed close to the patient and on a level with his left shoulder. The observer should sit rather obliquely, his left shoulder being turned well round towards the patient. The ophthalmoscope is held in the right hand, the larger mirror being opposite the eye-hole. If the observer be not emmetropic, he corrects his own error of refraction by means of the small lenses behind the mirror; light is then reflected into the patient's eye from the lamp. A red reflection from the retina should fill up the entire pupil unless there be any opacity of the media, as already mentioned. No details of the fundus should yet be visible. If any blood-vessels be seen, one can be sure that the patient's refraction is abnormal. If these seem to move in the same direction as the head of the observer, the patient is hypermetropic; if in the opposite direction, he is myopic. Having got the fundus fully illuminated, one proceeds to interpose the convex lens of the ophthalmoscope. Hold it between the finger and thumb of the left

hand, so that it rests opposite the joints between the first and second phalanges of the index. The finger should not be at all flexed. The forearm should be kept vertical, the hand drooping from the wrist, and the little finger supported on the patient's forehead. This is the position demanding the least muscular effort, and therefore the least fatiguing to the observer. The ordinary ophthalmoscopic lens is of about + 15 dioptres strength, and therefore requires to be held about $2\frac{1}{2}$ in. from the patient's eye.*

The exact point at which it should be held is arrived at by moving the lens backwards and forwards till a clear image is obtained.

A larger image of the fundus can be obtained by using a convex lens of about + 10 D (4 in. focus) and magnifying the image by means of a + 2 D lens placed behind the mirror. In many ways this is preferable to the ordinary method.

Having got the fundus into focus, one wishes to look at three parts of it—(1) at the optic disc, (2) at the macular region, (3) at the periphery. In order to bring the disc into view, the patient must be made to turn his eye somewhat inwards. If the left eye is being examined, ask him to look at the tip of your left ear; if the right is being examined, ask

* A lens of 1 dioptre (1 D) strength has a focal distance of 1 metre (40 in.); a lens of 2 D has a focal distance of 20 in.; and so on. To signify a convex lens the sign + is used, thus: + 2 D means a convex lens of 2 dioptres; − 2 D means a concave lens of 2 dioptres. A lens which is curved equally in every direction—i.e. which is part of a sphere—is called *spherical*. It may be either convex or concave. A concave spherical lens of 1 dioptre strength is indicated thus: − 1 D spher.

A *cylindrical* lens is part of a cylinder, and is therefore curved in one direction only. The direction corresponding to the axis of the cylinder is uncurved, and is spoken of as the axis of the lens. A convex cylindrical lens of 1 dioptre strength is written + 1 D cyl. As mentioned on p. 528, cylindrical lenses are required in astigmatism.

him to look at the tip of the little finger of the right hand, which is stretched out for the purpose beyond the handle of the ophthalmoscope.

In order to see the macular region, ask the patient to bring the eye slowly back from the above position towards the centre of your forehead. The macular region is reached at about two discs' breadth from the margin of the disc. Attempts to look straight at the macula in this way are sometimes baffled by the great contraction of the pupil which results, and by the reflection of light from the surface of the cornea. When this is the case it is better to ask the patient to look, not straight at the forehead, but a little to one side. The observer must then move his head till a view of the macular region is obtained.

The periphery of the fundus is seen by asking the patient to look first to his extreme right and left, then up to the ceiling, then downwards.

If, on gradually withdrawing the lens, the image of the fundus appears to become larger, there is *myopia* present; if it becomes smaller, there is *hypermetropia*. If it alters in one direction and not in others, there is *simple astigmatism;* if it alters in one direction more than others, the astigmatism is *compound*.

If, on moving the lens from side to side, one part of the fundus seems to move over the rest ("parallactic movement"), that part is at a higher level than its surroundings. Thus, if the disc be excavated, its margin will appear to move over the deeper part.

The examination of the fundus, and especially of its peripheral region, is greatly facilitated by a preliminary dilatation of the pupil. The best way of effecting this is to apply a 2 per cent. homatropine and cocaine ointment to the eye about half an hour before examining it. The effect may be afterwards

counteracted by instilling a drop of eserine solution (1 per cent.).

If the patient be unable to leave bed, indirect ophthalmoscopy may easily be carried out by the above method, provided he is able to be propped up. If that be impossible, place the lamp on the pillow above his head, and carry out the rest of the procedure as above.

2. **Direct ophthalmoscopy.**—The patient is placed as before, but with the light a little behind and above the shoulder corresponding to the eye under examination. The observer sits quite close to the patient, so that his eye can be advanced to within 2 in. of that of the latter. In examining the left eye of the patient, use your own left eye, and for his right use your right. Tilt the patient's head and your own in opposite directions, so as to avoid breathing one another's breath. Arrange the ophthalmoscope with the small oblique mirror opposite the eye-hole, and its surface directed towards the light. The apex of the wedge formed by the tilt of the mirror should be directed towards the root of the observer's nose, when the instrument is held flat against his cheek. If there be difficulty in getting proper illumination of the fundus, move the source of light about until the bright-red reflection is seen through the pupil. Ask the patient to look over your shoulder at a distant object, and try to relax your own accommodation entirely. This is the real point of difficulty in the direct method. As one is desirous of seeing clearly the fundus of the patient, and as that is so near, one almost instinctively accommodates his own eye for a near object. With practice, however, this difficulty can be overcome. It sometimes helps one to achieve the desired result if he tries to think in a dreamy way of some distant object, and to picture it to himself. If it be found impossible at first to relax

one's accommodation completely, it may be nullified by the use of a −2 or 3 D lens behind the mirror.

When the fundus has been brought into clear focus, its different regions must be systematically studied, just as in the use of the indirect method. In order to see the disc by the direct method, look backwards and inwards obliquely into the eye, telling the patient meanwhile to look straight in front of him.

In the case of a patient who is unable to rise from bed, the direct method may be applied in one of several ways.

(1) The observer kneels beside the bed at right angles to the patient, the light being placed on the pillow at the opposite side of the head of the latter. (2) The observer places himself at the top of the bed, so as to look down, as it were, on the patient's eyes, the light being placed on the opposite side of the head from the eye to be examined. (3) If the patient be a child, place him across the bed, and kneel at the patient's head, the light being held at the opposite side from the eye under examination.

The images furnished by the two methods of ophthalmoscopy differ. In the indirect method the image is inverted, so that what seems to be the upper part of the fundus is really the lower, and the inner (nasal) side appears to be the outer, and vice versa. The image, however, embraces a large part of the fundus at one time, so that one gets, as it were, a bird's-eye view of it. This method is, therefore, well suited for ordinary diagnostic purposes in medical cases.

The image obtained by the direct method is an upright image; consequently the different parts of the fundus are seen in their proper positions. The image embraces only a small part of the fundus at one time, but gives a magnified view of it. It is, therefore, well

suited for the minute study of pathological changes in the fundus.

The image obtained by the modification of the indirect method already described (i.e. by interposing a weak convex lens, and magnifying the image by a + 2 D lens behind the mirror) is intermediate between the images obtained by the ordinary indirect and by the direct method. It is an inverted image, pretty highly magnified, and shows also a fairly large part of the fundus at one time.

We have said that the optic disc, the macular region, and the periphery of the fundus must be studied in detail in each case. We may now pass to a consideration of the special points to be taken note of in each of these regions.

1. **The optic disc.**—Note—

(1) Its **shape.** Normally this is circular. Sometimes it is oval. If there be astigmatism present, the disc will appear to be oval, although it is really circular. This apparent oval shape may be distinguished from that which is real by moving the lens backwards and forwards. If the disc be really oval, it remains unaltered ; if it be only apparently oval, its shape will be found to vary with the position of the lens.

(2) Its **colour.** The normal disc is of a rosy tint, but distinctly paler than the rest of the fundus. The nasal side is normally rather redder than the other.

In atrophy of the optic nerve the disc becomes very pale, and may even be dead white or greyish in tint. In active hyperæmia of the disc its colour approaches in intensity to that of the rest of the fundus. Such active hyperæmia is often present in high degrees of hypermetropia. In passive hyperæmia of the disc the veins are alone affected, and the general tint is not altered.

(3) The presence or absence of a **physiological pit,**

and its size. Do not mistake the pallor produced by a very large pit for the pallor of optic atrophy.

(4) The character of the **vessels.** The arteries are normally distinguished from the veins by the following characters: They are only two-thirds to three-quarters the breadth of the veins, and they are not so dark in colour. They have a broader, better defined, and more continuous light stripe along their centres.

Normally, the arteries do not pulsate. They may be observed to do so in cases of aortic regurgitation, and in increased intraocular tension. The veins sometimes pulsate, even in normal eyes, owing either to the twisting of the arteries round them at points, or to high intraocular tension.

(5) The **edge of the disc.** It should be clear and well defined—especially at its outer side. As the vessels run across it, they should not be observed to tumble over at all. This "tumbling over," if present, is best evinced by the sudden disappearance of the central light stripe on the vessel.

(6) The **surroundings of the disc.** This part of the fundus should be carefully searched if one is on the outlook for the presence of hæmorrhages or tubercles, as both of these are more often encountered in the immediate neighbourhood of the disc than at other parts of the fundus.

Tubercles will be recognized as roundish, ill-defined, yellowish bodies, usually about half as large as the disc. *Hæmorrhages* occur as bright-red or dark-red blotches, with flame-like margins.

2. The **macular region** is situated, as we have said, about two discs' breadth from the outer edge of the disc. It is recognized by being rather darker in colour than the rest of the fundus, by being very devoid of blood-vessels, and frequently by being

surrounded with a halo of reflected light, producing a shot-silk appearance. The macula itself is in the centre of the region, and is rather pale in colour, and often glitters somewhat.

Changes in the macular region are important, in that they interfere more with vision than similar changes in any other part of the fundus. In cases of albuminuric retinitis, a circle of white spots may often be observed arranged around the macula (p. 539).

3. **Periphery.**—Inspection of the periphery of the retina is important, as it is here that some changes—such, for example, as disseminated choroiditis and retinitis pigmentosa—are the earliest to be detected.

The following is a brief description of the chief changes met with in the fundus which are of importance from a medical point of view :—

Optic neuritis.—In medical cases this is usually bilateral (double optic neuritis). It begins as a mere passive congestion of the disc, with slight œdema. At this stage the veins are fuller than normal ; on indirect examination the edge of the disc seems clear enough, but on closer inspection by the direct method it is seen to be slightly fluffy-looking. The change in the edge of the disc usually begins at its upper and lower margins. These parts should therefore always be most carefully inspected.

As the process progresses, it passes into true neuritis, or papillitis. The disc is now distinctly swollen. This is best recognized by the fact that, on direct examination, one requires (provided his accommodation be fully relaxed) the aid of a convex lens behind the mirror in order to bring the vessels on the disc clearly into focus. The veins are still larger than before, and distinctly tortuous. Pathological tortuosity of the veins occurs at right angles to the plane of the retina. Tortuosity in the same plane as the

retina may be quite normal. Often the veins can be observed to tumble, as it were, over the edge of the swollen disc. The arteries are smaller than normal, and may be partly obscured by the presence of exudation. The edge of the disc is no longer clear, even on indirect examination, but fades off into the surrounding retina. Small hæmorrhages may be observed on or near the disc.

It is often important to decide whether the papillitis is advancing or not. One ought not to form an opinion on this point unless he has already examined the disc on a previous occasion. The best criterion is the degree of swelling of the disc. In order to estimate this, use the direct method, and be sure that your own accommodation is thoroughly relaxed. Notice first whether the retina can be seen quite clearly without the aid of a lens. If the eye be emmetropic, one ought to be able to do so. If the refraction be abnormal, place behind the mirror the lens which is required to bring the vessels on the retina clearly into focus. Then look at the vessels on the disc. Owing to the swelling of the latter, the vessels are nearer the observer's eye than they should be, and a + lens must therefore be brought behind the mirror in order to enable one to focus them clearly. The strength of the lens required is the gauge of the amount of swelling which is present in the disc. Suppose, for example, that one requires to use + 1 D in order to focus the retina clearly (i.e. the patient has 1 D of hypermetropia), but that in order to focus the vessels on the disc one requires to make use of a + 6 D, then there is obviously + 5 D of swelling. Roughly speaking, every 3 D = 1 mm. of swelling. In this way one can estimate the amount of swelling from day to day, and so determine whether the neuritis is advancing or receding.

The above method, it is obvious, necessitates a

considerable amount of practice, and it is absolutely essential for success that the observer should be able thoroughly to relax his own accommodation.

Those who are unable to do this may use the following method instead of the above: Bring behind the mirror the weakest − or strongest + lens with which (*a*) the vessels on the fundus can still be clearly defined; do the same for (*b*) the vessels on the top of the disc. Then

$$\frac{b - a}{3} = \text{the height of the disc in millimetres.}$$

Optic neuritis is present in most cases of cerebral tumour at some period of the disease. It occurs also in about 50 per cent. of cases of tubercular meningitis, although it is often late in making its appearance. It is very uncommon to meet with it in the simple basal meningitis of infants, and in ordinary acute meningitis. It is also infrequent in cerebral abscess, and is not met with in cases of cerebral hæmorrhage, or thrombosis. On the other hand, it is not infrequent in other than intracranial diseases, and especially in Bright's disease (most commonly in the form which is associated with cirrhosis of the kidney and thickening of the blood-vessels). It is also met with in lead-poisoning, and in some cases of anæmia.

Optic atrophy.—We have already mentioned that the most striking change in the fundus in this condition is the pallor of the disc and the smallness of the arteries on it. The atrophy (*a*) may be primary (e.g. in locomotor ataxy)—this is sometimes called simple atrophy; or (*b*) it may be post-neuritic; or (*c*) it may follow degenerative changes in the retina. It is not always easy to say from a mere inspection of the fundus which variety it is that one has to deal with, and the longer the process has gone on the more difficult does the diagnosis become. Look at the edges of

the disc. If they be indistinct, especially if white streaks can be seen radiating from them and passing along the vessels into the retina, then one can be pretty sure that the atrophy is not primary but is postneuritic in origin. If the atrophy be secondary to changes in the retina the disc looks like a bit of dirty parchment, and pigmentary changes will be seen in the retina. In atrophy without previous optic neuritis the thinning of the nerve fibres renders very visible the fibres of the lamina cribrosa, and the disc acquires a "mottled" appearance. The presence of such mottling should always be carefully noted.

Albuminuric retinitis.—This condition of the fundus is met with in some cases of Bright's disease, especially in the very chronic forms. The changes consist in (1) the presence of optic neuritis with marked fullness of the veins; (2) the occurrence of hæmorrhages on or near the disc; (3) the development of white shining spots around the disc at a distance of about three discs' breadth from it, and of similar but much smaller spots arranged in a stellate form around the macular region. Any one of these sets of changes may be present without the others. These changes should be looked for in every case of chronic renal disease.

Tubercle of the choroid and **hæmorrhages in the retina** have already been mentioned, and their commonest sites pointed out. Tubercles may be looked for in cases of suspected acute miliary tuberculosis and tubercular meningitis. As they only appear very late in the affection, however, they are rather of pathological interest than of diagnostic importance.

Retinal hæmorrhages may be observed in any form of profound anæmia, but especially in pernicious anæmia and leucocythæmia. They also occur in purpura.

Embolism of the central artery of the retina may occur in cases of endocarditis. The appearance of the fundus is characteristic. Look at the macular region especially. There is present at it a peculiar round cherry-red spot. The disc itself is pale and its arteries are empty. The retina as a whole is somewhat milky-looking from the presence of œdema.

Disseminated choroiditis.—This is often an important sign of previous syphilis. The changes observed are the presence of small white patches of various shapes and sizes, and disturbance of the pigment around them. These can be recognized as being situated in the choroid by the fact that the retinal vessels pass over them. In congenital syphilitic choroiditis there is often a marked heaping-up of pigment around the patches, which are mostly circular in shape.

II. THE EAR

Examine first the **external ear.** Note any peculiarity of shape, observe the presence of any tophi, tumour, or swelling, or the existence of any skin eruption upon it. Observe whether there be any discharge from the meatus; if so, make a note of its character and odour. Note also whether there be any redness, tenderness, or swelling over the mastoid.

The **meatus** and **membrane** must next be inspected. Daylight is best for the purpose. Place the patient with the ear to be examined turned away from the light. Use a slightly concave mirror of about 3 in. diameter and a focal distance of 6 in., with an elliptical slit in the centre. In order to catch the light properly the surface of the mirror should be turned slightly upwards. By this means the external auditory meatus can be inspected. Note the presence of any foreign body, of an accumulation of wax, of eczematous eruption or furuncles, or any other abnor-

mality, as the presence of such may contra-indicate the use of the speculum.

Various forms of aspergillus may grow in the external auditory meatus and set up an otitis. The colour of the inspissated discharge depends upon that of the aspergillus, being black where *A. niger* is the fungus. The structure of the growth can be seen if some of the débris be mixed with a little liquor potassæ and glycerine and examined under the microscope.

In order to see the membrane a **speculum** must be employed. A metal speculum is best, unless one is wishing to make use of caustic applications, in which case it is better to use a vulcanite instrument. Choose a size of speculum appropriate to the ear under examination, warm it slightly, and introduce it so that the long diameter of its smaller end is placed almost vertically, but with a slight inclination from above downwards and forwards. Take care not to introduce it too far—not beyond the cartilaginous part of the meatus. There is, of course, no use in attempting to dilate the osseous part. Having introduced the speculum, hold it in position by means of the forefinger and thumb of one hand, while the pinna is grasped between the ring and middle finger of the same hand, the mirror being held in the other. The pinna should be pulled gently upwards and backwards, so as to straighten the meatus as much as possible. The mirror is held with its surface looking slightly upwards as before, and the membrane can then usually be seen on looking down the speculum. If the view be obstructed by the presence of impacted wax, the latter should be removed by being first softened with warm almond or olive oil, or soda solution (two teaspoonfuls of the bicarbonate to one pint), and then syringed out.

The student will find that it is not very easy at first to hold the speculum properly and at the same time

to pull the pinna upwards and backwards. It is therefore better, if one can, to have the mirror attached to the forehead by means of a spectacle frame. This leaves both hands free to manipulate the ear and speculum, or to make applications by means of the probe, etc.

If an ear mirror be not at hand, the large mirror of an ophthalmoscope can be made to serve the purpose. The lens of the ophthalmoscope can also be held close to the speculum, and so a magnified view of the membrane be obtained. This is especially serviceable in examining the ears of children.

A better magnified view of the membrane can be obtained by aid of the **auriscope.*** This instrument consists of a plated German silver cylinder, connected at one end with an ear speculum, and at the other with an adjustable magnifying lens. Light is admitted through a funnel-shaped tube in the side of the instrument, and is reflected on to the membrane by means of an oblique mirror. Artificial light and a bull's-eye should be used. The patient is seated with the light on a level with his ear, and the bull's-eye against the end of the funnel. The auricle is drawn upwards and backwards with the left hand and the speculum gently introduced with the right. It is best to use the largest speculum one can.

The first thing noticeable about the *normal membrane* (Fig. 136) is its bluish-grey colour and translucency. A small white knuckle-like prominence may be observed towards the middle of its upper part. That is the short process of the malleus. Passing downwards and backwards from it may be noticed the long process of the malleus, which ends in the umbo near the centre of the membrane. Passing forwards

* The best form is Hovell's modification of Brunton's auriscope, made by Messrs. Mayer & Meltzer.

and backwards from the short process of the malleus will be seen the anterior and posterior folds of the membrane. A triangular light portion of the membrane usually catches the eye, the apex of which meets the lower end of the handle of the malleus at an obtuse angle which opens forwards. This bright spot is due to the reflection of light. Its presence may usually be accepted as proof of a healthy state of the membrane. Immediately above the short process of the malleus a notch may be observed in the ring of bone which bounds the tympanic membrane. The part of the membrane which fills in this notch is called the membrana flaccida, or Shrapnell's membrane. It would be beyond the scope of this work to enter into a description of the various abnormal appearances which may be met with in the tympanic membrane. For the purpose of describing the situation of any abnormality which may be observed, it is customary to divide the membrane into an anterior part, which is in front of the handle of the malleus, and a posterior part, which lies behind it. Each of these is then divided by an imaginary line drawn through the tip of the handle into a superior and an inferior portion. Four quadrants are thus obtained, and in making notes one should say that a perforation (e.g.) is seen in the anterior superior quadrant, and so on.

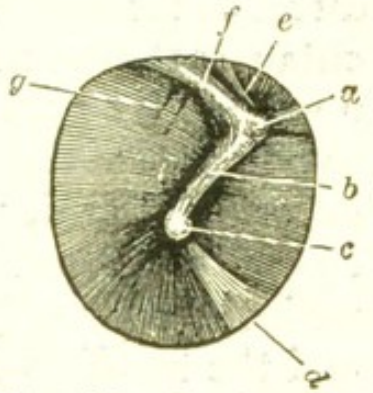


Fig. 136.—Outer aspect of right tympanic membrane—double the natural size. (*After Barr.*)

a, short process of malleus; *b*, handle of malleus; *c*, umbo; *d*, cone of light; *e*, membrana flaccida; *f*, posterior fold; *g*, long process of incus seen through the membrane.

Inflation of the middle ear.—It is sometimes desirable to inflate the middle ear with air. This is best effected by aid of a Politzer's bag. The bag should have a piece of rubber tubing, about $1\frac{1}{2}$ in. in

length, attached to its nozzle. Give the patient a mouthful of water, and bid him keep it in his mouth until he is told to swallow it. Introduce the rubber tubing into the lower part of one nostril, pinch firmly the other nostril and the upper part of the one into which the tubing has been introduced, and, holding the bag in the palm of the right hand, tell the patient to swallow. The moment the larynx rises, squeeze the bag firmly, and the air will enter the middle ear.

To ensure the entry of the air into the ear, or to direct it into one ear only, the **passage of the Eustachian catheter** may be necessary. Hold the instrument lightly near the broad end with the thumb and two fingers of the right hand. With the thumb of the other hand push the point of the patient's nose gently upwards. Pass the end of the catheter into the nostril with the curve of the instrument looking downwards, and the handle somewhat lower than the point. Pass it backwards along the floor of the inferior meatus, and as soon as the curved part has entered the nostril raise the handle of the instrument until it is level, and continue to push it backwards until it comes in contact with the posterior wall of the pharynx. Then rotate the instrument till its point is directed towards the middle line, and withdraw it until the curve hooks against the posterior end of the nasal septum. Now rotate the instrument, so that the point sweeps downwards and then upwards and outwards, the handle being kept pressed towards the nasal septum, and stop when the ring of the instrument is directed towards the outer canthus of the eye of the same side. The point of the instrument can then usually be felt to be arrested by the cartilaginous rim of the tube. The nozzle of a Politzer's bag may now be introduced into the outer end of the instrument, and the inflation accomplished.

If one end of a rubber tube, with an ear-piece at each extremity, be inserted into the ear of the patient, and the other end into that of the observer, the latter can hear the sound which the air makes as it impinges against the membrane. If a whistling sound be heard, it indicates the existence of a dry perforation. A bubbling sound shows the presence of fluid.

III. THE THROAT

The methods of examining the fauces and the pharynx have already been considered (p. 49). In order to obtain a view of the larynx, one must have recourse to laryngoscopy. In performing **laryngoscopy** the patient and observer should be seated opposite to one another in a darkened room, and about a foot apart. A light should be placed a little to the right (or left) of the patient's head and on a level with his mouth. An ordinary lamp will serve, but it is better to have an Argand burner, and it is also an advantage to have the light fitted with a bull's-eye condenser. The observer adjusts the reflector to his head by means of a forehead band or spectacle frame. If the former be used, the two knobs on the band should go against the root of the nose. It is then rotated on its ball-and-socket joint until the hole in the centre is directly opposite the right eye. This is ascertained by closing the left eye and observing whether one has a clear view through the aperture. One can also arrange the reflector so that it is in the centre of the forehead, and one then looks under its lower edge. This requires a little practice, but has the advantage of allowing one to make use of both eyes. It is also of advantage in the former method to have the aperture in the centre of the reflector in the form of an elongated slit rather than of a round hole, as a better view is thus obtained.

The observer should next manipulate the reflector with both hands until the light is directed on to the patient's mouth. He then selects a mirror, and warms it face downwards over the lamp until the moisture, which at first condenses on the surface, has all dried off. He should also touch his cheek with the back of the mirror before inserting it, in order to make sure that it is not too hot. Having warmed the mirror, he should hold it in such a way that it can be readily introduced and manipulated. On the whole, it is more convenient to hold the mirror like a pen than in any other way. It should also be held rather short, so that the hand of the observer can be steadied by resting the little finger on the patient's cheek.

The mirror being ready, the patient is told to crane out his neck a little, and to open his mouth and put out his tongue. The observer then throws a clean dry cloth over the anterior part of the latter, and grasps it firmly but gently between the forefinger and thumb of the left hand. It must be held firmly, but without any squeezing, and should then be, as it were, rolled out, as if round an imaginary axis situated near the hyoid. This manœuvre has the advantage of causing a better elevation of the epiglottis, whilst it prevents any risk of injuring the tongue against the lower incisor teeth. Before introducing the mirror, make sure that the light from the reflector is concentrated on the back of the patient's throat. This having been ascertained, the mirror should be introduced with its surface turned almost directly downwards, and passed rapidly back, care being taken to avoid touching either the tongue or the palate. The patient should be told to be sure to breathe regularly and through the nose. This serves to engross his attention. As the soft palate rises during an inspiration, the back of the mirror is placed gently against it, opposite the base of

the uvula. The soft palate is then gently pushed upwards and backwards, and the handle of the instrument lowered or raised until the back of the epiglottis comes into view. The patient is then told to say *Eh*, and that usually causes the vocal cords to become visible.

If the reflex excitability of the patient's pharynx be very great, so that any attempt to introduce the mirror induces retching, the application of a 10 per cent. solution of cocaine previous to beginning the examination will be found of great assistance.

It must be remembered also that one sometimes meets with a patient whose larynx baffles all attempts at inspection, owing to the position and shape of the epiglottis.

In studying the **view obtained,** the true cords usually first attract attention owing to their gleaming white colour. They should move freely on phonation. Tracing them forwards, they are seen to converge and disappear behind the cushion of the epiglottis. Posteriorly they diverge and terminate in knob-like prominences, which are the apices of the arytenoid cartilages. Immediately external to each of these is a smaller knob—the cartilage of Wrisberg. Passing backwards from each side of the epiglottis to the arytenoid cartilages are seen the ary-epiglottic folds. In favourable circumstances the observer can see down the trachea, and even as far as its bifurcation. This is facilitated by the observer placing himself at a lower level than the patient, and holding the mirror in such a way that its surface looks almost directly downwards.

In observing any abnormal condition of the larynx, the chief points to be attended to are—

1. The colour of the cords and mucous membrane.

2. The presence of any swelling or ulceration.
3. The mobility of the vocal cords.

1. The normal colour of the vocal cords is a pearly white. In laryngitis they become red. Any increase or diminution in the redness of the laryngeal mucous membrane should be noted. In tubercular affections the mucous membrane is abnormally pale; in syphilitic conditions it is unusually red.

2. The position and character of the swelling should be noted. Tumefaction of the ary-epiglottic folds and of the epiglottis should be looked for in suspected laryngeal phthisis. Tumours of various sorts on the true cords are occasionally met with. As regards ulcers, note their number, their position, and the character of their floor. Tubercular ulcers are usually multiple, and are met with very frequently on the interarytenoid fold. Syphilitic ulcers are usually single, and have a yellow, sloughy floor.

3. Observe whether the cords come together normally on phonation and open widely during inspiration. In **adductor paralysis** the affected cord fails to move inwards on phonation, or the cord makes a sudden movement inwards and then goes back, the position being unsustained (Fig. 137—*2*, *3*). In **abductor paralysis** the cord looks normal on phonation, but fails to move outwards again on inspiration (Fig. 137—*4*, *5*).

In paralysis of both abductors and adductors (paralysis of the whole recurrent laryngeal nerve, or **recurrent paralysis**) the cord is fixed in the cadaveric position—i.e. midway between complete adduction and abduction. This is much more common on the left than on the right side, owing to the greater liability of the left recurrent laryngeal nerve to be pressed upon by aneurysms (Fig. 137—*6*, *7*, *8*).

Adductor paralysis is usually the result of func-

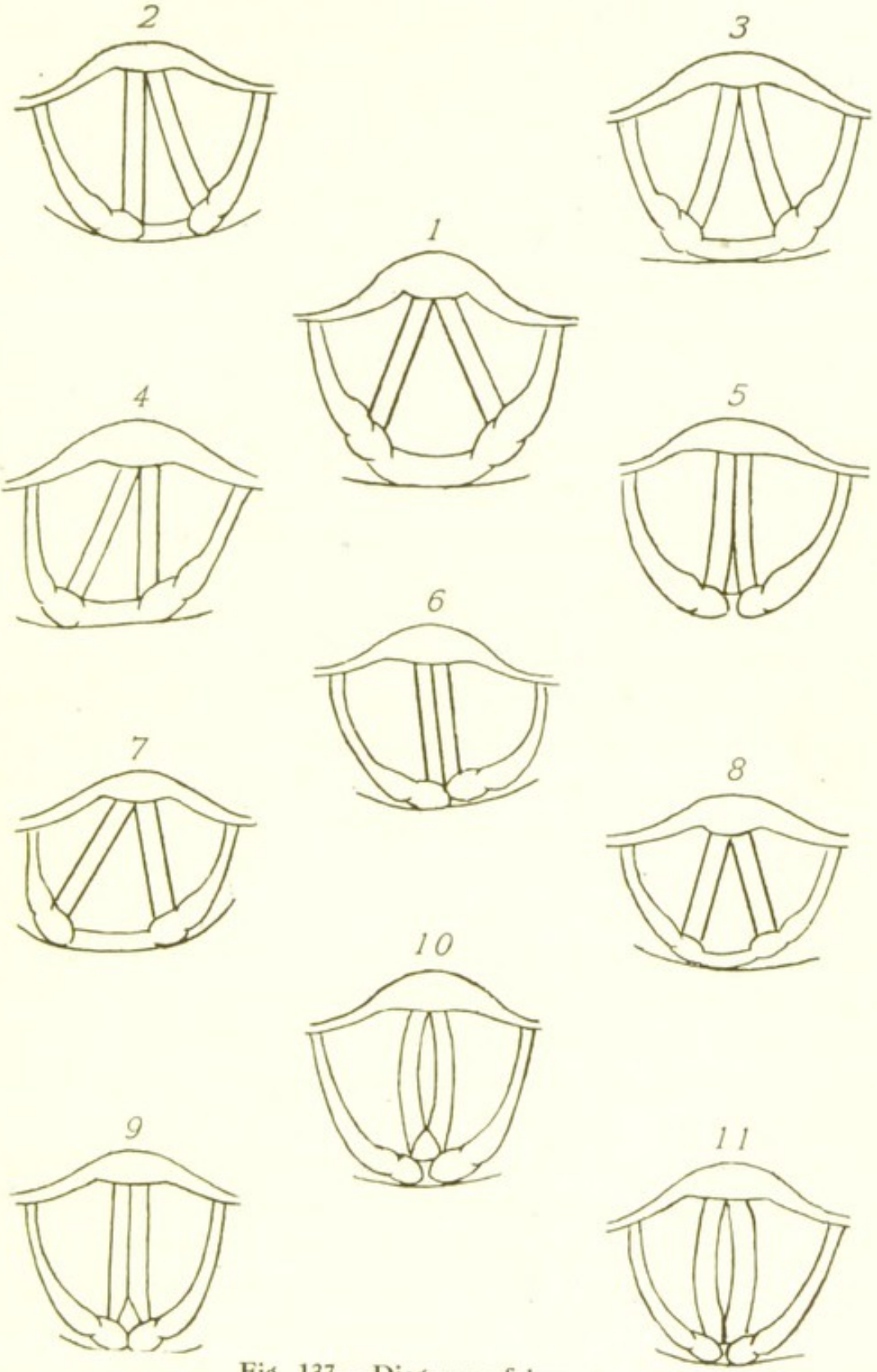


Fig. 137.—Diagram of larynx.

1, normal larynx, respiration. PARALYSES: 2, adductor, left, phonation; adductor, right and left, phonation; 4, abductor, left, respiration; 5, abductor, right and left, respiration; 6, recurrent, left, phonation; 7, recurrent, left, respiration; 8, recurrent, right and left, respiration and phonation; 9, arytenoid, phonation; 10, arytenoid and thyro-arytenoids, right and left, phonation; 11, thyro-arytenoids, right and left, phonation. All drawn as seen in the mirror, i.e. patient's left hand to observer's right.

tional disease. Abductor paralysis, on the other hand, is the form of paralysis characteristic of an organic lesion of the nervous system. Bilateral adductor paralysis or paresis is the cause of the condition known as hysterical aphonia.

If on phonation the cords come together incompletely, leaving an elliptical space betewen them, there is **paralysis of the internal thyro-arytenoids** present (Fig. 137—*11*). If the anterior two-thirds of the cords come together, but leave a triangular cleft behind, the **interarytenoid muscle** is affected (Fig. 137—*9*). For further details regarding these forms of paralysis special works must be consulted.

IV. THE NOSE

Anterior rhinoscopy. — The position of the patient and of the observer, and the arrangement of the light and reflector, are the same as for laryngoscopy.

The anterior nares should first be inspected without the aid of a speculum. Tilt the tip of the nose upwards with the finger, and note the presence of any eczematous or ulcerated condition of the mucous membrane or skin. Observe whether any dried secretion or blood can be seen. Look for any swelling, ulceration, or perforation of the cartilaginous part of the septum.

Having noted these points, take a solid two-bladed speculum, warm, and introduce it. Hold it in position with the left hand, and gently screw the blades apart with the right. The first object to be observed is usually the anterior end of the inferior turbinated body. Note whether it is larger than normal, or not. If it be enlarged, touch it with a probe, so as to ascertain whether the enlargement be osseous or due to swelling of the mucous membrane merely. Then

depress the patient's chin somewhat, so as to bring the inferior meatus into view, and ask him to hold his head a little back, so as to obtain a view of the middle meatus and middle turbinated body. The latter is considerably lighter in colour than the inferior turbinated. The superior meatus can never be seen, and the superior turbinated only very rarely.

The presence of polypi should be specially looked for in these parts. Their recognition is facilitated by the use of the probe. Lastly, turn the patient's head a little, so that the septum can be inspected. Note any deviation of it, or the presence of any prominence or spine, or the existence of any ulceration or perforation.

Swelling of the inferior turbinated body sometimes obstructs the view of the rest of the nasal cavity. The application of a little 10 per cent. cocaine on a pledget of wool will usually cause the swelling to disappear.

Posterior rhinoscopy.—This is the only method of obtaining a view of the posterior nares. In carrying it out, the position of the observer, the patient, the reflector, and the light should be the same as for laryngoscopy. The patient, however, should have the chin rather depressed, the neck not being craned out as in the examination of the larynx.

Select the smallest laryngeal mirror, warm it, and ask the patient to open his mouth. It is sometimes an advantage to have the shaft of the mirror bent back a little about $1\frac{1}{2}$ in. above the reflecting surface. Introduce a right-angled tongue-depressor, and hold down the tongue with the left hand. Take the mirror in the right hand, and pass it in with the surface looking upwards. Introduce it behind the soft palate, passing along between the uvula and the left anterior pillar of the fauces. Now turn the mirror a little, so that its surface looks upwards and forwards. The posterior nares will then come into view. What one

usually sees first is the posterior end of the nasal septum. It looks narrow, sharp, and pale in colour below, but expands a little and looks reddish above; a slight cushion-like swelling can also be often seen about the middle of it on each side (Fig. 138).

On the outer wall the posterior end of the middle turbinated bone can usually be easily seen as a large bluish-red swelling. Above it, one can see the superior

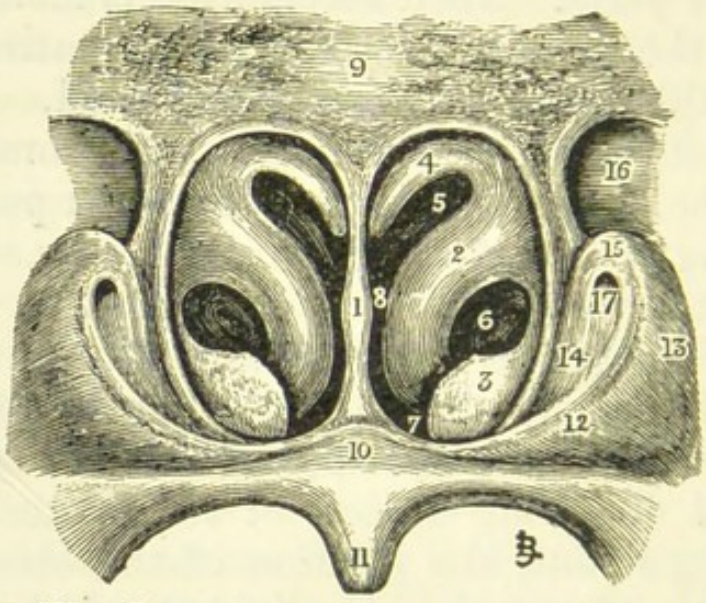


Fig. 138.—Posterior nares and surrounding parts.

1, septum; 2, middle turbinated bone; 3, inferior turbinated bone; 4, superior turbinated bone; 5, superior meatus; 6, middle meatus; 7, inferior meatus; 8, main passage of nostrils; 9, vault of pharynx; 10, cushion of soft palate; 11, posterior surface of uvula; 12, ridge formed by levator palati; 13, salpingo-pharyngeal fold; 14, salpingo-palatine fold; 15, Eustachian cushion; 16, fossa of Rosenmüller; 17, Eustachian orifice.

meatus and the end of the superior turbinated bone; below it are the middle meatus and the upper part of the inferior turbinate. Observe the presence of any increase in size of any of these objects. Note also the general character and colour of the mucous membrane, or whether any mucus or pus can be seen adhering to it. Next turn the mirror a little upwards and to one side, keeping it rather low down and with its back against the tonsil, and look for the cushion of the opposite Eustachian tube, which can usually be made out. It forms a bright-red rounded projection,

bounding a depression which leads to the orifice of the Eustachian tube. Observe whether there be any secretion at the mouth of the latter, or any adenoid swelling of the mucous membrane.

Lastly, turn the surface of the mirror upwards, and examine the vault of the naso-pharynx, noting especially the presence of any adenoid swelling or tumour in that region. Normally the roof should present an appearance not unlike that of the surface of the tonsil (Luschka's, or the pharyngeal tonsil).

Posterior rhinoscopy is often very difficult to perform. The difficulty may be due to there being very little room between the posterior wall of the pharynx and the soft palate. If this cannot be overcome by inducing the patient to breathe quietly through the nose, or to sniff, it may be necessary to introduce a palate retractor, but for a description of this process special works must be consulted. Even after the mirror is properly introduced, it is not always easy to recognize what one sees. This is largely due to the fact that only a small portion is seen at one time, and the mirror has to be turned about till every part has been viewed separately. Experience alone can overcome these difficulties.

CHAPTER XI

LOCOMOTORY SYSTEM (BONES, JOINTS, GAIT)

The locomotory system includes the muscles, bones, and joints. The examination of the muscles is most conveniently considered along with that of the nervous system (Chap. IX.). There remain for consideration the bones and joints.

Bones and Joints

In examining the long bones of the limbs, look (1) at the condition of the shaft, (2) at the articular ends.

As regards the **shaft,** note any distension or bending of the bone and any signs of a former fracture. Pass the hand along the bone, noting the presence of any tenderness or thickening of it. Thickening is most likely to be detected on the exposed surfaces of bones, e.g. over the anterior surface of the tibia and at the lower ends of the radius and ulna. Such thickening often affords valuable evidence of old periostitis, especially of the syphilitic form.

As regards the **ends of the bones,** note the presence of any general enlargement, such as occurs in rickets, or of any nodulation at the margins, such as one finds in rheumatoid arthritis.

In examining a **joint,** note first the points which can be made out by simple inspection. Observe the position in which the patient keeps the joint; note any alterations in its contour, such as local or general swelling, and the presence or absence of any redness. Then pass to palpation, noting whether or not there

be any increase of local heat in the joint, whether it is tender to the touch, and whether one can make out the presence of any fluctuation in the joint cavity. Then try to move the joint, observing the degree of mobility in each direction, and whether or not attempts at movement produce pain. If the joint be movable, note whether any sensation of grating is produced on movement. If the mobility be limited in one or every direction, try to form a conclusion as to the cause of the limitation, and especially whether it be due to changes in the components of the joint itself, e.g. contraction of ligaments, or fibrous or bony ankylosis, or whether it be due to changes in the structures surrounding the joint, e.g. shortening of tendons. Next turn your attention to the synovial membrane. Try to make out whether there is any thickening or bogginess of it. Lastly, examine the articular surfaces of the bones, moving the joint (if possible) so that the edges of the articular surfaces can be examined with the fingers. Note the existence of any irregularity or thickening of these, and the presence of any outgrowth or "lipping" of them.

The **vertebral column and skull** demand special attention. Observe in the former the presence of any local projection of the vertebral spines. If such there be, state which are the vertebræ involved, and at which the projection is most prominent. In counting the vertebræ for this or any other purpose, one can take as landmarks either the spine of the vertebra prominens or the last rib, tracing the latter back to the 12th dorsal vertebra.

In many cases, however, the last rib cannot be distinctly felt. It is therefore rather untrustworthy as a guide.

Note also the presence of any curvature of the spinal column as a whole, or of one part of it,

distinguishing carefully such general curvature from the local projections above referred to.

The curvature may be in an anterior or a posterior direction, or laterally. Anterior curvature is termed *lordosis*, and is commonest in the lumbar region. General posterior curvature is spoken of as *kyphosis*. It occurs most typically in the dorsal region in old persons, and must be distinguished from the localized angular curvature of spinal caries. Lateral curvature is termed *scoliosis*, and may be towards either the right or the left side. It is always accompanied by a rotation of the bodies of the vertebræ in such a way that the spines come to point towards the concavity of the curve. An absence of the normal curves of the spine may be an indication of commencing vertebral disease.

Ask the patient to stoop down, and notice the degree of mobility of the vertebral column, and the occurrence of any pain during stooping, noting the exact site of the latter if present. Then pass the hand down the vertebral column, and observe whether any tender spots can be made out. Such tender spots are not infrequently met with in hysteria and in cases of irritation of the posterior nerve roots. Their presence can often be more easily elicited by drawing a sponge wrung out of hot water down the vertebral column : the patient complains of pain whenever the hyperæsthetic area is reached. To elicit more deep-seated tenderness of the vertebræ, it may be necessary to "punch" the spines gently with the fist from above downwards, observing the point at which the patient complains of pain, and verifying the observation by repeating the process from below upwards.*

* A better method of eliciting deep-seated tenderness of the spine (e.g. in early caries) is to press upon the top of the patient's head or upon the shoulders whilst he is in the erect position.

In studying the **skull,** note first its **size.** For this purpose it may be necessary to take **measurements.** This should be done in three directions: (1) antero-posteriorly from the root of the nose to the external occipital protuberance; (2) circumferenti lly at the level of a line drawn horizontally round the skull from the supraorbital ridges in front to the external occipital protuberance behind; (3) coronally from one auditory meatus to the other. If the skull be abnormally small, the patient is microcephalic. This is frequent in some forms of idiocy. Abnormal enlargements of the skull occur in hydrocephalus, in osteitis deformans, and in acromegaly.*

Next observe the **shape** of the skull. Is it of the dolichocephalic (long-headed), or the brachycephalic (bullet-headed) type? Are the two sides of the head symmetrical? Certain well-recognized types of abnormal skull are met with. In *hydrocephalus* the skull tends to assume a globular form. The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed. The lateral aspects of the skull (above the ears) project outwards. If the patient be a child, as is usually the case, the fontanelle is wide and bulging, and often fluctuates very distinctly. The sutures may be opened up, and imperfectly ossified areas (craniotabes) may be detected in the bones. In *rickets* the skull tends to be square or oblong and box-shaped. The frontal and parietal bones often show central thickening ("bossing"). The forehead, however, does not overhang, nor are the eyes depressed, and although the fontanelle

* The average normal circumference of the skull at different ages is as follows: —

Birth	13 inches.	Seven years ..	20 inches.
Six months ..	16 ,,	Fifteen ,, ..	21 ,,
One year	18 ,,	Adult	22 ,,
Three years ..	19 ,,		

is usually widely open, it does not bulge as it does in hydrocephalus, nor are the sutures opened up. In *congenital syphilis* the forehead is vertical, the frontal eminences are often exaggerated, and the bridge of the nose is depressed.

Having noted the general shape of the skull, ask the patient to open his mouth, so that one may see the **hard palate.** Observe its width and degree of arching. A high, much-arched, and narrow palate is often one of the minor signs of mental deficiency.

Next proceed to the **palpation of the skull.** Note first the thickness of the scalp, and whether it moves freely, as it ought, upon the subjacent bone. Atrophy and adherence of the scalp are apt to be associated with skin diseases in this region, and are often a bar to successful treatment. Observe the presence or absence of inequalities in the bones, such as may indicate the site of former injury or fracture. If a swelling be detected, pay special attention to its margins, noting whether a hard rim can be made out, and whether or not the rim disappears on firm pressure steadily applied by the finger for a minute or two. In blood extravasations the rim disappears, in a depressed fracture it persists. Note also whether the swelling can be moved as a whole upon the skull, or not. If the patient be a child, note the condition of the fontanelles and sutures, and look for the presence of unossified areas in the bones (craniotabes). The best place to look for these is in the neighbourhood of the lambdoidal suture. They feel like little spots which are covered only by parchment. Observe the presence of any tender spots or areas on the skull. For this purpose it may be necessary to tap the skull gently all over with the forefinger. If tenderness be detected, note carefully its maximum point. Such tender points

are sometimes met with in inflammatory affections of the cranial bones or membranes, and in cases of superficially situated intracranial tumours, but they may also be present in neuralgic affections of the scalp.

The Gait

The character of a patient's gait is often an important indication of the nature of the affection from which he is suffering. It is specially important in cases of nervous disease.

In studying the gait, it is well, if possible, to have the legs fully exposed. For this purpose the patient should have on only a night-shirt or dressing-gown, which should be brought through between the legs from behind, and pinned up in front. The feet should be bare. The patient is told to walk away from the observer, to turn round at a given point, and then to come towards him again.

If it be desirable to obtain a permanent record of the patient's footprints, one can have recourse to photography. Put on the feet a pair of woollen socks, and dip them in rather thick whiting. Ask the patient to walk along a smooth floor. The marks left by the feet can then be photographed.

In studying the gait, the **points to be noted** are—(1) Can the patient walk at all? This being decided, one has to ask oneself—(2) Does he pursue a straight line, or does he tend to deviate to one side or the other, or to both alternately? To bring out this point, it is well to ask him to walk along a straight line—e.g. a crack in the floor. (3) Does he tend to fall, and, if so, in what direction? These questions being settled, the next point to be decided is whether the gait conforms to any of the well-recognized abnormal types. Before one tries to make up one's mind in this matter, however, it is well to be quite

sure that the peculiarity in the patient's gait is not due to some surgical cause or to local disease of a joint—e.g. rheumatoid arthritis of the hip. For example, we have known the peculiar gait which is affected by patients with congenital double dislocation of the hip to be mistaken for the result of weakness of the muscles of the back, and treated by massage and electricity. A previous examination of the bones and joints will eliminate such sources of fallacy.

The three chief types of abnormal gait due to nervous affections are—

1. The spastic.
2. The ataxic.
3. The reeling.

In taking a patient's case, it is usually sufficient to state that the gait belongs to one or other of these types, or is a combination of one or more of them. The chief peculiarities of each variety are as follows :—

1. The **spastic** may be described as a "sticky" gait. The patient has difficulty in bending his knees, and drags his feet along as if they were glued to the floor, the toes scraping the ground at each step. The foot is raised from the ground by tilting the pelvis, and the leg is then swung forwards so that the foot tends to describe an arc.

This gait is seen most characteristically in patients with lateral sclerosis of the cord. The **hemiplegic gait** is essentially a spastic gait in which only one leg is affected.

2. The **ataxic** may be described as a "*stamping*" gait. The patient raises his feet very suddenly, often abnormally high, and then jerks them forward, bringing them to the ground again with a stamp, and often heel first. He seems to exhibit, also, an indefiniteness of purpose in the place chosen to put down the foot;

and the feet while in the air do not move in one plane, but are waved about, as it were, before being set down. By adopting a "broad base" the patient tries to counteract the unsteadying effects of his style of progression. This gait is best seen in cases of locomotor ataxy.

3. The **reeling** gait may be described as a "*drunken*" gait, and, therefore, requires no further description. It will be observed that patients with this gait walk "on a broad base," the feet being planted widely apart. It is important to notice whether supporting the patient by his axillæ abolishes the reeling tendency. In some cases of cerebellar disease, such support has been observed to abolish the patient's vertigo for the time during which he is supported.

This gait occurs most typically in cases of cerebellar lesion. It is, therefore, sometimes referred to as a "cerebellar gait."

Some rarer varieties of abnormal gait may be briefly referred to. These are—

The **"festinant gait."** This is the form of gait met with in typical cases of paralysis agitans. The patient is bent forwards, and advances with rapid short shuffling steps, so that, as has been said, "he looks as if he were trying to catch his centre of gravity." In some cases, if one suddenly pulls the patient backwards, he begins to walk backwards, and is unable to stop himself, though he is leaning forwards all the time. This peculiar phenomenon is spoken of as "*retropulsion.*"

The **waddling** or oscillating gait is like the gait of a duck. The body is usually tilted backwards, there being a degree of lordosis present; the feet are planted rather widely apart; and the body sways more or less from side to side as each step is taken. The heels and

the toes tend to be brought down simultaneously. The chief peculiarities of this gait are due to a difficulty in maintaining the centre of gravity of the body owing to weakness of the muscles of the back. It is met with in pseudo-hypertrophic paralysis and in congenital dislocation of the hip.

The **high-stepping** or prancing gait is a device adopted by the patient to avoid tripping from his toes catching the ground. It is, therefore, met with in cases where the toes tend to droop from weakness of the extensor muscles, e.g. in peripheral neuritis affecting the anterior tibial nerve. The name sufficiently describes its characters.

CHAPTER XII

CLINICAL EXAMINATION OF CHILDREN

The clinical examination of young children is a matter full of difficulty to the inexperienced, for it has to be carried out not merely without the help of the patient, but often in spite of his strenuous opposition. In this chapter we propose to point out the best methods of ascertaining the necessary facts, and also the chief points in which the child differs from the adult in a clinical sense.

The history of the patient and his illness must, of course, in the case of young children, be ascertained from the mother or friends. The best **scheme of interrogation** to employ will be found on p. 11. Whilst the history is being elicited, opportunity may be taken to cultivate the friendship of the child or, at all events, to get him accustomed to one's presence. The history having been ascertained, one proceeds to an examination of the child. This requires gentleness and deliberation, combined with infinite patience and good temper. If one is at all hurried or rough, the child begins to cry at once, and the subsequent examination is rendered a thousandfold more difficult. We would emphasize the fact that it is almost impossible to be really systematic in one's examination of children. Certain things must always be looked for, but no definite order can be observed in looking for them. One has to seize the opportunity of ascertaining a fact as it presents itself, and a rigorous adherence to "systems" is often out of the question. In the first place, a number of points can be ascertained before

the child is undressed. One can study the **facies** of the child, note its complexion, the colour of its lips, and whether or not the alæ nasi are acting. One should also at this period of the examination count the respiration and pulse-rate; it is very important to get these noted while the child is still undisturbed.

The **respirations** can usually be counted by merely watching the movements of the child's abdomen, that being very much more affected by respiration in young children than is the chest. The normal rate of a new-born child is 40 or so respirations per minute, by the second year they have fallen to 30 or so, at the fifth year they are about 25, and by the fifteenth they have sunk to 20. Much more important than the absolute number of respirations is the ratio of respiration to pulse. Normally this should be as 1 : $3\frac{1}{2}$ or 4.

The **pulse** is best counted by allowing the mother to hold the child's hand in hers; the fingers of the physician are then quietly slipped over the mother's hand on to the child's wrist, and the pulse counted. If the child has begun to cry, it is useless to take the pulse-rate, as it may be at least 20 beats above the normal rate. The pulse-rate at birth should be 130, by the second year it is 110, by the fifth 100, by the eighth 90, and by the twelfth 80; after this it gradually sinks to the normal adult rate. During sleep the pulse-rate always falls about 10–20 beats. As a matter of fact, the examination of the pulse in infancy is of comparatively little clinical value. It is of little use as an index of the vital powers, the fontanelle replacing it in that respect. The vessel being extremely small, the characters of the pulse-wave can hardly be ascertained; irregularity by itself is of comparatively little significance, being very common, even

in healthy infants, and being almost the rule in sleep. A pulse which is continuously *slow* and irregular is, however, of great significance.

These preliminary facts having been noted, the child should be stripped and placed in a blanket on the knee of the mother or nurse; examination must then be proceeded with by the usual methods of inspection, palpation, auscultation, and percussion. In the clinical investigation of children it must be noted that the two former methods are of much the greatest assistance.

It is well to begin by looking and feeling the child all over. One notes the general state of development and nutrition; the state of the skin, whether dry and fevered, or moist, and the presence or absence of any rash or skin eruptions, and whether or not the normal degree of elasticity is present. The shape of the chest and the degree of prominence of the abdomen should be noted, it being borne in mind that the rickety and pigeon-breasted types of chest are very common in diseased children, and that a rather protuberant abdomen is to be regarded as normal. The hand should then be lightly passed over the head. The state of the **anterior fontanelle** should first be investigated. The fontanelle closes normally between the fifteenth month and the second year. If it remain patent after the second year, it is often a sign of disease—usually of rickets. Too early closure of the fontanelle occurs in some forms of microcephaly and idiocy.

The degree of tension of the fontanelle is of great importance. In health it pulsates distinctly, and is neither sunken nor unduly elevated. A depressed fontanelle is an important sign of exhaustion; a tense fontanelle indicates increased intracranial pressure. It must be borne in mind, of course, that the

fontanelle is normally tense when the child is crying. The systolic bruit heard over the fontanelle is of no clinical importance.

The **shape of the head** and of its bones must be investigated. The development of "**bosses**" ("Parrot's nodes") on the frontal and parietal bones is a common occurrence in rickets, especially in syphilitic children. One should also look for evidence of craniotabes (in young babes) and of rheumatic nodules (in older children). The general shape of the head as a whole should always be noted; it may be box-shaped as in rickets, or globular as in hydrocephalus; it may be abnormally small or large, or it may be asymmetrical.*

Passing from the head, one may examine the **long bones.** In children this is of extreme importance; many of the commonest and most serious diseases of infancy affect the bones more prominently than any other part of the body. Look for thickening or tenderness along the shafts of the bones. This may be due to scurvy, to syphilitic or to suppurative periostitis, or to tumours. Examine carefully the **epiphyses.** In rickets these become enlarged. This is most easily seen where the ribs join their cartilages, the thickening there forming a row of bead-like prominences ("*rickety rosary*"). It is also easily seen at the wrists. The frequency of inflammatory affections of the epiphyses should also be borne in mind. The presence or absence of "*rheumatic nodules*" should also be noted. These are little fibrous bodies varying in size from that of a large pin's head to a pea, or even bigger. They occur not in the periosteum, but in the deep fascia where it covers superficial bones, and also in the sheaths of tendons. They should be looked for especially over the olecranon and patella. They are usually

* For the normal circumference of the skull, *see* foot-note to p.557.

movable, but not tender. If found, they are pathognomonic of rheumatism. The **vertebral column** should always be examined for signs of tubercular disease or curvature.

At this point, if not earlier, the child's **temperature** should be taken. In young children the thermometer should be inserted into the rectum, or placed in the groin or axilla; in older children it may be placed in the mouth. It should be remembered that the temperature in children is much more variable than in adults, and that it often rises on very little provocation.

One must now proceed to the examination of the **thorax and abdomen.** The front of the chest and abdomen may be examined together, and either after or before the posterior aspect of the chest. The order adopted should be, first, inspection and palpation, then auscultation, and, last of all, percussion. Percussion is left to the last owing to the fact that it frequently makes the child cry.

In **palpation,** be sure that the hand is quite warm; this is even more important in examining a child than in the case of an adult. In **auscultation** one should use either the immediate method—the ear being applied to the skin directly (this is only applicable in examining the back)—or else one should use a binaural stethoscope.* The latter enables one to follow slight movements on the part of the child better than one can with a wooden instrument. If the chest-piece of the stethoscope be made of metal, remember to warm it before applying it to the chest. There is only one point to be observed in the **percussion** of a child, and that is, that the stroke should be *light.*

* The chest-piece of the stethoscope should be short, for when the child is on his mother's knee there is often but little space between him and her.

This is not merely in order to avoid frightening the little patient, but also to escape the confusion which is apt to arise from the excessive resonance of the child's chest.

When the abdomen and front of the chest have been run over in this way, one should turn his attention to the posterior aspect of the lungs. For the examination of these, the child should not be laid on his face, as that interferes with respiration, and causes the abdominal viscera to push up the diaphragm, but he should be held against the mother's breast with his head looking over her shoulder. In this way the whole of the back of the chest can be gone over.

Last, but by no means least, comes the examination of the **mouth and throat.** It is impossible to exaggerate the importance of systematically examining the mouth and throat in all cases of illness in children. At the same time, it is just this part of the clinical examination in which we are most likely to meet with opposition; and for that reason it is left to the last, as it may be necessary to employ coercion in order to get it carried out.

Begin by looking at the **tongue.** Sometimes the child will put out the tongue when asked. In little babies gentle pressure on the chin will often cause the mouth to be opened, when a view of the tongue can be obtained. Or, if a drop of milk or a little sugar be placed just outside the lip, the child will often put out its tongue in order to lick it off. In more refractory children it may be necessary to push the lower lip over the teeth, and then to press the lip down against the lower incisors. The child then opens the mouth in order to avoid having the lip cut. With very obstinate children one may be obliged to compress the nostrils until the mouth is opened to get breath.

Once the child has been induced, either voluntarily or by aid of one of the above devices, to open the mouth, one should note the state of the **buccal mucous membrane,** remembering the frequency of thrush, stomatitis, and ulcerations in children. In cases of suspected measles, *Koplik's spots* should be carefully looked for. They consist of irregularly stellate or round rose-red spots, with a bluish-white speck in the centre of each. They are to be found on the inside of the lips and on the buccal mucous membrane, especially opposite the upper molars. At first they are very sparse, but later on become more numerous, and the red parts may then coalesce into large areas dotted with the bluish-white specks. They should always be looked for in strong sunlight if possible, and never by artificial light. They are of considerable diagnostic importance, for they may precede the appearance of the skin eruption by three or even five days. The number and character of the **teeth** should be observed (*see also* p. 46), and the finger should be run along the gum to feel for any teeth that may be about to come through.

One must then proceed to an examination of the **throat.** The child should be wrapped in a towel to restrain the movements of its arms. The mother or nurse sits down opposite a good light and takes the child on her lap. Another assistant steadies the head from behind. The child having then been induced or compelled to open its mouth, one introduces a small-sized tongue-depressor and holds down the tongue, thus exposing the pharynx. The finger will often serve instead of an instrument, and has the advantage of frightening the child less. Look for any enlargement of the tonsils, for any redness of the mucous membrane, and especially for the presence on it of any membranous patches.

Palpation of the pharynx must also be carried out in many cases. To do this one must stand behind the child, and, when the mouth is open, push in the cheek from one side between the molar teeth. This serves as a gag, and effectually prevents the child from attempting to bite. The forefinger is then passed to the back of the pharynx and up behind the soft palate. Note the presence of any adenoids, or any bulging into the posterior wall of the pharynx, which may be the indication of the presence of a retropharyngeal abscess.

We have now indicated the general routine method to be employed in examining a child, but there remain some special points which we have not yet taken up.* These we shall consider briefly under the different systems :—

1. **General condition.**—Special importance attaches to the regular weighing of children. Alterations in **weight** from time to time are of much help in prognosis and treatment. It should be remembered that a healthy child should weigh at birth about 7 lb. This should be doubled by the time the fifth month is reached, and trebled in the first year. By the sixth

* The following summary of the chief facts to be noted in the general inspection and palpation of a child may be of service. It is taken from a scheme in use by Dr. John Thomson :—

Appearance (if healthy or otherwise)—nutrition and development—complexion (anæmia, cyanosis, jaundice, etc.)—state of skin (dryness, moisture, eruptions, desquamation, pigmentation, œdema)—attitude, expression, demeanour, temper.

Shape of head and state of its ossification (fontanelle, craniotabes)—facial irritability—hair—eyes, nose, and ears (formation of, and if any discharge from)—shape of thorax, abdomen, back, and limbs (especially the hands)—enlarged glands—evidence of rickets, syphilis, and tuberculosis.

Character of voice, cry, and cough—rate and character of respiration; if noisy, dyspnœic, or painful—movements of alæ nasi—rate and character of pulse—temperature.

Palpation of abdomen (tenderness, resistance, fluid, size of liver and spleen, tumours, etc.).

year it is again doubled, so that a healthy child of six should weigh about 3 stones. This is again doubled when the fourteenth year is reached.

Measurement of the head is often of importance. Two measurements are usually sufficient—a coronal measurement from one auditory meatus to the other, and a circumferential measurement at the level of the root of the nose and external occipital protuberance.*

2. **Alimentary system.**—Note that the **liver** is normally rather large in children, and usually reaches at least ½ in. below the costal margin. Enlargement of the **spleen** is very frequent in infantile diseases. It is best made out by palpation, the hand being passed across the child's abdomen from right to left. By depressing the finger-tips opposite the 11th interspace the edge of the spleen, if the organ be enlarged, may be felt as it descends during inspiration.

Inspection of the **stools** should never be omitted. The healthy infant, on the breast or bottle only, has two or three stools daily. These should be of the colour and consistence of beaten-up eggs. Any alterations in frequency, colour, or consistence, or the presence of worms, should be carefully noted.

3. **Circulatory system.**—Note that the **apex beat** of the child is normally rather higher than in the adult. It is normally outside the mammary line up to the third year, in the mammary line from the third to the tenth year; after that age it gradually assumes the adult position. It should also be observed that alterations in the general contour of the **præcordia** are much more frequent results of cardiac disease in children than in adults. As regards **auscultation**, it should be remembered that the pulmonary second sound in a young child is normally rather louder than

* For normal circumferential measurements of the skull at various ages, *see* p. 557, foot-note.

the aortic. The pulmonary second is accentuated if it be permanently louder than the first. The aortic second is accentuated if it be as loud as the pulmonary.

Remember also that hæmic bruits are very rare in babies, while congenital bruits are relatively very frequent. We have already mentioned that the cardiac rhythm in the child is not infrequently irregular even in health.

4. **The blood.**—It is sometimes difficult to get a large enough drop of blood from the ear of a child. In that case, a piece of woollen thread should be twisted round the great toe—not too tightly—and the latter punctured with a triangular needle at the root of the nail. In very young children, nucleated red blood-corpuscles are normally present in the blood in small numbers.* The leucocytes are more numerous in the child than in the adult; 12,000 per c.mm. is about the average number throughout infancy. The uninucleated leucocytes are both relatively and absolutely more abundant than in the adult, amounting to about 45 per cent. of the total leucocytes in the first three years. In new-born babies the percentage of hæmoglobin is often very high, but throughout the rest of infancy it is lower than in the adult.

5. **Respiratory system.**—A child uses the diaphragm much more than the intercostals in breathing. Hence the movements are chiefly abdominal, and there is little real chest expansion. Indrawing of the lower interspaces on inspiration should always be looked for. It occurs whenever there is obstruction to the entrance of air (e.g. diphtheria), but may also be present when there is collapse of the lower parts of the lungs, and also in pneumonia. In "extra-auscultation" one should be on the look-out for any stridor, and for the existence of a short, grunting

* This is only true during the first few days of life.

expiration. The latter is a frequent sign of severe respiratory disease. In the adult the normal cycle of respiration is, of course, inspiration, expiration, pause. In the child this is often reversed, so that one gets first a short expiration, succeeded by a longer inspiration, and then by a pause. This reversal is specially frequent in respiratory disease or embarrassment. The respiratory pauses are often very prolonged in the child, so that one has to wait a long time if one is auscultating before the next inspiration is heard. The normal breath sound in the child is, after the age of six months or so, **puerile** in type. **Vocal resonance** is often difficult to estimate. In babies one may make use of the cry as a producer of vocal resonance; in older children one may ask them their name, get them to count, etc. It should be remembered as a general rule that if the breath sounds are distinctly harsher on one side than the other, then the harsh side is probably the normal. Children's chests conduct sound very readily. Hence abnormal sounds, especially crepitations, are very apt to be heard on both sides, although they are really only being produced on one. The great frequency of collapse of part of the lung should be borne in mind in diagnosing pulmonary disease in infancy. In percussing the lungs in children one must, as already mentioned, *use a very light stroke.* One should also take care only to percuss when the chest has been filled by an inspiration, otherwise one may be led into thinking that there is dullness present.

It should also be remembered that the chest wall of a young child is so elastic that one can often obtain the "cracked-pot" sound on heavy percussion, even although the lung be perfectly healthy. This is especially apt to occur if the child be crying.

6. **Urinary system.**—It is difficult to collect the total quantity of urine passed by a child per diem.

A rough table of the average quantity for each child will be found at p. 307. Sugar is very rarely present in the urine of children, but albumin is often met with, even in healthy babies.

7. **Nervous system.**—**Motor paralysis** is to be made out by watching whether the child ever moves the suspected limb. One cannot estimate the paralysis as one does in adults, by means of passive resistance. Remember that inability to walk is not necessarily a sign of paralysis of the legs. One must note whether the legs are moved when the child is sitting or lying. Thus a rickety child may not be able to walk, but moves his legs freely if one tickles the soles. A child with infantile paralysis of the legs cannot move the limbs in any circumstances.

The **knee jerks** in little children are best elicited by placing the child's foot on one's hand as a stirrup, and then gently percussing the tendon. The latter lies rather to the outer side in the child, and is comparatively narrow, so that one may easily miss it.

The **superficial reflexes** are usually more brisk in healthy children than in adults. The exact localization of **sensory paralysis** is extremely difficult in children, but sensory lesions occur only rarely in infancy.

In examining the **eyes** with the ophthalmoscope, the direct method is the best to employ. The child may be examined while lying on its back, a lamp being held alongside the head, but at a somewhat lower level. It may be necessary to hold open the lids; but as far as possible avoid touching the child at all. One must often be satisfied with mere fleeting glimpses of the disc.

In testing light perception in little children, it is best to hold a candle in front of the eyes, and see if they attempt to follow its movements. One may also threaten the cornea by suddenly bringing the finger

near it, and observing whether the child winks before the eye is touched.

In examining the **ears,** one must remember the shortness of the auditory meatus in the child, and the great obliquity of the drum membrane. The magnification of the view by means of an ophthalmoscopic lens is a useful aid in these cases.

It is often difficult to gauge the **intellectual capacity** of a young child. Early signs of idiocy are —inability to support the head, which often rolls about helplessly; causeless screaming; inability to notice things; and backwardness in grasping.

In older children we can inquire as to progress at school, etc., or ask the patient questions; get him to count, multiply, and so on. The position of the child in the school is also a rough guide to the development of the intelligence.

A normal child should have begun to walk a little by the age of 18 months. Talking begins at a variable time after this. The distinction drawn by West between children that are idiotic and those that are merely backward may also be of help in the investigation. A backward child would be normal for a younger age; an idiot would be abnormal at any age.

CHAPTER XIII

EXAMINATION OF PATHOLOGICAL FLUIDS

In this chapter we propose to deal with the method of examining fluids which may be obtained from one of the body cavities or from abnormal growths, in order to acquire information which may be of help in diagnosis.

The fluid is obtained by means of **exploration.** An ordinary hypodermic needle may be employed, but special exploring needles—which are really merely large and strong hypodermics—are also sold. The needle should be of such calibre as to be capable of sucking up oil. If it can do that, it will be able to suck up any fluid likely to be met with in exploring. Before being used; the needle should be sterilized. This is best done by slipping it into a test tube, covering it with water or weak carbonate of soda solution, and boiling for three minutes. It should then be placed in boric lotion. The use of strong carbolic lotion for sterilizing exploring needles is to be avoided, as it produces a coagulation of albuminous fluids, which may block the needle or cause the fluid to seem opalescent. The patient's skin should be cleansed with some 1-in-20 carbolic acid, or with iodine solution, at the spot selected for puncture. As a rule, it is not necessary to employ any local anæsthetic. In very nervous patients a small spot of skin may be frozen by means of the ethyl chloride spray. It should be remembered, however, that the local reaction after freezing often causes more pain than the

original puncture. The needle should be held short, with the forefinger of the operator resting on it near the point. It should be introduced rapidly and steadily, but without any "stab." When the needle has been fully entered, the piston is withdrawn. Should no fluid be obtained, the needle is drawn slowly outwards, whilst a negative pressure is maintained in the syringe. It may then be found that fluid is obtained nearer the surface.

Where to Puncture

In the case of the **pleural cavity** the puncture is best made in the 9th or 10th space midway between the posterior axillary and scapular lines, this being the point at which fluid that is lying free in the cavity is most likely to be obtained. In cases of localized dullness, one must be guided, of course, by circumstances. Usually, one selects that point where the dullness, as estimated by the feeling of "resistance" on percussion, is most absolute and the breath sound faintest.

Puncture of the **peritoneal cavity** may be performed either in the middle line through the linea alba, or laterally, about a point on a line with, but rather above, the anterior superior spine. The former position ensures that no large blood-vessel will be injured; but by lateral puncture one is more certain of entering fluid, especially if the patient be turned over somewhat on to the side of operation. Before puncturing in the middle line, be sure the bladder is empty, and never insert a needle at any point unless it yields a dull note on moderately heavy percussion.

In puncturing the **pericardium** it is probably safest to select a spot between the apex beat and the outer limit of the pericardial dullness. A fine needle should be used.

Lumbar puncture is now commonly resorted

to for ascertaining the character of the effusion in cases of increased exudation into the subdural space.

It is performed as follows: Draw a line horizontally across the patient's back at the level of the highest points of the iliac crests. Make the puncture ½ in. below and to the right of the middle point of this line (i.e. in the 4th lumbar space). The patient should be lying in the left lateral position with knees and chin approximated. Local anæsthesia may be produced with the ethyl-chloride spray. A platinum-iridium needle, about 8 cm. in length and provided with a stiletto, is the best puncturing instrument, and it may be mounted for convenience (but not for suction) on an all-glass syringe. An antitoxin needle or a fine trocar will also serve the purpose. The needle should be passed horizontally forwards and a little inwards, pushing firmly through the ligamentum subflavum until the arachnoid sac is reached. The syringe is then detached and the fluid allowed to escape slowly until about 5 c.c. have been collected in a sterilized test tube. The puncture is closed with collodion. Strict antiseptic precautions must, of course, be observed throughout the operation, and it should not be performed unless the patient can rest for at least twenty-four hours afterwards.

For the characters of the fluid, *see* p. 584.

In the exploration of **cysts,** etc., one must be guided by local circumstances, the rule being to select for puncture that part of the tumour which is nearest the surface, and where one is not likely to injure important structures.

Examination of the Fluid

The fluid, having been obtained, should be transferred to a conical glass and allowed to settle.

Note first its **physical characters.** The chief

of these are the colour, consistence, specific gravity, odour, and the appearance of the deposit (if any).

As regards the **colour** of the fluid, one of the most important points to note is whether it be blood-stained or not. It must be borne in mind, however, that a small amount of blood is apt to get into the fluid in the process of exploring. Observe, also, whether the fluid be transparent, opaque, or opalescent.

Opacity is usually due to the presence of cellular elements; opalescence to fatty particles or large numbers of micro-organisms.

Opacity or opalescence due to fat may be removed by adding to the fluid some caustic potash solution, then shaking up with ether. The fat is dissolved out, and, if the ether be sprinkled on to blotting-paper, leaves a stain. Fluid which is opaque from the presence of much fat is usually spoken of as "chylous," and is derived from the thoracic duct. It may be simulated very closely by a "pseudo-chylous" fluid in which the milkiness is due to a lecithin-globulin complex which is held in suspension by the inorganic salts present. Removal of these by dialysis causes the precipitation of the lecithin-globulin body and the disappearance of the opalescence.

Pathological fluids are usually of a more or less watery **consistence.** Viscidity usually indicates the presence of mucin. It should be carefully noted whether or not the consistence of the fluid alters on standing. Many pathological fluids clot after standing for some time. The clot consists of fibrin.

The **specific gravity** is taken with a urinometer; the same precautions being used as in the case of urine (p. 314).

Most fluids are devoid of **odour**; sometimes, however, they are extremely fetid.

The amount and colour of the **deposit** should be

noted. If red, it probably consists of red blood-corpuscles ; if white, it may be made up of leucocytes, cancer cells, etc.

For **chemical investigation** the fluid should first be filtered. In the examination of the filtrate the following points must be attended to :—

1. The **reaction.** This is almost invariably alkaline. Sometimes it is neutral.

2. The presence of **serum albumin** and **serum globulin.** This is ascertained by means of the same tests as have already been described for the urine (p. 339). If these proteins are present in large amount, the fluid is coagulated on boiling, even although the reaction be alkaline. If proteins are scanty, the fluid should first be rendered slightly acid by means of dilute acetic acid.

As in the case of the urine, nothing is gained by testing for albumin and globulin separately. **Proteoses** and **peptone** are almost never found in the fluids under consideration.

The quantitative estimation of albumin and globulin cannot be accurately carried out in ordinary clinical work. Approximate results may be obtained by the use of Esbach's tube. The fluid must first be very freely diluted, so as to bring the specific gravity down to 1008, and should then be rendered acid by means of acetic acid.

3. The presence of **mucin** or **nucleo-protein** is proved by the appearance of a precipitate on the addition of acetic acid insoluble in excess.

4. **Sugar** should be tested for by rendering the fluid slightly acid, boiling, and filtering. The filtrate is then evaporated down to a small bulk, and the tests for glucose described at pp. 351–57 are applied.

5. **Urea** is not often present, except in traces, in ordinary pathological fluids. In fluids derived from

the urinary organs it may be more abundant, and should be tested for by removing all proteins by heat, evaporating the filtrate to a small bulk, and then testing for urea as described on p. 329.

Microscopical Examination of the Sediment

Some of the deposit is taken up with a pipette, and a drop of it placed on a slide, covered, and examined. If desired, films of it may be made in the same way as in the case of blood (p. 231). This succeeds fairly well if the deposit consists of cancer cells.

One may recognize under the microscope (1) elements derived from the **blood**—altered red and white corpuscles. The recognition of altered white corpuscles or pus cells is facilitated by mixing with a drop of the deposit a small quantity of a 1 per cent. solution of acetic acid to which a little methyl green has been added. The nuclei are then more easily recognized. (2) **Epithelial cells** of various sorts. The recognition of cancer cells is of special importance. The addition of a little picrocarmine or magenta facilitates the process. Do not mistake ordinary endothelial cells for them. Cancer cells should be large, numerous, and show grouping here and there. The cells met with in fluids derived from malignant ovarian cysts, or malignant peritonitis following such cysts, are known as Foulis' cells (Fig. 139). They are large cells containing one or more nuclei about the size of a red blood-corpuscle. They may either have a smooth outline or may show little buds or projections indicating rapid proliferation. Very similar cells are met with in effusions due to malignant disease of the pleura. In fluid derived from hydatid cysts, scolices and *hooklets* may be found (p. 106). (3) **Crystals**—e.g. of cholesterin (Fig. 140), or of fatty acids (Fig. 141), fragments of muscular tissue, etc.—are sometimes met with. (4)

The pus from cases of **actinomycosis** contains small seed-like nodules. If one of these be bruised between a slide and a cover-glass, and examined with the high power, it will be seen to consist of a central mass

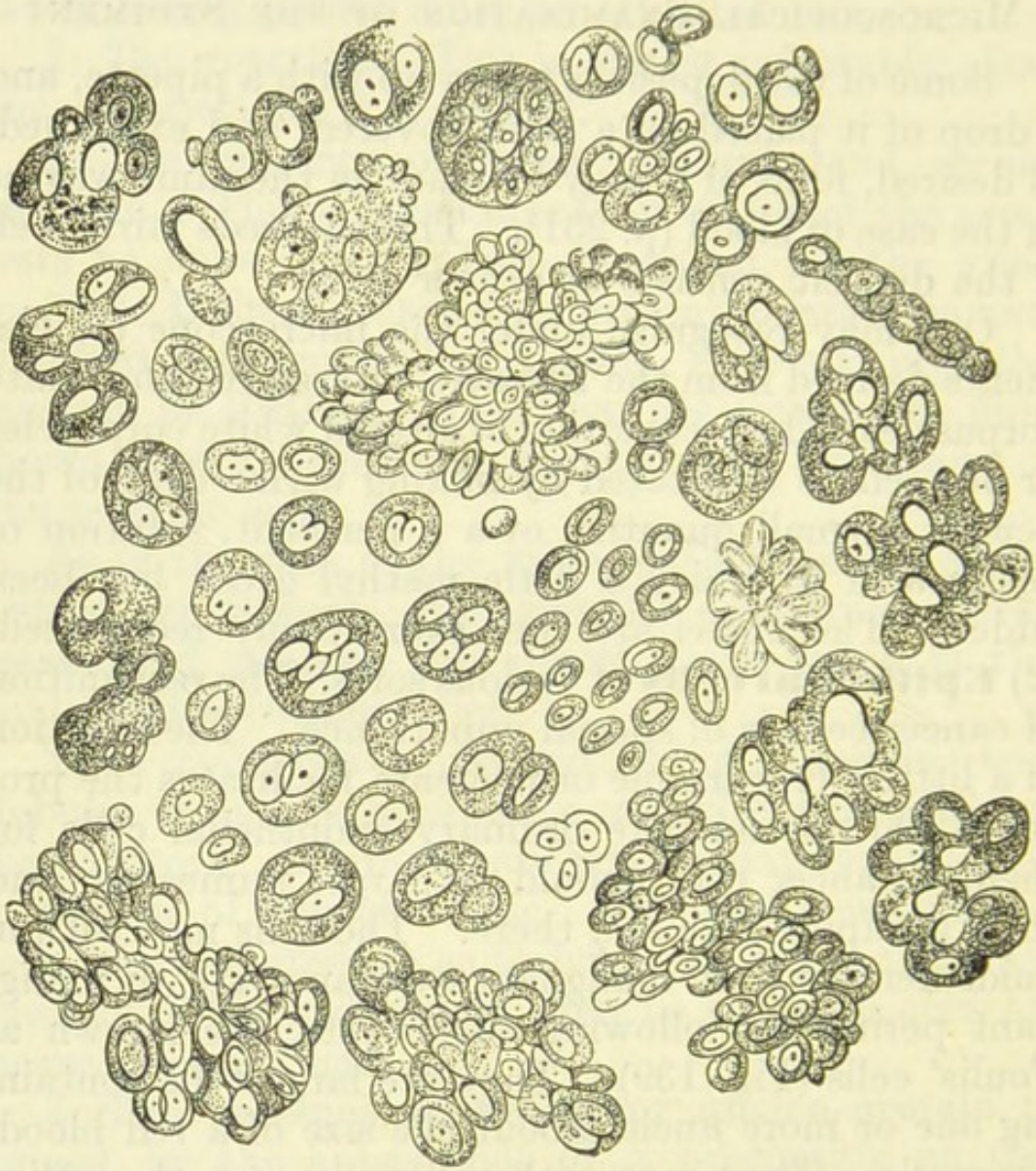


Fig. 139.—Foulis' cells.

of detritus, radiating from which are a number of club-shaped highly refractile bodies (Fig. 142). If there be any difficulty in identifying the fungus, cover-slip preparations should be made, and stained by Gram's method (Appendix, 23). For other staining methods, *see* pp. 630–31. (5) The detection of the

Amœba dysenteriæ in the pus of abscesses, especially in the liver, is often of great diagnostic value. The organism is described at p. 109. If one fail to find it in pus removed by exploration, or in that obtained when the abscess is first opened, one must not jump to the conclusion that it is not present in the abscess. It frequently does not appear in the discharge until three or four days after the abscess has been opened. This is probably to be explained by the fact that the habitat of the organism is in the wall of the abscess (Manson).

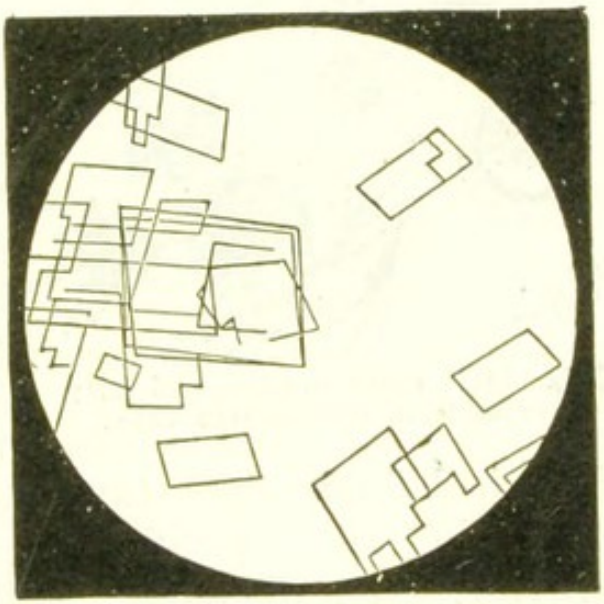


Fig. 140.—Cholesterin crystals.

General Characters of the Principal Fluids

1. **Inflammatory and dropsical effusions.** — Inflammatory effusions are often spoken of as exudates; dropsical effusions as transudates. They present the same general appearances, being clear fluids of a yellowish-green colour, and containing much albumin and globulin. It is very difficult to tell a dropsical from an inflammatory fluid by chemical or other examination. It would appear that the amount of proteins in an effusion depends much more upon site than upon cause. Pleural fluids contain the highest percentage of protein, peritoneal fluids rather less, and subcutaneous fluids very little. The fluid in cardiac dropsy is more highly albuminous than in dropsy of renal origin. From a diagnostic point of view, all that one can say is that a fluid with a specific gravity of

more than 1018, which contains more than 4 per cent. of protein, is almost certainly inflammatory; while one with a specific gravity of less than 1015, and a protein percentage of less than $2\frac{1}{2}$, is certainly dropsical. Between these limits one must be in doubt. Nor is the occurrence of coagulation in the fluid after tapping of much help. If the coagulation be very rapid and complete, the fluid is probably inflammatory, but considerable coagula may form even in purely dropsical fluids after standing for some time. Variations in the specific gravity, etc., of fluids obtained by repeated tapping in the same case are of no prognostic value. A marked rise in specific gravity and amount of protein may, however, indicate the supervention of inflammation.

Fig. 141.—Fatty needles and fatty crystals in degenerated cells.

2. **Fluid obtained by lumbar puncture.**—Ordinary cerebro-spinal fluid is perfectly clear and colourless,

Fig. 142.—Colony of actinomyces in pus, from a case of actinomycosis of the cæcum. × 300.

Characters of various Pathological Fluids

	Hydatids	Cerebro-Spinal Fluid	Hydro-nephrosis	Distended Gall-Bladder	Ovarian Cysts	Parovarian Cysts	Pancreatic Cysts	Amniotic Fluid
Colour ..	Colourless or slightly opalescent	Colourless	Colourless or yellowish	Colourless or bile-stained	Varies — brown, green, yellow, etc.	Colourless	Colourless or yellowish and turbid	Greenish-yellow
Consistence	Watery	Watery	Watery	Slightly viscid	Viscous	Watery	Watery	Watery
Specific gravity	1006–10	1007–8	1008–20	Low	1002–50	Low	Low but variable	1006–11
Coagulable proteins	Very scanty	Very scanty	Vary — may be fairly abundant	Usually scanty	Vary	Scanty	Variable	Scanty
Special characters	Presence of scolices or hooklets	Contains a cupric oxide reducing substance (dextrose)	*May* contain urea or uric acid.	May contain bile. Mucin usually present	Presence of pseudo-mucin (gives a white precipitate with alcohol after other proteins have been removed by boiling)	—	Contains cholesterin, and (if recent) will digest egg-albumin in alkaline medium, and may convert starch	Heavy animal odour. Contains some urea (at least in later months)

resembling distilled water, with a specific gravity of 1006. It contains very little globulin ($\frac{1}{50}$ per cent.), and if evaporated to a small bulk and tested will be found to reduce Fehling's solution, owing to the presence of dextrose. This is the kind of fluid which is found in cases of cerebral tumour. In cases of meningitis the fluid is not clear, but turbid from the presence of cellular elements. The turbidity may be so slight that it is only noticed after shaking the fluid and holding it up to the light. It contains quite an appreciable amount of protein, varying from $\frac{1}{2}$ per cent. in chronic cases up to 1 or 2 per cent. in those which are acute; and a small clot of fibrin usually appears in it after standing for some time ("cobweb-coagulum"). It does not usually contain any reducing substance. In acute cases of meningitis the cellular deposit consists chiefly of lymphocytes when the disease is due to the tubercle bacillus, and chiefly of polynuclears when it is due to the meningococcus, pneumococcus, or a pyogenic organism. In subacute chronic inflammatory cases of all sorts, and in some degenerative conditions—e.g. tabes and general paralysis—lymphocytes predominate. The bacteriological examination of the fluid is of special importance, and should be carried out as in Chap. XIV. The detection of trypanosomata is dealt with in Chap. V.*

3. For the chief characters of the other fluids likely to be met with on exploration, *see* table on p. 585.

* The following method is sometimes helpful in the detection of tubercle bacilli in cerebro-spinal fluid: Four or five c.c. of the fluid are placed in a sterilized test tube, which is wrapped in cotton-wool and allowed to stand for three to six hours. A small fine coagulum will then have formed in which the tubercle bacilli are gathered in clusters. The clot is removed, placed on a large cover-glass, teased, and dried. The cover-glass is passed thrice through a flame, stained with carbol fuchsin, decolorized with nitric acid (1 to 5 of water), washed in alcohol, and counterstained with methylene blue.

CHAPTER XIV

CLINICAL BACTERIOLOGY

In order to make a complete bacteriological examination, three methods of observation are necessary. These are microscopic study of the organisms, their cultivation on suitable media, and the effects produced by their inoculation into various animals. To obtain material, and to carry out the subsequent examination, the following instruments, all of which must be thoroughly sterilized immediately before use, are required :—

1. **Platinum needles,** about 3 in. in length and sufficiently stout not to bend very readily, fused into glass handles. Some of these should have their ends curved into a small loop, others ought to have the last half-centimetre bent at right angles to the rest of the wire, whilst a few should remain straight and have their free extremity somewhat flattened like a narrow spatula.

2. **Small sterile pipettes.** For clinical use, ordinary vaccination tubes, carefully sterilized and then sealed off, are very serviceable. A few larger ones are also needed.

3. **Swabs** for collecting particles from false membranes. To make these, a piece of stiff copper wire, about $1\frac{1}{2}$ mm. in diameter (No. 17 standard wire gauge), should be employed. A portion, 7 in. long, should have a flat loop made at one end, round which a small piece of absorbent cotton-wool must be firmly twisted. Wool must not be used that has been rendered antiseptic with mercuric chloride or other

disinfectant. A test tube about an inch shorter than the wire is then selected, and its mouth plugged with a stopper of cotton-wool, through which the wire passes. Thereafter the tube with its contained swab is carefully sterilized by heat. Several such swabs should be taken to the patient, and, after having been used to collect the material which requires examination, can be replaced in their test tubes and brought back to the laboratory without fear of contamination.

4. A **hypodermic syringe** which can be sterilized by boiling. One which the writers have found convenient is designed by Strauss, and sold by Collin of Paris. The plunger is made of elder-pith, and by a simple adjustment can readily be fitted to the piston rod, and the other joints are also rendered secure by discs of the same substance. Boiling improves rather than injures the pith fittings, and when one has worn out, a new disc can easily be cut with a penknife from a piece of fresh pith. Another pattern much used in pathological laboratories is the "Record" syringe, which has a glass barrel and a very accurately fitted metal-plunger.*

In order to sterilize the swabs, test tubes, and other apparatus, a small dry-heat oven is required; the workroom should also contain a centrifuge, test tubes, beakers, flasks, funnels, filter-papers, a Bunsen burner, capsules, watch-glasses, forceps, slides and cover-slips, stains and reagents, and a good microscope, magnifying 750–1,000 diameters. If cultures are to be made, an incubator and a number of other accessories are necessary in addition to those already enumerated.

The **capsules** should be of silica, nickel, or platinum, and should be used, wherever heat has to be applied to staining fluids, in place of watch-glasses,

* Supplied by A. Young & Son, Forrest Road, Edinburgh.

which crack readily. These capsules should be scrupulously washed, and then sterilized by heating to redness in a flame.

Slides and cover-glasses. The latter must be of No. 1 thickness, and both should be cleansed with Van Ermengem's solution. Thereafter they may be kept in a covered vessel under alcohol until they are required. (*See* Appendix, 32.)

The most generally useful **forceps** for holding cover-glasses are those known as Cornet's. In these the spring is so arranged that the cover-glass is firmly held until released by pressure, and the blades are constructed so that the cover-slip is in a horizontal position when the forceps are laid down. The two blades of the forceps should be dissimilar, one side having an aperture punched in the spring. The film side of the cover-glass should invariably correspond with the aperture; this obviates all risk of error in preparing and mounting the specimen. In selecting a pair of Cornet's forceps one should reject any which when laid on the table fail to hold the cover-slip horizontally, and also those the ends of whose blades meet at a somewhat acute angle, as they are less secure in their grip, and are apt by capillary action to draw off the stain from the film.

The **stains** which are most employed belong to the basic aniline dyes, and are either simple aqueous and alcoholic solutions, or contain a mordant, such as an alkali, carbolic acid, or aniline oil, which makes the bacteria take up the stain better and retain it more firmly. For most purposes the following stains suffice:—

1. Fuchsin	.	Saturated alcoholic solution.
2. Gentian violet	.	„ „ „
3. Methylene blue	.	„ watery „
4. Bismarck brown (vesuvin)	.	Aqueous „

5. Gram's reagents.
6. Ziehl-Neelsen's stain (for tubercle)
7. Löffler's stain
8. Carbol thionin

See Appendix, 23–26.

The stains must not be kept too long, must be frequently filtered, and should be examined from time to time for bacteria, which occasionally invade them —especially when the solutions are aqueous—and, unless detected, may lead to serious mistakes.

The stains most used consist of 1 part of a saturated alcoholic solution and 9 parts of distilled water. Examples of the stronger stains are found in the Gram, Ziehl-Neelsen, and Löffler stains. The first of these is of special importance, as by means of it specific differences in bacteria emerge according as they do or do not stain when the method is applied. For ordinary purposes methylene blue or carbol thionin blue should be used; neither of these tends to overstain a preparation. Where, with one of the stronger stains, overstaining occurs, this may be corrected by washing out the excess with alcohol or weak acetic acid.

For mounting the specimens one employs Canada balsam in xylol. It is very important to remember that some stains make bacteria look larger than others do, and also that their apparent size is less when they are mounted in balsam than when the examination is made with a drop of water. When cultures are desired, tubes containing bouillon, nutrient gelatin, blood serum, plain agar, and glycerine agar must be provided. These can be obtained from dealers, but are better prepared in a well-equipped laboratory. The details of preparation are outside the scope of this textbook.

The **method of examination** which is most readily available in clinical work is the preparation of

films on cover-slips. The technique is as follows : A cover-slip, which must be thin enough to admit of the film being examined through it by an oil immersion lens, and which is thoroughly clean and dry, is taken, and a very small drop of the fluid under examination is spread over it in a thin layer by a sterilized platinum needle. If the material be too solid, as may be the case when cheesy particles are being examined, a drop of distilled water must be added and intimately mixed with the mass before the cover-slip is smeared with it. The excess of fluid is then allowed to evaporate, and the film is dried and fixed by holding the preparation, film upwards, some height above a flame. Sufficient of the stain to cover the entire surface of the cover-glass is now filtered on to the film. After the lapse of a couple of minutes, wash the slip with water, dry it thoroughly, and mount in xylol balsam.* A film may be made on a slide and either mounted in balsam or examined by putting a drop of immersion oil directly upon it.

The specimen must be examined with a high-power objective. In most instances an oil immersion lens of $\frac{1}{12}$ in. focus should be used, although in many cases a lower power, such as a Zeiss D objective, may be sufficiently strong for clinical purposes. The microscope should be provided with an Abbé condenser, the diaphragm of which must be used fully opened, and the plane mirror employed to reflect the light. The inner tube of the microscope should be drawn out to the length for which the objective is constructed. Continental objectives mostly work to best advantage with a tube length of 160 mm. English objectives require a tube length of 10 in.

* Some stains, e.g. Löffler's methylene blue, take much longer, and others, e.g. carbol fuchsin, stain so deeply that partial decolorization may be necessary. For details, *see* Appendix, 25, 27.

In using the oil immersion lens, a drop of prepared cedar oil is placed on the cover-glass, and the objective lowered by the hand or coarse adjustment until it touches the surface of the oil; the focusing is then carefully performed with the fine adjustment. As the lens is delicate and the working distance small, great care must be taken not to bring the lens and cover-glass into contact; and when the observation is completed the cedar oil must be gently wiped from the surface of the objective with a piece of old silk.

Living bacteria may be observed by means of a hanging-drop preparation. To make this, a slide with a hollow ground in its surface is required, and for use vaseline is smeared round the hollow. A clean square cover-glass is laid on the bench, a drop of bouillon placed on its surface, and a little of the culture is emulsified in the bouillon. The vaselined slide is lowered upon the cover-glass, which adheres to it. The preparation is then turned cover-glass side up.

In examining a hanging-drop preparation, as in testing for Widal's reaction, care must be taken not to break the unsupported cover-glass in focusing with the oil immersion lens. Curtis advises the following procedure in such cases: Find the edge of the drop with a low-power objective, and observe that beyond this there is an area of minute points of water condensed on the under surface of the cover-slip. This dew should be brought to the centre of the field, the low-power lens replaced by the oil immersion, and the latter directed upon the dew, which is much more easily focused than are the contents of the hanging drop itself. The slide is then moved, *without altering the focus*, until the drop occupies the field of vision, and its contents both near the centre and towards the periphery can be examined without any risk of breaking the preparation. Once the focus of the upper sur-

face of the drop has thus been defined, careful use of the fine adjustment will readily bring any other plane of the preparation into sight.

The organisms most commonly present in pathological secretions may be separated by smearing the material on the surface of sloped agar or blood serum. To do this the tube containing the pathological fluid is held between the thumb and first finger of the left hand, so that the mouth with its stopper of cotton-wool is on the palmar side. The tube to be inoculated is similarly held between the first and second fingers of the same hand, with the surface of the nutrient medium upwards. Both tubes should be held as horizontally as possible. A platinum needle is then taken in the right hand, sterilized by heating to bright redness, and allowed to cool again. The plug of the specimen tube is gripped between the fourth and fifth fingers of the right hand, the dorsal surface of the hand being towards the tube, and withdrawn. The needle is then dipped into the specimen, and the smallest trace of it is taken. The plug is now replaced in the specimen tube. The plug of the nutrient tube is then removed in the same manner, and the charged needle is drawn gently in a waved line along the surface of the nutrient medium. The plug is at once replaced, the needle again sterilized, and the culture placed in an incubator as soon as convenient.

When micro-organisms are very abundant in the specimen, several tubes should be successively inoculated without recharging the needle. In this way, though the growth is excessive in the first, in the third or fourth the colonies are more scattered, and pure cultures may be separated out.

When a number of tubes are being dealt with, a cheap metal pen-rack is a convenient support on which to lay the needle after it has been sterilized.

Special Methods

1. **Sputum.**—The sputa which are of the greatest importance bacteriologically are those of pneumonia and of phthisis. In pneumonia the diplococci are most readily found when the disease is at its height. Select a rust-coloured portion of sputum, spread it in as thin a film as possible, dry, and stain with carbol fuchsin or with Hiss's capsule stain (*see* Appendix, 29).

In tubercular sputum much of the success depends on the choice of a suitable specimen. The sample should be chosen from the interior of one of the muco-purulent masses that are seen underneath the serous fluid portion. If the mass is so tenacious that a suitable piece will not adhere to the needle, spread the sputum in a shallow glass vessel and either pick out the part required with sterilized forceps or plunge a hot needle into the mass, when a portion will adhere to it; care must be taken not to use the central charred mass. When the sputum comes from a phthisical cavity, it may contain almost pure cultures of tubercle bacilli in its interior, though the outer surface of the mass is naturally contaminated during its passage through the mouth. To prevent confusion from spread of contamination, the sputum ought always to be examined as soon as possible after it has been expectorated. (Frontispiece, *b*.)

In cases of hæmoptysis the best specimens in which to seek for bacilli are often the small dark-red clots that are coughed up a day or two after the attack has subsided.

In acute miliary tuberculosis without typical tubercular sputum, it may be almost impossible to demonstrate the presence of the bacilli.

The detection of tubercle bacilli, when these are scanty, in sputum, pus, etc., is facilitated by the use of "antiformin," which has a dissolving action on the

tissue elements present. Antiformin is a mixture of equal parts of liquor sodæ chlorinatæ (B.P.) and of a 15 per cent. solution of caustic soda. One part is added to five or six of the material to be examined, and allowed to act for two or three hours; the fluid is then centrifugalized. Films are made from the deposit, which ought to be scanty, and are stained for the bacilli in the usual way.

When a suitable specimen has been obtained, it should be spread very thinly and evenly on a cover-glass, and dried carefully over a flame. It should then be placed film downwards on the top of a little carbol fuchsin stain contained in a capsule, and the fluid heated to boiling. Generally, it is well to repeat the heating three or four times. Then wash in water and decolorize in a 20 per cent. solution of sulphuric acid until all the elements except the bacilli have parted with the stain. As a rule, this is accomplished in from one to three minutes. Wash once more thoroughly in water, then treat for one or two minutes with alcohol, dry, and mount in balsam.

The specimen must be examined with a high power and good illumination—by preference with an oil immersion lens and Abbé condenser—though for clinical work a lower power is often sufficient.

Many workers prefer to counterstain with a watery solution of methylene blue for half a minute. This has the advantage of revealing the other elements present in the sputum. It is, however, important to avoid overstaining the background, as this makes it more difficult to pick out the tubercle bacilli. All that is necessary is to colour the other elements enough to make them visible.

2. **Blood.**—The only satisfactory method of examining the blood for bacteria is to withdraw 2–5 c.c. from a vein by means of a syringe. The syringe,

which should have a capacity of at least 10 c.c., must be rendered thoroughly aseptic by boiling before use. As steel needles are rapidly rusted by exposure to the air after being boiled, they may, on removal from the water, be plunged into a tube of absolute alcohol and kept there until required, or, instead of boiling water, hot oil may be employed to sterilize them. The median basilic vein is usually selected as suitable in size and position, and after the surrounding skin has been scrupulously cleansed it is rendered prominent by applying a proximal bandage to the arm. The needle is quickly but steadily introduced against the blood-stream, care being taken that its point remains in the lumen of the vessel, and the required amount of blood is abstracted. This should be immediately transferred to several tubes of nutrient broth, glucose broth, nutrient agar, or other media, not more than 1 c.c. of blood being added to each tube. The tubes are incubated and further examination, by the microscope and by subculture, is made in the laboratory. This method should always be adopted when the presence of bacteria in the blood is suspected. Puncture of the spleen has been advocated on the Continent, but it may lead to unpleasant symptoms.*

The direct microscopic examination of films of the blood is practically valueless, because in most cases the number of bacteria present in the circulating blood is so small.

3. **Urine** may be examined, after careful disinfection of the meatus, either by drawing off a sample from the bladder with a sterile catheter, or by making the patient pass water, and, after the first portion of the urine has cleansed the urethra, collecting the

* Similarly, some observers have punctured the lungs, liver, and other organs to secure uncontaminated samples of the bacteria which they contained.

remainder in a sterile wide-mouthed stoppered bottle. The centrifuge should be invariably used to secure a deposit without delay, and the latter then examined.

One of the most important bacteria that may be found in the urine is the tubercle bacillus. The pus which is separated from the suspected urine is spread not too thinly on a cover-glass, which is washed in sterile distilled water, the centrifuge being employed for the purpose, and then manipulated in the manner already described for tubercular sputum. In cases where the sediment contains small purulent-looking lumps, these should be selected to smear on the cover glass. Washing with distilled water dissolves some of the salts present, and the tubercle bacilli are then much more easily demonstrated.

The bacilli when found frequently occur in clumps, but it may be necessary to examine six or more preparations before a clump is discovered.

One of the chief sources of fallacy in examining for tubercle bacilli results from the presence of the smegma bacillus, which has very similar morphological characteristics, and especially resists the decolorizing action of sulphuric and nitric acids. To distinguish it the cover-slip should be immersed for ten minutes in a solution of hot caustic soda to which 5 per cent. of alcohol has been added. The specimen is thereafter washed with water and absolute alcohol. Tubercle bacilli thus treated still retain the stain when exposed to the action of mineral acids, whilst the smegma bacillus is decolorized. Our experience has been that the smegma bacillus is readily decolorized by a minute's exposure to absolute alcohol after treatment with sulphuric acid, and therefore we regularly treat all films in which we suspect the presence of tubercle bacilli with alcohol, as described under the examination of sputum (p. 595).

In cases of cystitis the most commonly found organisms are the bacillus coli, the gonococcus, and the tubercle bacillus. In some instances, especially in women or in persons on whom catheters have been passed, the ordinary staphylococci of suppuration are also present in large numbers.*

In urethritis resulting from gonorrhœal infection the gonococcus is present, but is often associated with numerous other diplococci, which are not very readily distinguished from it. Since it is of importance medically as the cause of certain affections which resemble rheumatism, it is necessary to be able to recognize it. Films may be made in the ordinary way, stained lightly with watery solution of methylene blue, or by Gram's method, using weak carbol-fuchsin (1 part to 20 of water) as a counterstain. The organism will be described subsequently.

4. **Pus and other fluid exudations** are best obtained from the cavity in which they lie by aspiration with a sterile hypodermic syringe, after thorough disinfection of the skin where the needle is to be inserted. Among the most important of such exudations are those into the pleural cavities. These may be either sero-fibrinous or purulent. In nearly three-fourths of the cases of empyema which have been examined, the organisms that have been found are either pneumococci, staphylococci, or streptococci, and in the case of adults the last constitute fully half of the total. In children the pneumococcus preponderates. As regards prognosis, the presence of streptococci gives much the gravest outlook, and this agrees with the relatively favourable issue of the

* The name "bacteruria" has been given to conditions where the urine, on being examined immediately after it is passed, is found to contain bacteria. Many writers, however, limit the name to cases where the bacteria present are not tubercle bacilli or the ordinary micrococci of suppuration.

disease in children. There can be no doubt that a very large proportion of pleurisies and empyemata are due to tubercular infection. It is extremely difficult to demonstrate the bacilli either by culture methods or in cover-slip preparations, but inoculation experiments have frequently been successful.

5. Specimens from **false membranes** are best obtained by removing a small portion with a swab or forceps. Care must be taken not to touch any other part of the patient's mouth with the swab. In cases of suspected diphtheria it often happens that so many organisms are present on the surface of the membrane that it is almost impossible to identify the bacillus of diphtheria among the other bacteria which are associated with it. Under these conditions the piece of membrane should be washed in a flask with a little boiled water. After being agitated in this for a short time, the cocci, mucus, and other adventitious elements which have adhered to the surface become for the most part detached, and the specific organism can thereafter be obtained in a much purer condition by thrusting a platinum needle into the membrane after its removal from the flask, and inoculating several tubes successively without recharging.

6. **Cerebro-spinal fluid.**—The technique of lumbar puncture has been described on p. 578. For bacteriological examination the fluid must be caught in a sterile test tube. It ought to be centrifuged and films stained by Gram's method, counterstaining with carbol-fuchsin 1–20. Pneumococci if present will be stained violet, and the meningococcus pink. Films should also be stained for the tubercle bacillus, and the occurrence of influenzal meningitis must be kept in mind. Cultures ought to be made on ascitic agar and on blood agar.

7. **Fæces.**—The fæces are always rich in micro-organisms; it is therefore of great importance to lift only a very minute fragment for examination, whether on the slide or by cultures.

To isolate the different organisms a plate culture should be made on MacConkey's neutral-red lactose agar.*

A small portion of the fæces is rubbed up with 10 c.c. of bouillon, one loopful of the product is transferred to the margin of the plate, and spread by means of a bent glass rod. The colonies of those organisms which cause fermentation of the lactose and thereby produce acid, e.g. B. coli, will be of a crimson or rose-red colour, whilst those which do not produce acid in lactose media, e.g. B. typhosus and Shiga's B. dysenteriæ, will not alter the colour of the medium and can thus be easily picked out from the others.

The following are the most important bacteria from the point of view of clinical examination :—

I. Bacilli

1. **Bacillus tuberculosis.**—It is rather small and slender (2 to 4 μ in length, and 0·3 μ in breadth). As seen in sputum it is not infrequently a little curved; and often two bacilli are found lying end to end, making an obtuse angle with one another. The staining may be uniform, or there may be small clear spaces of an ovoid form which are disposed at intervals. It is best stained by Ziehl-Neelsen's method. Primary cultivation from sputum is rarely successful on account of the presence of other bacteria, but cultures may be obtained from non-ulcerating lesions by using Dorset's egg medium.† The growth appears in five to fourteen days. In doubtful cases inoculation of a guinea-pig is the only satisfactory method of determining the presence of the tubercle bacillus with certainty.

* *Journ. of Hygiene*, v. 333.

† For composition of this medium, *see* Muir and Ritchie's "Manual of Bacteriology," 5th edit., p. 267.

For diagnostic purposes **Calmette's ophthalmic tuberculin reaction** is often of great value. It is conducted in the following way: A solution is prepared by precipitating tuberculin with alcohol and dissolving 5 mg. of the dried precipitate in ten drops of sterilized water. One drop of this solution is placed at the inner canthus. In three to six hours the reaction takes place. The conjunctiva becomes congested and slightly œdematous, the lachrymal caruncle appears red and swollen, or in some cases covered with exudation with a trace of pus. The eye may smart slightly, or in a few cases become more sharply inflamed, and remain so for five or six days.

In rare cases the reaction may be delayed, and only occur after twenty-four or forty-eight hours.

Statistics appear to show that the reaction is fairly reliable. It is found to be positive in about 95 per cent. of cases of obvious tuberculosis, in 81 per cent. of suspected cases, and in 8 per cent. of cases where there is no other evidence of the existence of tubercle. When positive it does not prove that the tubercular process is active at the time of applying the test, as the reaction may occur equally well where there is an old quiescent focus. In some cases of very acute tuberculosis the test gives a negative result. It should never be used when there is the slightest suspicion of local tuberculosis in the eye.

Von Pirquet's cutaneous tuberculin reaction is also used for diagnostic purposes. The skin over the deltoid region or the flexor aspect of the forearm is thoroughly washed, then well rubbed with ether and allowed to dry; a small area is scarified with an ordinary vaccine scarifier to act as a control. At some little distance on each side a drop of "old" tuberculin is placed on the skin, which is now similarly scarified through the fluid. The fluid is allowed to soak up into a small pledget of cotton-wool, which is removed at the end of ten minutes. Sometimes human tuberculin is used for one spot and bovine for the other. In the case of a positive reaction, some redness and swelling appear within a few hours, and on the following day there ought to be an inflammatory papule, sometimes vesiculated. Reaction goes on for about forty-eight hours. In a negative reaction there is no greater change in the areas treated with tuberculin than in the control area which has been merely scratched.

A considerable number of bacilli, not usually regarded as pathogenic in man, share with the *Bacillus tuberculosis* the property of resisting the decolorizing action of acids and of alcohol. This group of bacteria are described as "acid-fast,"

and since they possess many of the other morphological characters of the true tubercle bacillus, and have been occasionally found in sputum from the lungs, in mucus from the respiratory passages, and not very infrequently in urine containing pus, they may give rise to errors in diagnosis. So far as is known at present, the most characteristic difference is that, even at the most favourable temperature, tubercle bacilli grow much more slowly on culture media than do the other members of the "acid-fast" group, many of which, unlike the tubercle bacillus, can also flourish at the ordinary temperature of the laboratory. As these bacteria are fairly numerous outside the body they may readily contaminate secretions which have been laid aside for examination, and, besides this, there can be little doubt that they have often been mistaken for the tubercle bacillus in milk and other articles of diet.

2. **Pfeiffer's influenza bacillus** is a minute rod (1·5 μ long by 0·3 μ broad) with rounded extremities; the ends stain more deeply than the centre. It is not coloured by Gram's method, but can be demonstrated by a weak solution (1 in 10) of carbol-fuchsin applied for 5–10 minutes. It is found in the sputum and occasionally in the blood of patients suffering from influenza, in certain septicæmic conditions, in middle-ear disease, and occasionally as a cause of meningitis. Its culture is difficult, and blood agar must be used for primary inoculation.

3. **Bacillus diphtheriæ** (Klebs-Löffler).—This bacillus averages about 3 μ in length, the long variety considerably more, and 0·6 or 0·7 μ in breadth. The ends are rounded and rather stouter than the centre, and stain more deeply; frequently there is unequal staining of the protoplasm. (FRONTISPIECE, *a*, *a'*.) In most instances the bacillus is very readily coloured by Gram's method.

The procedure which should be adopted in cases where a patient's throat is to be examined for the bacillus of diphtheria is as follows: Set the patient in a good light. Depress the tongue with the handle

of a spoon ; then take a swab, pass it to the back of the mouth without touching the lips, gums, or tongue, and press it gently against the suspected patch on the tonsil or fauces. By rotating the swab a portion of the false membrane is entangled and brought away, and the specimen thus obtained is returned to the tube and taken to the laboratory. A cover-glass preparation may be made directly from it, but is likely to prove unsatisfactory, as many different organisms are so abundantly present that it is seldom possible to detect the Klebs-Löffler bacillus with certainty. To start cultures, a platinum needle, previously sterilized and allowed to cool, is charged from the false membrane, and four tubes are successively inoculated from the needle, which is drawn in a streak along the surface of each without recharging. Frequently it is necessary to make a bacteriological examination of a throat which may present none of the clinical characteristics of diphtheria. Here the swab is stroked over both tonsils and also over the back of the pharynx. Cultures are made by, in turn, stroking the swab over the surface of one or more tubes of culture medium. By far the most satisfactory medium for demonstrating the diphtheria bacillus is blood serum. Gelatin and glycerine agar are untrustworthy, and should never be employed for diagnostic cultures of the diphtheria bacillus.* After inoculation the tubes are placed in an oven at 37° C. If the Klebs-Löffler bacillus is present, growth will often be manifest in eighteen, and always in twenty-four hours, by which time the colonies in the third and fourth tubes will be as large

* Failing blood serum, a medium composed of agar prepared with ascitic, pleuritic, or hydrocele fluid, containing 2 per cent. of a 10 per cent. solution of caustic potash, with 5 per cent. glycerine, and 1 per cent. grape sugar, as recommended by Kanthack, may be tried.

as pinheads. In colour they are dull white, and appear distinctly denser in the centre when viewed by transmitted light. The colonies are circular, and spread rather rapidly. In the first and second tubes the characters are not so well seen, because the colonies are so numerous that they rapidly fuse, and so lose their contours. Specimens obtained from the individual colonies must be microscopically examined, and pure cultures can be started from some of them by re-inoculating fresh serum tubes. In old cultures, and sometimes also in the membrane obtained from cases of diphtheria, involution forms of the bacillus, usually club-shaped, are common.

In doubtful cases the diagnosis may be established only after inoculations have been performed. Neisser has described a special method of staining the diphtheria bacillus, which he considers important when a differential diagnosis has to be made. A culture of the bacilli in question must first be prepared on Löffler's solidified blood serum, at a temperature of 34° or 35° C.—temperatures over 36° C. render the reaction untrustworthy. After an incubation period of ten to twenty hours, a cover-slip preparation is made and stained for three or four seconds in the following solution :—

Methylene blue (Grübler) . . .	1 part.
Alcohol (96 per cent.) . . .	20 parts.
Distilled water	950 ,,
Glacial acetic acid	50 ,,

The film is then rinsed in water, and counterstained for three or four seconds in a filtered solution made by dissolving 1 part vesuvin in 500 parts of boiling distilled water. If the organisms be true diphtheria bacilli, their bodies will be stained brown throughout their whole length, but one or two, sometimes three, granules of a bright-blue colour, known

as Babes-Ernst bodies, are also visible. Where there are only one or two granules they occupy positions near the ends of the bacillus; the third, when present, is situated about the middle. The granules appear to have a greater diameter than the rest of the bacillus, and their shape is slightly oval.

Occasionally, when the membrane is peculiarly rich in adventitious bacteria, it may be necessary to place it in a flask with some boiled water and to treat it in the manner described above, before proceeding to inoculate the tubes.

When information is sought regarding other bacteria which may also be present in the specimen, it is best to break up a small fragment in bouillon and inoculate various culture media (e.g. blood agar, glucose agar for anaerobic bacteria) with a drop of the fluid.

In practice one finds that sometimes the membrane contains a preponderant proportion of Klebs-Löffler bacilli; in many cases, however, other bacteria occur in large numbers along with it, streptococci being frequently present, particularly in severe cases. In other instances pneumococci and staphylococci occur.

In addition to the typical form of Klebs-Löffler bacillus there is a stunted form which is shorter and more irregular in outline. One cannot, however, estimate with any certainty the toxicity of a specimen from its morphological characters, nor, on the other hand, is one justified in considering that the bacillus is not a true diphtheria bacillus because it happens to be devoid of virulence, although the intensity of its toxin will obviously influence the clinical history of the case from which it is derived. It is not, therefore, expedient to classify such non-virulent bacteria as "pseudo-diphtheritic," since experience has shown that the virulence of diphtheria bacilli may vary

exceedingly even when they have sprung from a common source.

The term "pseudo-diphtheritic bacillus" has also been applied to various bacteria whose morphology is almost identical with the true, especially the short form, but which, under the tests of staining, culture, and inoculation, are found to deviate more or less from the latter. Used in this sense the term is convenient, if not strictly accurate. As alkaline bouillon containing a trace of glucose is gradually rendered acid by the true diphtheria bacillus, the change usually occurring within two days, whilst many of the "pseudo" forms do not alter its reaction, the test may be employed within certain limits to discriminate between them.

There is good reason for believing that the recrudescence of diphtheria in a community is frequently due to the diphtheria bacillus persisting in the throats of those who have recovered from the disease. It is thus of the greatest importance that a convalescent should not be allowed to mix with healthy individuals until bacteriological examination shows the throat to be free from the organism. Besides the throat the other situations where the diphtheria bacilli occasionally find a settlement are the nose, the conjunctiva, and the vulva.

4. **Bacillus typhosus** (Eberth).—This organism can sometimes be obtained in the spleen during the course of the disease, from the blood, from the lymph, in the rose-coloured spots, and from the abscesses which sometimes occur in various parts of the body during typhoid fever. It is observed also in the urine if albuminous, and in the stools. It is motile, and by suitable methods flagella can be demonstrated. The ends are rounded, the length 3 or 4 μ, the breadth about 1 μ. It does not stain by Gram's method,

but is readily coloured by the ordinary aniline basic dyes.

In arriving at an early diagnosis of typhoid fever **Widal's test** takes a very prominent position. It is based on the fact that after a certain time has elapsed the blood serum of a typhoid patient acquires a power of interfering with the ordinary behaviour of the bacillus, and produces the phenomenon known as *agglutination*. It is conducted as follows :—

Apparatus.—A small lancet-shaped needle to obtain blood. This must be capable of ready sterilization.

A sterile pipette (Fig. 143) to collect the blood and transmit it to the laboratory.

A platinum needle ending in a loop 1 mm. in diameter. (This will lift about 2 mg. of fluid.)

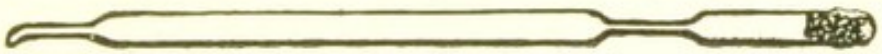


Fig. 143.—Widal's pipette. Actual size.

Several sterilized watch-glasses, slides, and cover-glasses.*

A supply of sterile bouillon or peptone solution.

A tube of bouillon containing a culture of typhoid bacillus no more than twenty-four hours old, or an emulsion of the bacilli made by adding a small portion of agar culture to bouillon. In the latter case the mass of bacilli on the platinum loop should be broken down at the margin of a watch-glass and gradually mixed with the contained bouillon. The emulsion should show a faint turbidity. The fluid containing the bacilli should always be examined microscopically to see that no spontaneous clumping is present.

* An ordinary slide may be used if care be taken not to press down the cover-slip unduly ; or a slide with a central depression may be preferred, and a hanging drop preparation employed.

A microscope capable of magnifying at least 250 diameters (preferably from 300 to 400 diameters) should be used, though with lower powers a good deal may be made out.

Method. — (*a*) Carefully sterilize the patient's finger, and obtain several drops of blood by a prick in the thin skin near the root of the nail. The blood is drawn into the pipette, which is then sealed off and taken to the laboratory.

(*b*) When sufficient time has elapsed to allow the serum to separate from the clot, break off the ends of the pipette and expel the serum into a sterile watch-glass previously labelled "serum."

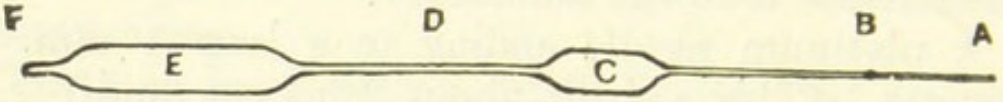


Fig. 144.—Wright's pipette. One-half actual size.

A, open end of capillary tube; B, index; C, mixing chamber; D, capillary tube connecting C and E; E, air chamber; F, sealed end.

(*c*) By means of a pipette* (Fig. 144) dilute 1 measure (say 5 c.mm.) of the serum with 19 measures (say 95 c.mm.) of sterile bouillon or peptone solution, and expel this into another watch-glass marked "1 in 20."

(*d*) With the platinum needle take four loopfuls of the 1 in 20 serum and deposit them separately

* Any simple form of pipette may be used. Wright's is easily made from a piece of glass tubing in the following manner: One end of the tube is sealed, and at about 1½ in. from this end it is drawn out to form a capillary tube about 2 in. long and open at its lower end. The thicker part of the tube is again heated at about 1 in. from its sealed extremity and drawn out so that one finally has a pipette consisting of a couple of bulbs, the upper one sealed at one end and at the other end communicating by a narrow tube with the lower, which in turn communicates with the outer air by the capillary tube. On warming the upper bulb the contained air is rarefied, and, as it again cools, the partial vacuum which is formed will suck up fluid into the lower bulb. Instead of careful graduation, a single mark is made by means of a speck of sealing-wax at a selected point of the capillary tube. Each

on the slide or cover-glass. Then sterilize the loop, and, when it has cooled, deposit six loopfuls of the bouillon culture or emulsion in the same manner. Mix the drops as quickly as possible, place the cover-slip on the slide, and seal the edges with vaseline to prevent evaporation. This preparation should then be marked "1 in 50." In cases of difficulty, dilutions of 1 in 30 (four loopfuls of the 1 in 20 serum and two of bouillon culture or emulsion) and 1 in 100 (two loopfuls of 1 in 20 serum and eight of bouillon culture or emulsion) should also be made, whilst a control experiment must be simultaneously performed. In the latter the blood of a healthy person should be substituted for that of the suspected typhoid patient, and the test carried out exactly as described above. The slides should then be systematically examined with a magnification of about 300 to 400 diameters. If the serum be taken from a patient who is not the subject of typhoid fever, the bacilli preserve their motility unimpaired, and continue to do so for days if the specimen be suitably preserved. Moreover, they are diffused with tolerable uniformity through the fluid, and show no distinct tendency to form clumps.

If, however, the serum be taken from a patient after the first week of an attack of typhoid fever, or even on the third or fourth day of the illness, though

time the fluid which is being aspirated reaches this mark the end of the tube is raised for an instant to admit a tiny bubble of air. When this in turn reaches the mark the bulb is again raised, and the process repeated until the required number of measures have been drawn into the lower bulb.

In performing Widal's test extreme accuracy in the degree of dilution is not essential, and therefore ungraduated tubes, such as Wright's, used with reasonable care, meet the requirements of the case. Those who for any special purpose desire a very exact dilution should employ the leucocyte pipette of a Thoma-Zeiss hæmocytometer, or Hawksley's graduated pipette with a screw compressor.

in this case the phenomena will be less distinct, the following facts will be observed : The motility of the bacilli is almost at once impaired, and after the lapse of a short time their movements wholly cease. They also tend to become agglomerated into clumps, which in a typical case will begin to form soon after the microscopical examination has been commenced, and by the end of half an hour or an hour hardly any solitary bacilli will be found in the specimen. Even where the serum is weaker, as in a certain proportion of cases of typhoid, the same changes eventually supervene, but they occur much more slowly. As a rule, however, impaired motility and a tendency to form clumps are distinctly visible within half an hour, and there are but few cases in which the observations need to be extended over two hours.

In conducting the above examination the following points must be attended to : (1) The slides, watch-glasses, and other pieces of apparatus must be scrupulously clean. If they are not so, appearances of agglutination may occur even where the serum is taken from a man in good health. (2) As one is dealing with bacilli which are alive and virulent, any piece of apparatus which has been used must at once be thoroughly disinfected by heat or by 1-in-500 corrosive sublimate solution, and on no account laid aside before this has been done. (3) Never allow concentrated serum to come into contact with the bouillon culture even for an instant ; therefore dilute the serum before mixing it with the culture. Concentrated serum from a perfectly healthy person may induce clumping. (4) It is not necessary that the serum be separated from the clot. The red blood-corpuscles do not interfere with the reaction.

Widal's reaction may be obtained by employing dead cultures instead of a fresh one in bouillon. The bacilli are killed by heating the bouillon to 60° C. for one hour, or by adding one drop of formalin to every 150 drops of bouillon. This is then kept for use in sealed glass tubes, and has the advantage that it can be

utilized when the observer has no facilities at hand for the cultivation of living bacteria.

The agglutination test can also be performed macroscopically; the technique for this method is fully described in a valuable paper by Wright.*

The agglutination test may be applied to numerous other bacteria, and will be referred to when they are described. The technique in these other cases is identical with that of Widal's test for typhoid.

It need hardly be remarked that to examine a specimen of the stools directly under the microscope, with the view of detecting Eberth's bacillus, is certain to fail in its aim.

It is now recognized that an important factor in the maintenance of typhoid fever in a community is the existence therein of "typhoid carriers," i.e. individuals who, either with or without having suffered from the disease, harbour the bacillus in the intestine or urinary tract. When the occurrence of such a case is suspected the urine and fæces must be examined by cultural methods every fortnight for several months. The site of the continued growth of the bacillus in such cases is frequently the gall-bladder, and gall-stones are often present. All gall-stones should be subjected to careful bacteriological examination, and the typhoid bacillus should also be looked for in suppurative conditions about the gall-bladder.

5. **Bacillus coli** is a flagellate bacillus which appears under a considerable variety of forms. It is important as the cause of some cases of cystitis, pyelitis, and intra-abdominal suppuration. Some of its forms resemble Eberth's bacillus, but can be distinguished by the fact that it produces fermentation in lactose, in which process the medium becomes acid, as can be shown by the addition of litmus, and gas

* *Brit. Med. Journ.*, 1898, i. 355.

bubbles are formed. To isolate it from other organisms in cases of cystitis, cultures may advantageously be made on MacConkey's medium. The B. coli exhibits the agglutinative reaction, but it has been found that serum which agglutinates one species of colon bacillus may fail to produce the reaction in another.

6. **The paratyphoid bacilli.** — This group comprises a number of organisms isolated, especially from the fæces, in important pathological conditions. They include the paratyphoid bacillus itself, which occurs in the intestine and also frequently in the blood, in cases clinically resembling typhoid fever, but tending to pursue a milder course; the Gärtner bacillus and its allies, which are the cause of most of the cases of food poisoning (so-called "ptomaine poisoning"), and which again is found in the intestine and also, it may be, in the blood; the dysentery bacillus, which is associated with many cases of non-amœbic dysentery occurring from time to time, especially abroad, in an epidemic form; this organism is also responsible for some of the epidemics of so-called summer diarrhœa in America and for outbreaks of a dysenteric disease in lunatic asylums in this country. All the bacteria of this group closely resemble the typhoid bacillus, differing only in certain of the reactions on media containing sugars and in the serum reactions. These latter, however, can only be worked out in a laboratory.

7. **Bacillus choleræ** (Koch) is a curved vibrio with a flagellum at each end. It possesses very marked motility. The organism is shorter but slightly thicker than the bacillus of tuberculosis. Involution forms are rather common. It is readily coloured by basic aniline dyes, but is not stained by Gram's method. It responds to the agglutination test. (FRONTISPIECE, *c*.)

The gelatin plate cultures, and the so-called

cholera-red reaction in broth, or better in peptone solution,* are very characteristic, and should be practised in case of doubt.

In examining the stools of a suspected case, the organism will be found most abundantly present in the mucoid masses. A film should be made from one of these, and, after staining with dilute Ziehl's fluid, examined with an oil immersion lens. In cases where they are fairly numerous, and where their disposition is not too much interfered with by the simultaneous existence of other organisms, they will be seen to lie in rows, end to end, and all pointing in the same direction, like fish in a stream.

Equally characteristic is the appearance when a drop of the suspected stool is added to 2 c.c. of a decidedly alkaline solution of 1 part of sodium chloride and 10 parts of peptone in 100 parts of water. After eight to twelve hours' incubation at a temperature of 37° C., an abundant growth of the cholera bacillus will be found on the surface of the fluid, and from it plate cultures may be made. In cases of difficulty Pfeiffer's reaction should be tried. For details of this reaction the reader must be referred to textbooks on bacteriology.

8. **Bacillus anthracis.**—This organism, primarily the cause of anthrax in cattle, may secondarily infect man, either by producing a local skin lesion, the "malignant pustule," or by giving rise to extremely

* The red reaction is due to a nitrosoindol body. Many bacteria produce indol in bouillon, but the bacillus of cholera produces both indol and nitrites in twelve to twenty-four hours. The bacillus of Metchnikoff and the bacillus of Finkler, both of which resemble morphologically the cholera organism, also give this reaction, but in a less marked degree and after a longer period of growth. The addition of an acid capable of acting on the nitrites, and so liberating nitrous acid, is all that is necessary to produce the red coloration; and the best acid to use is pure sulphuric. The sulphuric acid must be absolutely free from nitrites, or the reaction may be obtained with other indol-producing bacteria.

fatal lesions in the respiratory tract, to which it gains access through the inhalation of infective material; the former lesion occurs chiefly in butchers and workers in hides and hair, the latter in wool-sorters. The organism can usually be easily isolated by infecting agar tubes with material from the pustule or, in respiratory cases, from the sputum. It is a rather large bacillus, and is recognized chiefly by characteristic appearances when growing on solid media. It is not usually found in the blood of man except sometimes just before death. Its presence cannot be certainly recognized by mere microscopic observation.

9. **Bacillus pestis.**—This bacillus is the cause of plague. It commonly gains access through a breach in the skin or mucous membranes, and makes its way by the lymphatic system to the neighbouring glands. In them it leads to the formation of buboes, and if not arrested there, passes on into the blood-stream, inducing a septicæmic condition. It is most readily demonstrated in the pus which forms in the buboes, but, when septicæmia has developed, bacilli can also be demonstrated in the blood. The appearances presented by the bacillus, alike in cultures and in material derived from patients, are very variable. Its most typical form is that of a small oval rod with rounded ends, devoid of motility. It often presents a characteristic bipolar staining owing to the central segment remaining uncoloured, or only feebly tinted by the dye; this is well brought out when methylene blue is employed, after preliminary treatment with dilute acetic acid. It is decolorized by Gram's method.

The trend of recent investigation points to plague being essentially an endemic disease of certain rodents—e.g. the rat (India), varieties of the marmot (China), and certain squirrels (America). From time to time man becomes infected from these animals

through the agency of fleas. Thus, when plague breaks out it is important to have a thorough investigation of the rodents of the locality, especially of animals found dead. In these the occurrence of swellings in the glands of the neck is suspicious, and such animals should be sent to a laboratory for complete examination.

10. **Tetanus, glanders, relapsing fever,** and numerous other more or less rare diseases are also due to bacilli, but cannot be further referred to here.

II. Micrococci

1. **Staphylococci.**—These occur in small masses and are readily stained by ordinary aniline dyes and by Gram's method. The individual cocci measure about 1 μ in diameter. The chief varieties are S. pyogenes aureus, S. pyogenes albus, and S. citreus.

2. **Streptococci.**—This group contains a considerable number of pathogenic organisms. They occur in shorter or longer chains.

(*a*) **Streptococcus pyogenes.** — Stains as the staphylococci. It produces severe suppurations with a great tendency to indeterminate extension, is the common agent in puerperal septicæmia, and causes many of the inflammatory conditions in the throat.

(*b*) **Streptococcus erysipelatosus** is very closely related to the last, and certainly cannot be distinguished by cover-glass preparations or by culture methods. The chains contain from five or ten to as many as forty cocci. Many authorities regard it as only a virulent form of S. pyogenes.

3. **Pneumococcus** (Diplococcus pneumoniæ, Fränkel).—These diplococci are the cause of croupous pneumonia, of many cases of empyema and peritonitis (especially in children), and also may give rise to meningitis (though they frequently occur also in

normal saliva). They are lancet-shaped, and the points of the lancets are directed away from each other. The diplococcus is enclosed within a capsule which stains less deeply than the coccus itself. They stain readily, and are coloured by Gram's method, which aids in distinguishing them from Friedländer's pneumobacillus, as the latter cannot be stained by it. (FRONTISPIECE, *d*). In cases of difficulty the capsules should be stained by Hiss's method (Appendix, 29).

4. **Diplococcus intracellularis** (Weichselbaum).—This organism is found within the pus cells of many cases of epidemic cerebro-spinal meningitis, and may be recognized in the fluid removed by lumbar puncture during the life of the patient, especially after centrifuging. In cover-slip preparations the diplococcus can be stained by several aniline dyes ; Löffler's methylene blue process yields satisfactory evidence of the presence of the organism, but films should always be stained by Gram's method and counter-stained with Ziehl-Neelsen 1–20. The organisms being decolorized by the Gram stain are coloured with the fuchsin. They are arranged in pairs, the long axes of the individual cocci lying parallel to each other ; the diplococcus thus presents a somewhat striking likeness to the gonococcus. Cultures should be made, in doubtful cases, on ascitic agar. Dr. Still has described a somewhat similar, but less virulent, diplococcus as occurring in the non-tuberculous posterior basic meningitis of children. Still's organism is more easily grown than the true Diplococcus intracellularis.

5. **Micrococcus tetragenus.**—This organism is found in the sputum which comes from phthisical, bronchiectatic, and other cavities in the lungs. The cocci are about 1·5 μ in diameter, and occur in groups of four enclosed in a capsule. They are readily coloured by Gram's method and by basic aniline dyes.

6. **Gonococcus.**—This is a diplococcus, the two cocci of which lie very close together and are thus difficult to distinguish. With high magnification they are seen to be kidney-shaped and have their concave sides facing one another. Sometimes the gonococci are found free; at other times, and more characteristically, enclosed in pus corpuscles, in which they occur in groups. They are readily stained by aqueous solutions of basic aniline dyes. They are decolorized by Gram's method, which often aids in clinching the diagnosis in a doubtful case. Advantage may also be taken of the fact to secure a double stain, the cover-glass being first treated by Gram's method and then counterstained with vesuvin or Bismarck brown. Most of the other microbes then stain violet, the cellular elements are light brown, and gonococci are darker brown. (FRONTISPIECE, *e*.)

To determine the identity of the gonococcus in doubtful cases the following criteria are available :—

i. The occurrence in the pus cells of paired bean-shaped cocci. At least four pairs in a cell should be recognized before venturing on a diagnosis.

ii. The typical cocci should be readily decolorized when treated by Gram's method.

Culture of the gonococcus is a matter of great difficulty unless the primary inoculations be made direct from the patient on to special media warmed to body temperature.

The organism is the cause of an important group of joint affections, and it can be isolated from the synovial exudate. It also occasionally gives rise to ulcerative endocarditis.

7. **Micrococcus melitensis** (Bruce) is a very minute oval coccus about $\frac{1}{3}$ μ in diameter, which can be isolated from the spleen of cases of Mediterranean (or Malta) fever. In cover-slip preparations from

cultures it is readily stained by basic aniline dyes, but it is decolorized by Gram's method. The cocci are for the most part arranged singly, but sometimes they form short chains. The Micrococcus melitensis exhibits the agglutinative reaction very distinctly, and it is by means of this that in doubtful cases the diagnosis ought to be made. The blood serum from the suspected patient should be mixed with a pure culture in exactly the same manner as has been detailed for typhoid fever (p. 607). By this means it has been proved that some of the Indian and other tropical fevers are due to the same organism as Mediterranean fever. The disease is spread in nature by means of the milk of the goat.

8. **Sarcina ventriculi** is described elsewhere (p. 92).

III. Streptothriciæ

Several members of this higher group of bacteria are pathogenic in man, and they can only be distinguished from one another by cultural and other characters, the details of which are beyond the scope of this textbook. The most important member of the group is the Streptothrix actinomyces or "ray fungus." The commonest lesions produced in man are granulomata (tending to break down into abscesses) about the jaw, neck, lungs, and intestine (especially the appendix). The organism occurs in the form of minute granules, lying free in the pus or other fluids, and easily visible to the naked eye. The granules are usually of a yellowish colour, but they may be white, greenish, or almost black.

If one of the granules be broken up under a cover-glass and examined, it is found to be made up of a felted mass of filaments which show true dichotomous branching. At the periphery of the mass the filaments are often arranged in a radiating manner, and elongated

pear-shaped bodies which are formed by a swelling of the sheath round the free extremity of a filament may sometimes be seen.

Film preparations made in the ordinary way may be stained with thionin blue or with carbol fuchsin, but the filaments are best demonstrated when stained by Gram's method.

IV. Other Organisms

1. **Spirochæte pallida.**—This organism is best demonstrated by the special technique of dark-ground illumination, but with an ordinary microscope it can often be recognized in the secretion from a chancre or condyloma, or in scrapings from the organs in cases of congenital syphilis, by the use of the Indian-ink method. For this, a fine quality emulsion of Indian ink is sterilized by steaming and allowed to settle; the upper parts, diluted with an equal quantity of distilled water, are employed. A drop of the secretion is thoroughly mixed with an equal quantity of the ink on a glass slide, spread out into a film, allowed to dry and examined with the oil immersion lens without the interposition of a cover-glass. The spirochætes show out white on a dark background. They are minute spiral organisms with six or eight curves, 4–14 μ long and about 0·2 μ broad.

Wassermann's Reaction

The diagnosis of syphilis in cases of difficulty has been rendered much more accurate by the discovery, in 1906, of this test. It depends on the fact that syphilitic serum in the presence of certain lipoid tissue materials leads to a special serum reaction. This reaction can only be carried out by skilled observers in the laboratory*; but the following directions will

* For full details consult Browning and Mackenzie's "Recent Methods in the Diagnosis and Treatment of Syphilis." London, 1911.

enable the clinician to obtain the necessary sample from the patient :—

1. The blood is most easily obtained from one of the large veins at the bend of the elbow.
2. Sterilize a needle of about 1·5 mm. external diameter and a syringe of 10 c.c. capacity by boiling, and rinse out in sterilized normal saline solution.
3. Cleanse the patient's skin well with alcohol, avoiding the use of soap and strong antiseptics, which are apt to cause hæmolysis, and otherwise affect the reaction.
4. Withdraw 5 to 10 c.c. of blood. In doing so the needle should be inserted in the direction opposite to that of the blood-stream in the vein.
5. Express at once into a sterilized centrifuge tube which has been rinsed out with sterile saline solution, being careful to avoid frothing, as this interferes with the separation of the clot ; and plug the tube with cotton-wool.
6. After ten or fifteen minutes free the clot by passing a cold sterilized platinum needle between it and the walls of the tube.
7. Reinsert the plug of cotton-wool and send it to the laboratory, avoiding, as far as possible, any shaking of the tube.

In some cases of nerve disease, e.g. in locomotor ataxia, the reaction may be carried out with spinal fluid as well as with blood serum.

2. **Hydrophobia.**—The chief modern methods of recognizing this disease in an animal are the search for Negri bodies in the hippocampus major, and the inoculation of animals. These procedures can only be carried out in the laboratory. The important point for the practitioner to observe is that the head of the suspected dog should be cut off, wrapped in a cloth, and packed in ice and sawdust for transmission.

Opsonic Technique

For the theory underlying this procedure the student is referred to textbooks on bacteriology ; the following is the method of carrying out the determination of the opsonic index :—

Apparatus.—This consists of—

1. A tube for the separation of leucocytes. This should be about ½ in. wide by 3 in. long, and the lower end should be conical.

2. Several serum tubes or "blood capsules." These are short pieces of glass tubing drawn out to a capillary at each end. The wider part in the centre should be about 1 in. long.

3. A mixing pipette about 8 in. long, consisting of a piece of tubing, one end of which is covered by a rubber nipple, and the other drawn out to a capillary tube. A mark is made on the capillary part about 1 in. from its open end as in Wright's pipette for Widal's test.

4. Microscope slides, prepared by rubbing them lightly with the finest emery paper, and thereafter wiping them carefully.

The **technique** is as follows :—

(*a*) A bacterial emulsion is first prepared. In the case of **pyogenic cocci** and **B. coli** a little of the living culture is taken from an agar tube and rubbed up in a watch-glass with 0·85 per cent. saline solution. The product is centrifuged to clear it of clumps of bacilli. The emulsion should only appear slightly cloudy to the naked eye. In the case of **tubercle bacilli** a short form should be used. A mass of the living growth is removed from the surface of the solid medium, killed by exposure to steam for 1½ hours, washed repeatedly in distilled water, dried on filter-paper in a Petri dish, and rubbed up in an agate mortar with 1·5 per cent. saline solution. The uniform distribution of bacilli in this emulsion should be controlled by the microscope. The thick emulsion which results is transferred to a number of small tubes in which, after sterilization, it can be sealed up and stored. From this stock a weak emulsion can be made as required, by

diluting a few drops with a sufficiency of 1·5 per cent. saline solution. The weak emulsion should be freshly prepared on the day of use. The stock tube may be resealed and used on subsequent occasions, provided that it remains uncontaminated.

(*b*) Preparation of serums. A blood capsule is charged with normal blood drawn from the fingers of one or more healthy persons, and a second with blood from the patient. The ends of the capsules are carefully sealed, they are placed in a centrifuge, and the serum is separated.

(*c*) Washed leucocytes are prepared as follows: The leucocyte tube is nearly filled with a solution containing 1·5 per cent. of sodium citrate and 0·85 per cent. of sodium chloride. The observer's own finger is pricked, and 10–20 drops of blood are withdrawn, the tube being inverted several times during the process to ensure rapid mixing of the blood and solution. The tube is then centrifuged, the supernatant fluid removed, and fresh 0·85 per cent. sodium chloride solution mixed with the deposit. Thereafter the tube is again centrifuged, the fluid is drawn off, and the thin layer of leucocytes which forms above the deposit of red blood-corpuscles is carefully removed with a pipette and expelled into a watch-glass.

(*d*) Equal parts of leucocytes, normal serum, and bacterial emulsion are drawn into the mixing pipette and thoroughly mixed together by repeatedly expelling the contents of the pipette on to a slide and then drawing them up again. The mixture is finally drawn well up into the pipette, the open end is sealed, the nipple removed, and the tube placed in an incubator at 37° C. for fifteen minutes. After removal from the incubator the tube is unsealed, the mixing process repeated, a small droplet is expelled upon a prepared slide, and a film is made, dried, and stained with

Leishman's stain for pyogenic organisms and B. coli, or, after fixation in saturated solution of perchloride of mercury and subsequent washing, with Ziehl-Neelsen's stain in the case of the tubercle bacillus.

Another film is produced in the same manner with the patient's serum in place of normal serum.

(*e*) The number of bacteria found in fifty polymorphonuclear leucocytes is ascertained in each film, and the ratio between the number counted in the film prepared with the patient's serum and the number counted in the film made with normal serum constitutes the "opsonic index." This is expressed in the form of a vulgar fraction whose numerator is the average number of bacteria per leucocyte in the patient's film, and the denominator that in the normal film.

APPENDIX

Weights and Measures

1. English weights and measures.

1 grain, gr.	
1 ounce, oz.	= 437·5 grains.
1 pound, lb.	= 16 ounces = 7,000 grains.
1 minim	= 0·91146 grain.
1 fluid drachm	= 60 minims.
1 fluid ounce	= 8 fluid drachms.
1 pint	= 20 fluid ounces.
1 gallon	= 8 pints.

2. Relation between English and metric systems.

1 grain	= 64·8 milligrammes (mg.).
1 ounce	= 28·3 grammes (grm.).
1 lb.	= 453·6 grammes.
1 gramme	= 15·432 grains.
1 kilo	= 2 lb. 3 oz.
1 minim	= 0·059 cubic centimetres (c.c.).
1 fluid drachm	= 3·5 c.c.
1 fluid ounce	= 28·39 c.c.
1 pint	= 567·9 c.c.
1 c.c.	= 16·9 minims.
1 litre	= 35·2 fluid ounces.
1 inch	= 2·54 centimetres (cm.).
1 foot	= 30·48 cm.
1 yard	= 91·44 cm.
1 cm.	= 0·39 in.
1 metre	= 39·37 in.

3. Conversions.

To convert grammes per 100 c.c. into grains per ounce, multiply by 4·375.

To convert grammes into ounces avoirdupois, multiply by 10 and divide by 283.

To convert litres into pints, multiply by 88 and divide by 50.

To convert kilos into pounds, multiply by 1,000 and divide by 454.

4. Centigrade and Fahrenheit scales.

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9.

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table and figure show the relation of degrees Fahrenheit to Centigrade, as far as is likely to be required in clinical work :—

Centigrade.	Fahrenheit.	Centigrade.	Fahrenheit.
110°	230°	37°	98·6°
100	212	36·5	97·7
95	203	36	96·8
90	194	35·5	95·9
85	185	35	95·0
80	176	34	93·2
75	167	33	91·4
70	158	32	89·6
65	149	31	87·8
60	140	30	86
55	131	25	77
50	122	20	68
45	113	15	59
44	111·2	10	50
43	109·4	+ 5	41
42	107·6	0	32
41	105·8	- 5	23
40·5	104·9	-10	14
40	104·0	-15	+5
39·5	103·1	-20	-4
39	102·2	0·54°	1°
38·5	101·3	1	1·8
38	100·4	2	3·6
37·5	99·5	2·5	4·5

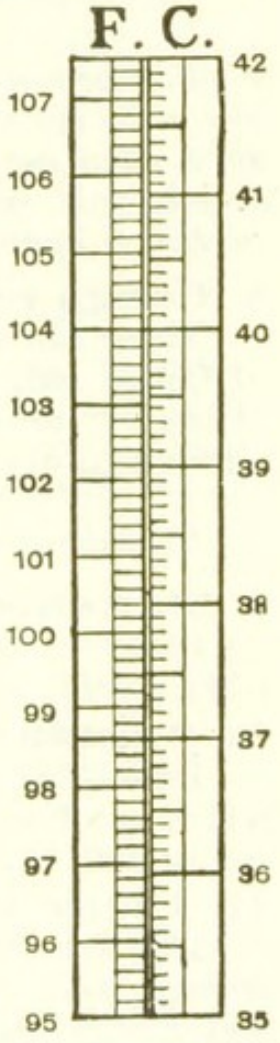


Fig. 145. Fahrenheit and Centigrade scales compared.

Solutions required for Examination of Gastric Contents

5. Phloroglucin and vanillin solution.

Dissolve 2 grm. of phloroglucin and 1 grm. of vanillin in 30 c.c. of absolute alcohol. Keep the solution in the dark, and use it economically.

6. **Boas's resorcin reagent.**

Resorcin	75 gr.
White sugar	45 gr.
Dilute spirit	3½ oz.

Dissolve.

7. **Uffelmann's reagent.**

Carbolic acid (1 in 20)	10 c.c.
Distilled water	20 c.c.

Mix.

Add one or two drops of liq. ferri perchlor. An ametnyst blue solution results. It should be prepared fresh each time, as it does not keep. Lactic acid turns it yellow. Hydrochloric acid simply discharges the blue colour. Acetic and *combined* hydrochloric acid turn it somewhat brownish.

8. **Congo-red test papers.**

These are made by soaking bibulous paper in a solution of Congo red, of the strength of 1 decigramme to 100 c.c. of water, or in a saturated alcoholic solution. They are allowed to dry, and are then ready for use.

Solutions required for Urinary Testing

9. **Standard nitrate of silver solution.**

Dissolve 29·063 grm. of pure *fused* silver nitrate in distilled water, and fill up to 1 litre. Keep in the dark.

10. **Standard uranium solution.**

Dissolve 3·5 grm. of uranium nitrate in 90 c.c. of water to which has been added 2·5 c.c. of glacial acetic acid ; then fill up to 100 c.c. 1 c.c. = 5 mg P_2O_5.

11. **Acetic solution of sodium acetate.**

Dissolve 100 grm. of crystals of sodium acetate in some water ; add 100 c.c. of strong acetic acid, and dilute with water to 1 litre.

12. **Hypobromite solution.**

Dissolve 100 grm. of caustic soda in 250 c.c. of water. Cool, then add 25 c.c. of bromine. The solution is apt to undergo the following decomposition :—

$$3NaBrO = 2NaBr + NaBrO_3.$$

It is therefore better to prepare it as required by adding 2·5 c.c. of bromine to 25 c.c. of the caustic soda solution.

The bromine is supplied in small tubes, which readily break when shaken up smartly with the soda solution in a stout stoppered bottle.

13. Esbach's reagent.

Dissolve 10 grm. of picric acid and 20 grm. of citric acid in about 900 c.c. of boiling water; cool, and add water to 1 litre.

14. Fehling's solution.

(*a*) Take 34·64 grm. of pure sulphate of copper which has been powdered and pressed between bibulous paper, dissolve in 200 c.c. of warm distilled water, cool, and fill up to 500 c.c.

(*b*) Dissolve 180 grm. of crystallized Rochelle salt in 300 c.c. of hot water, filter, and add 70 grm. of pure caustic soda, or 100 grm. of potash; cool, fill up to 500 c.c.

When required, mix equal volumes of (*a*) and (*b*). The result is an alkaline solution of potassic cupric tartrate, of which 1 c.c. is exactly reduced by 5 mg. of pure glucose.

14*a*. Nylander's (or Böttger's) reagent.

Dissolve 10 grm. of caustic soda in 100 c.c. of water, warm, and add 4 grm. of sodio-potassium tartrate and 2 grm. of bismuth subnitrate. Shake thoroughly. A hydrated oxide of bismuth is formed which is kept in solution by the tartrate. The solution should be filtered and kept in a tightly stoppered bottle in the dark.

15. Pavy's solution.

Cupric sulphate	4·158 grm. (36½ gr.).
Rochelle salt	20·4 grm. (178 gr.).
Caustic potash	20·4 grm. (178 gr.).
Strong ammonia .. (Specific gravity 0·880)	300 c.c. (6 oz.).
Water to	1 litre (1 pt.).

Dissolve the Rochelle salt and potash in part of the water, and the sulphate of copper in another (with the aid of heat), pour the copper solution into that of the alkali and Rochelle salt, cool, add the ammonia, then fill up to 1 litre or 1 pt.

Keeps indefinitely. 10 c.c. = 5 mg. glucose.

16. Peptone solution for testing for bile acids.

Powdered peptone (Savory and Moore's)	½ dr.
Salicylic acid	4 gr.
Acetic acid	½ dr.
Distilled water to	8 oz.

Filter repeatedly until transparent.

17. **Solutions for use with the purinometer.**

No. 1

Magnesia mixture*	100 c.c.
Ammonia solution (20%) ..	100 c.c.
Pure talc (finely ground) ..	10 grm.

No. 2

Silver nitrate	1 grm.
Strong ammonia	100 c.c.
Pure talc (finely ground) ..	5 grm.
Distilled water	100 c.c.

Solutions required in the Examination of Blood

18. **Diluting fluid for hæmocytometer.**

Sulphate of soda	104 gr.
Acetic acid	1 dr.
Distilled water	6 oz.

19. **Diluting fluid for hæmocytometer (by Strong's method).**

Sodium chloride	0·95 grm.
Sodium citrate	0·85 grm.
Commercial formalin	½ c.c.
Distilled water	100 c.c.

20. **Toisson's solution** has the following formula :—

Methyl violet, 5B	0·025 grm.
Sod. chlor.	1·000 grm.
Sod. sulph.	8·000 grm.
Neut. glycerin.	30·000 grm.
Aq. destill.	160·000 grm.

Should be filtered just before use.

21. **Hayem's solution.**

Common salt	1 grm.
Sulphate of soda	5 grm.
Corrosive sublimate	0·5 grm.
Distilled water	200 c.c.

22. **Teichmann's test for blood (hæmin test).**

Take up in a pipette some of the deposit to be examined. Rub it up with a small amount of common salt, and evaporate a little of the mixture to dryness on a slide. Moisten the

* Magnesia mixture consists of :—

Magnes. chloride (crystals)	100 grm.
Ammon. chloride	110 grm.
Ammonia	250 grm.
Water to	1,000 c.c.

residue with glacial acetic acid, and put on a cover-glass. Gently heat this over a very small flame for several minutes, avoiding boiling. Let a little glacial acetic acid run in from the side of the cover-glass from time to time during the process. Allow to cool, and examine for hæmin crystals with a high power (Fig. 146).

Some Staining Methods

23. Gram's method.

The following solutions are required :—

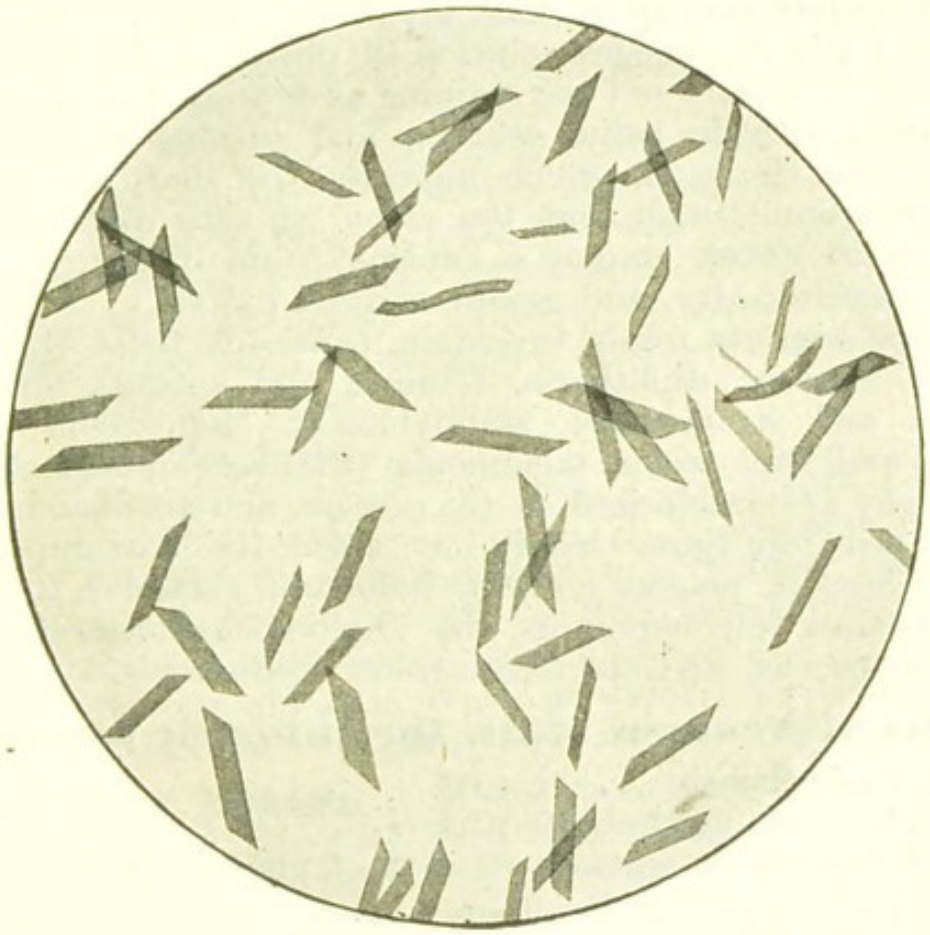


Fig. 146.—Hæmin crystals. × 330.

(1) A solution of gentian violet in aniline water.

Prepare a saturated alcoholic solution of gentian violet and filter it. To 9 parts of aniline oil water (*see* 28) add 1 part of the gentian violet, and filter the mixture. The two solutions should only be mixed shortly before use ; after twenty-four hours the stain becomes less trustworthy.

(2) A solution of 1 grm. of iodine and 2 grm. of iodide of potash in 300 c.c. of distilled water.

Float the cover-slip, face downwards, in a capsule containing some of the stain. In the cold the film is usually

stained in five minutes; if the fluid is heated till steam rises, in about one minute. Without washing, place the cover-slip for half a minute to a minute in solution 2. Here the film becomes black. It is then washed in alcohol or methylated spirit until it ceases to lose colour and becomes a pale grey. Wash in water. Dry between filter-papers. Mount in xylol balsam. It is usual to counterstain the preparation for a few seconds with weak Ziehl-Neelsen (1 part to 20 of distilled water).

The following modification of Gram's method will be found simpler and more efficient: For aniline oil water substitute a 1-in-20 aqueous solution of phenol in the same proportions, and carry out the staining as detailed above. After treatment with the iodine solution, and washing with alcohol, transfer to clove oil, which increases the sharpness of the differentiation; wash out the clove oil with alcohol, then transfer to water, employ a contrast stain if desired, wash again in water, dry, and mount in xylol balsam.

Some bacteria retain this stain, especially those of tuberculosis, leprosy, diphtheria, tetanus, and anthrax amongst bacilli, and streptococci, staphylococci, Micrococcus tetragenus, and Diplococcus pneumoniæ (Fränkel) amongst cocci but many are decolorized by the process, and are thereby distinguished from those already mentioned, the most important being those of plague, cholera, influenza, glanders, typhoid, the Bacillus coli communis, the Diplococcus intracellularis, the gonococcus, and the Micrococcus melitensis.

24. Ziehl-Neelsen stain for tubercle bacilli.

A. Basic fuchsin .. 1 part
Absolute alcohol, 10 parts } dissolve.

Add of 5 per cent. aqueous solution of phenol, 100 parts.

B. Twenty per cent. sulphuric acid.

C. Watery solution of methylene blue. The solution should be nearly saturated. The addition of a trace of ammonia increases the precision of the staining.

Method.—Heat A till steam rises, then float cover-glasses, film down, on it for three or four minutes, rinse in water, immerse in B till decolorized, wash thoroughly in water, counterstain if desired in C for one minute or more, wash rapidly in water, dry, and mount in xylol balsam for permanent preparations, or in Farrant's medium if not to be preserved. Sections require longer staining and must not be dried; clove oil should not be used for clearing purposes, as it often decolorizes the bacteria.

25. **Löffler's stain.** (This should be freshly prepared.)

Concentrated alcoholic solution of methylene blue	1 c.c.
Caustic potash in 0·1 per cent. aqueous solution	3 c.c.

Specimens are stained in five to thirty minutes. Excess of stain is discharged by rapid washing in water acidulated with acetic acid (2 drops of acid in a watch-glassful of water) and all traces of acid well washed out. The specimen is then dried and mounted.

26. **Carbol thionin blue.** (Prepared freshly.)

Saturated solution of thionin blue in 50 per cent. alcohol ..	10 c.c.
1-in-40 solution of phenol in water	100 c.c.

This stain is one of the best for film preparations. After staining, which is rapidly effected, wash the specimen in water, then dry and mount. Sections should, after washing, be passed through alcohol containing a trace of ammonia, thereafter dehydrated by absolute alcohol, cleared with xylol, and mounted in balsam.

27. **Carbol fuchsin.**

Fuchsin	1 part.
Absolute alcohol	10 parts.

Add of 5 per cent. aqueous solution of phenol, 100 parts.

This stain is one of the best for film preparations, and is more permanent than carbol thionin.

Stain for one minute. Wash in water. Decolorize very slightly with alcohol. Wash again in water. Dry and mount.

28. **Aniline water** is made by shaking up 1 part of *colourless* aniline oil with 3 parts of distilled water in a bottle of dark glass. The excess of oil sinks to the bottom, and the supernatant aniline water is decanted and filtered through a filter-paper previously moistened with distilled water, when it is ready for use. Both the aniline oil and the aniline water must be kept in bottles of dark glass.*

29. **Hiss's method of capsule staining.**—This is very useful for the demonstration of pneumococci in the sputum or in pneumococcal exudates. The stain consists of 1 part of a saturated alcoholic solution of fuchsin and 19 parts of distilled water. A film of the material to be examined, having

* It is now usual to substitute a 1-in-20 aqueous solution of phenol for aniline water. It is more readily prepared, keeps better, and its mordant action is quite as efficient.

been dried and fixed, has a few drops of the stain placed upon it, and is heated for a few seconds until steam rises. The stain is then washed off with a 20 per cent. solution of copper sulphate; without being washed in water, the preparation is dried between filter-papers and mounted in balsam.

30. Ehrlich's triacid stain.

Prepare saturated watery solutions of *chemically pure* and crystalline orange-G, acid fuchsin, and methyl green. Then make the following mixture:—

Orange-G solution	13–14 c.c.
Acid fuchsin solution	6–7 c.c.
Distilled water	15 c.c.
Alcohol	15 c.c.
Methyl green solution	12·5 c.c.
Alcohol	10 c.c.
Glycerine	10 c.c.

The fluids must be measured out in the above order, in the same glass, and from the addition of the methyl green onwards the mixture is thoroughly shaken. The solution can be used at once and keeps indefinitely.

Blood films stain in it in one to five minutes, depending upon the particular blood under examination and the mode of its fixation. The exact time required can therefore only be found out by experiment.

31. The Romanowsky stains.

These stains depend for their action on the compounds formed by the interaction of methylene blue and eosin, as originally introduced by Romanowsky. They are used for staining blood films, the cells in pathological fluids, films or tissues containing bacteria, and material containing protozoal parasites, e.g. malaria.

The chief combinations in use are those of Leishman, Jenner, Giemsa, and J. H. Wright. The solutions can be had, made up by Grübler of Leipzig, through any dealer.

Leishman's preparation is perhaps that most used, and the best results are obtained if the worker procures the powder from Grübler and makes the solution himself; 0·15 grm. is dissolved in 100 c.c. of Merck's purest methyl alcohol as follows: The powder is placed in a clean mortar, a little of the alcohol is added, and well rubbed up with a pestle; the undissolved powder is allowed to settle and the fluid decanted into a dry bottle; the process is repeated with fresh fractions of the solvent till practically all the stain is dissolved. The

bottle in which the stain is stored must be well stoppered. If the preparation be too blue, this may be corrected by careful washing with acetic acid 1 in 1,500; if the eosin tint be too strong, it can be lightened by the use of 1-in-7,000 solution of caustic soda. The application of the stain is described in Chap. V. (p. 234).

With this stain the red blood-corpuscles are coloured pink, the nuclei of leucocytes a reddish purple, and any acidophil or basophil granules pink and blue respectively. The nuclear substance of the malarial parasite is stained a reddish purple. In trypanosomes the two nuclei have a purplish stain, the protoplasm is blue, and the edge of the undulatory membrane is stained pink.

32. **Van Ermengem** recommends the following solution for cleaning slides and cover-glasses :—

Concentrated sulphuric acid ..	6 parts.
Potassium bichromate	6 parts.
Water	100 parts.

Leave the glasses in the above solution for twelve hours; wash in water until all trace of bichromate is gone; thereafter preserve in absolute alcohol.

INDEX

PRINTED BY CASSELL & CO., LIMITED, LA BELLE SAUVAGE, LONDON E.C.

R/R